Emotion Regulation Difficulties Related to Depression and Anxiety: A Network Approach to Model Relations among Symptoms, Positive Reappraisal, and Repetitive Negative Thinking

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Abstract

Frequent repetitive negative thinking and infrequent positive reappraisal use are theorized to increase risk for depression and anxiety. Yet, research has studied these regulatory strategies at the disorder level, ignoring the clinical heterogeneity and differential relations among their individual symptoms. This study examined the associations among repetitive negative thinking, positive reappraisal and individual symptoms of depression and anxiety disorders. Regularized partial correlation network models were estimated using cross-sectional data from 468 participants. Results showed that repetitive negative thinking and positive reappraisal were differentially related to affective, cognitive, and somatic symptoms of depression and anxiety. Moreover, repetitive negative thinking was more central than positive reappraisal with stronger connections to individual symptoms. Finally, repetitive negative thinking was more important than positive reappraisal in connecting clusters of depression and anxiety symptoms. These findings cast light on potential pathways through which repetitive negative thinking and positive reappraisal may operate within depression and anxiety.

Keywords: Depression; Anxiety; Emotion Regulation; Repetitive Negative thinking; Positive Reappraisal; Network analysis.

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Depression and anxiety disorders are among the most prevalent, comorbid, and burdensome mental illnesses (Baxter, Scott, Vos, & Whiteford, 2013; Kessler & Bromet, 2013). It is therefore of paramount importance to identify the etiological and maintaining factors for these disorders to improve exiting intervention strategies. Theories of depression and anxiety disorders have implicated emotion regulation difficulties in the onset and maintenance of these disorders (Hofmann, Sawyer, Fang, & Asnaani, 2012; Joormann, 2010; Mennin, Heimberg, Turk, & Fresco, 2006; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Emotion regulation refers to a range of processes that influence the frequency, intensity, and duration of emotional experiences (Gross, 2014). In depression and anxiety disorders, difficulties occur in the use of specific emotion regulation strategies to downregulate negative emotional experiences (Campbell-Sills, Ellard, & Barlow, 2014; Dryman & Heimberg, 2018; Liu & Thompson, 2017).

Two regulatory strategies that play a prominent role in depression and anxiety disorders are the increased use of repetitive negative thinking and decreased use of positive reappraisal (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Drost, van der Does, van Hemert, Penninx, & Spinhoven, 2014; Kivity & Huppert, 2018). Repetitive negative thinking refers to a transdiagnostic process of excessive thinking about negative topics that is passive and/or difficult to control (Mahoney, McEvoy, & Moulds, 2012; Watkins, 2008). Engaging in repetitive negative thinking is related to heightened emotional reactivity (Ruscio, Seitchik, Gentes, Jones, & Hallion, 2011), enhanced negative memory retrieval (Nolen-Hoeksema et al., 2008), and impaired stress recovery (Watkins, 2008). Positive reappraisal has been defined as cognitively reframing the meaning of a distressing event in a less negative or more positive manner to minimize its emotional impact (Garnefski & Kraaij, 2007; Gross, 2014). Decreased

positive reappraisal use may prevent individuals with elevated depression or anxiety levels from obtaining beneficial outcomes associated with this strategy such as increased positive and decreased negative emotions (Gross & John, 2003), more benign interpretations of ambiguity (Everaert et al., 2017), and better stress recovery (Jamieson, Nock, & Mendes, 2012). In sum, extensive research indicates that the use of repetitive negative thinking and less frequent use of positive reappraisal are associated with critical affective, cognitive, and somatic aspects of depression and anxiety disorders.

To date, the pathways through which repetitive negative thinking and positive reappraisal are related to individual symptoms of depression and anxiety remain elusive. Prior research has generally studied problematic emotion regulation at the disorder level. That is, studies have compared the use of repetitive negative thinking and/or positive reappraisal across different diagnostic groups (D'Avanzato, Joormann, Siemer, & Gotlib, 2013) or examined individual differences in the use of these emotion regulation strategies in relation to total scores on self-report measures of depression or anxiety symptoms (Garnefski, Kraaij, & Spinhoven, 2001). However, this dominant focus on the disorder level may be problematic because depression and different anxiety disorders are heterogeneous syndromes characterized by diverse affective, cognitive, and somatic symptoms (Fried & Nesse, 2015; Nandi, Beard, & Galea, 2009; Zimmerman, Ellison, Young, Chelminski, & Dalrymple, 2015). Neglecting the symptomatic heterogeneity of these disorders is an important limitation because it may conceal differential associations between clinically different symptoms (e.g., sad mood vs. suicidal ideation or fear of worst happening vs. numbness) and the use of emotion regulation strategies (Gross & Jazaieri, 2014). This seems highly plausible in light of prior research showing that individual symptoms of depression are differentially related to functional impairments (Fried & Nesse, 2015), adverse life events (Keller, Neale, & Kendler, 2007), as well as cognitive and biological risk factors (Beevers et al., 2019; Marchetti et al., 2018; Santos, Fried, Asafu-Adjei,

& Ruiz, 2017). Knowledge of whether repetitive negative thinking and positive reappraisal are (uniquely) associated with individual symptoms may provide insight into whether variation in the use these emotion regulation strategies may be related to variation in the clinical presentation of anxiety and depression disorders. To gain such a fine-grained understanding of the mechanisms in depression and anxiety, research should adopt a symptom-level approach considering common affective, cognitive, and somatic symptoms of these disorders (Gross & Jazaieri, 2014; Jones, Heeren, & McNally, 2017; Wichers, 2014).

A promising approach to revealing complex relations among individual symptoms of mental disorders and their risk factors is the network approach. According to this perspective, mental disorders are not reflective of a latent common cause, but arise from complex reciprocal influences between their constituting symptoms (Borsboom, 2017; Borsboom & Cramer, 2013). Recently, research has expanded symptom networks to integrate cognitive and biological factors that are hypothesized to play a causal role in mental disorders (Bernstein, Heeren, & McNally, 2017; Heeren & McNally, 2016; Jones, Heeren, et al., 2017). The expanded network models aim to map the causal structure of risk factor – symptom relations in mental disorders. In such networks, symptoms and risk factors are represented by nodes and their pairwise interactions are represented by edges connecting the nodes.

Applying the network approach to depression and anxiety, research has revealed critical differences in the overall importance or connectivity of its constituting symptoms (Bos et al., 2018; Bryant et al., 2017; McNally et al., 2014). For example, loss of interest/pleasure and sad mood emerged as highly connected or central nodes in the depressive symptom network (Boschloo, van Borkulo, Borsboom, & Schoevers, 2016; Fried, Epskamp, Nesse, Tuerlinckx, & Borsboom, 2016) and worry-related symptoms and problems to relax were central nodes in the generalized anxiety disorder network (Beard et al., 2016). Moreover, network studies on the comorbidity between depression and anxiety disorders have found that symptoms of these

disorders were strongly interconnected (Borsboom, Cramer, Waldorp, van der Maas, & Borsboom, 2010; Heeren, Jones, & McNally, 2018; McNally, Mair, Mugno, & Riemann, 2017). For example, of the connections between symptoms of depression and generalized anxiety disorder, a study found that guilty feelings were related to worry-related symptoms and sadness was related to nervousness (Beard et al., 2016). By elucidating the connectivity of symptoms within and across disorders, these network studies are casting new light on the structure and comorbidity of depression and anxiety disorders.

The present study sought to extend prior work by uncovering pathways through which emotion regulation strategy use connects common affective, cognitive, and somatic symptoms that may characterize depression and various anxiety disorders. To this end, this study adopted an expanded network approach to model repetitive negative thinking and positive reappraisal within separate and combined symptom networks of depression and anxiety (cf. Jones, Heeren, et al., 2017). The aims were threefold: (1) to specify differential associations among repetitive negative thinking, positive reappraisal, and individual symptoms of depression and anxiety disorders; (2) to determine the relative importance or connectivity of repetitive negative thinking and positive reappraisal within separate symptom networks to understand their role in the coherence of depression and anxiety symptoms; and (3) to examine whether positive reappraisal and/or repetitive negative thinking act as 'bridges' connecting the clusters of depression and anxiety symptoms to understand their role in the co-occurrence of depression and anxiety symptoms. In addressing these objectives, this study sought to keep with the Research Domain Criteria (Insel et al., 2010) by considering varying degrees of repetitive negative thinking and positive reappraisal along the continuum of symptom severity. In this way, this study attempted to improve the understanding of mental health and illness in terms of complex relations between individual differences in repetitive negative thinking, positive reappraisal, as well as symptom severity of depression and anxiety.

Method

Participants and sampling strategy

This study employed a dimensional approach that considered individual differences in repetitive negative thinking and positive reappraisal along full range of normal to abnormal symptom severity levels of depression and anxiety (cf. Research Domain Criteria; Insel et al., 2010). Therefore, recruitment of participants for this study was unselected. A total of 468 participants (see supplement S1 for demographics) were recruited through Amazon's Mechanical Turk (MTurk). MTurk provides an online crowdsourcing platform with access to large and diverse samples suitable for clinical research collecting mental health data (Chandler & Shapiro, 2016).

Participation in the study was restricted to MTurk users who were 18 years or older and resided in the United States of America. The MTurk workers were recruited between October 2016 and February 2017 to participate in a twenty-wave longitudinal study on emotion regulation and mental health. This article presents data from the first wave of data collection. All participants gave informed consent in accordance with the Institutional Review Board at Yale University. Participants were remunerated per survey and up to a total of 15.20 USD for completing all waves of data collection.

Data quality requirements

Following recommendations for research using crowdsourced samples (Chandler & Shapiro, 2016), only MTurk workers with a history of providing good-quality responses (i.e., an acceptance ratio of $\geq 95\%$) were allowed to participate. To further ensure high data-quality, three questions were presented during the survey to discriminate attentive from inattentive MTurk workers. For example, one validation question read: "Thank you for your work in this survey so far. To show that you are a human, please refuse to answer this question: How many fingers does a typical person have on each hand?". Respondents were then given four response

options (e.g., five, six, ten, and three) which they had to leave blank. These questions were presented at irregular intervals and participants were required to correctly answer all questions. Data from participants failing to meet this requirement were not considered in the analyses. Finally, the data were screened for repeating GPS coordinates to ensure that responses were unique and minimize the possibility that random responses from non-human entities (e.g., bots) contributed to the results of this study. With these requirements, previous research has demonstrated that MTurk data are comparable to those collected in the laboratory (Chandler & Shapiro, 2016).

Procedure and measures

This study utilized widely-used questionnaires to assess individual differences in anxiety and depression symptoms as well as the use of repetitive negative thinking and positive reappraisal as emotion regulation strategies in response to negative events or affect. In light of the study's objectives, symptom questionnaires were selected that measure common cognitive, affective, and somatic components of depression and anxiety because the emotion regulation strategies under investigation have been shown to be related to abnormalities in each of these domains (cf. supra). All questionnaires were presented in randomized order. Participants were instructed to complete the questionnaires in reference to the *past week*. This was to standardize the temporal orientation across all questionnaires and waves of data collection.

Depression symptoms

The Beck Depression Inventory – II (BDI-II; Beck et al., 1996) is the most frequently used self-report instrument to measure depressive symptom severity. On 21 items, respondents indicate the degree to which they have experienced a certain symptom on a four-point scale from 0 to 3. The cognitive, affective, and somatic symptoms assessed by the BDI-II align with the criteria of major depression from the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013). The BDI-II has been shown to have the largest

overlap in symptoms with other common depression measures (Fried, 2017). The psychometric properties of the BDI-II has been extensively supported in both nonclinical and clinical adult samples (Erford, Johnson, & Bardoshi, 2016; Joiner, Walker, Pettit, Perez, & Cukrowicz, 2005).

Anxiety symptoms

The Beck Anxiety Inventory (BAI; Beck et al., 1988; Steer & Beck, 1997) is a widely-used 21-item self-report measure of the severity of common affective, cognitive, and somatic symptoms of anxiety (Clark & Watson, 1991). The BAI has been designed to have limited overlap with symptoms measured by the BDI-II (Beck et al., 1988) and was selected for this study to avoid inflated correlations with BDI-II symptoms because of symptom overlap. Importantly, research has shown that the BAI can be used as an anxiety symptom severity indicator in patients with different anxiety disorders, including social phobia, panic disorder, agoraphobia, and generalized anxiety disorder (Muntingh et al., 2011). On each item of the BAI, respondents indicate the degree to which they have experienced a certain symptom on a four-point scale from 0 (‘not at all’) to 3 (‘severely’). The psychometric properties of the BAI have been extensively documented in adult patient and community samples (Bardhoshi, Duncan, & Erford, 2016).

Repetitive negative thinking

The repetitive negative thinking subscale of the Repetitive Thinking Questionnaire (RTQ; McEvoy, Mahoney, & Moulds, 2010) is a transdiagnostic measure of perseverative negative thinking. The RTQ was developed by modifying items from commonly used measures of worry, rumination, and post-event processing to remove diagnosis-specific content (Mahoney et al., 2012). The 27 items of the repetitive negative thinking subscale are scored on a five-point scale from 1 (‘not true at all’) to 5 (‘very true’) in reference to a recent past distressing situation. Example items are: “My thoughts overwhelmed me” and “I had thoughts

or images asking 'Why do I always react this way?'". Psychometric research evaluating the repetitive negative thinking subscale in nonclinical and clinical samples has demonstrated that the subscale has a good to excellent high internal consistency with α's ranging from .88 to .93 in these sample types (Mahoney et al., 2012; McEvoy et al., 2010). Supporting the convergent validity, the repetitive negative thinking subscale converges with measures of depression and anxiety as well as other related constructs of negative emotions, metacognitive beliefs, cognitive avoidance, and thought suppression (McEvoy et al., 2010). Providing evidence for the divergent validity, the repetitive negative thinking subscale shows divergence with measures of extraversion and alcohol use (Mahoney et al., 2012). The reliability of the repetitive negative thinking subscale of the RTQ in this study was excellent (Cronbach's α=.97 and McDonald's ω_t=.97).

Positive reappraisal[1]

The use of positive reappraisal was measured using the subscale of the Cognitive Emotion Regulation Questionnaire (CERQ; Garnefski, Kraaij, & Spinhoven, 2001) which is one of the most widely used questionnaires to assess emotion regulation strategy use. The 4-item reappraisal subscale specifically measures the use of positive reappraisal in response to negative events. The positive reappraisal subscale does not include diagnosis-specific content. Example items are "I think I can learn something from the situation" and "I think that I can become a stronger person as a result of what has happened". On each of the 4 items, respondents rate the extent to which they engage in positive reappraisal using a 5-point scale from 1 ('almost never') to 5 ('almost always'). The positive reappraisal subscale of the CERQ has good to excellent internal consistency ranging from .82 to .85 (Garnefski et al., 2001; Ireland, Clough,

[1] This study also administered the six cognitive reappraisal items of the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003). The items of the ERQ reappraisal subscale measure the use of reappraisal in response to positive and negative emotional states. This article reports the results obtained with the CERQ positive reappraisal subscale to gain insight into reappraisal in response to *negative* events or experiences. Importantly, identical conclusions were reached when estimating the networks including the ERQ vs. CERQ reappraisal subscales.

& Day, 2017) and an acceptable test-retest reliability coefficient of .57 for a fourteen-month time interval (Garnefski et al., 2001). In support of the convergent and divergent validity in both nonclinical and clinical samples, the positive reappraisal subscale converges with measures of anxiety and depression (Garnefski & Kraaij, 2006, 2007; Garnefski et al., 2001), and diverges with certain problems in emotion regulation such is the limited access to emotion regulation strategies (Ireland et al., 2017). In this study, the reliability of the positive reappraisal subscale of the CERQ was excellent (Cronbach's α=.90; McDonald's ω_t=.93).

Network analysis

To address the first two study aims, a network modeling emotion regulation strategy – symptom relations was specified separately for depression and anxiety symptoms. The emotion regulation – depression symptoms (ER-DEP) network, consisted of the repetitive negative thinking subscale score, the positive reappraisal subscale sore, and the individual BDI-II symptom scores as nodes as well as their edges (i.e., connections among the nodes). Moreover, the emotion regulation – anxiety symptoms (ER-ANX) network, consisted of the repetitive negative thinking subscale sore, the positive reappraisal subscale score, and the individual BAI symptom scores as nodes as well as their edges.

To address the third study aim, a network combining both depression and anxiety symptoms was specified. This network will be referred to as the emotion regulation – depression and anxiety co-occurrence symptom (ER-COO) network and included as nodes the repetitive negative thinking subscale score, the positive reappraisal subscale score, and symptoms of the BDI-II and BAI as well as their edges.

For each network, the conceptual overlap between regulatory strategies and individual symptoms was examined. To this end, the goldbricker function of R package *networktools* (Jones, 2017) was used to identify strongly correlated item pairs ($r \geq .70$) that had less than 20% unique correlations with other items (see also Bernstein, Heeren, & McNally, 2019).

Importantly, the algorithm did not identify potentially redundant nodes in the ER-DEP, ER-ANX, or ER-COO networks.

Network estimation and inference

The networks were estimated via Gaussian Graphical Models (GGM) using the R package *qgraph* (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012). In a GGM, the edges represent pairwise relations between two nodes controlling for all other nodes in the network. The GGMs were estimated based on non-parametric Spearman rho correlation matrices. The GGMs were regularized using a graphical least absolute shrinkage and selection operator (glasso) algorithm. This regularization procedure shrinks all edges and sets small edges to zero to return parsimonious networks (Friedman, Hastie, & Tibshirani, 2008). This powerful method avoids estimating false positive edges and provides insight into strong relations in the dataset (Epskamp, Kruis, & Marsman, 2016). The GGM tuning parameter was set to the conservative value of 0.5 to increase the specificity of the estimated networks (Epskamp & Fried, 2016). This method enables examination of unique relations among repetitive negative thinking, positive reappraisal, and individual symptoms in the networks. In the visualized networks, blue edges represent positive relations and orange edges represent negative relations between the network nodes. Thicker edges indicate stronger associations between the nodes.

The relative importance of repetitive negative thinking and positive reappraisal within the ER-DEP and ER-ANX networks was examined using the (one-step) expected influence metric (Robinaugh, Millner, & McNally, 2016) using the R package *networktools* (Jones, 2017). This metric is more appropriate than traditional centrality metrics (e.g., strength centrality) when networks contain both positive and negative edges (Robinaugh et al., 2016). Expected influence is defined as the sum of all edges extending from a given node (maintaining the sign of each edge). Higher expected influence values indicate greater importance in the network.

The role of positive reappraisal and repetitive negative thinking as potential nodes connecting clusters of depression and anxiety symptoms in the ER-COO network was examined using the (one-step) bridge expected influence metric (Heeren et al., 2018; Jones, Ma, & McNally, 2017) using the package *networktools* for R (Jones, 2017). Bridge expected influence is defined as the sum of all edges that exist between a given node and the nodes in the other clusters. In line with prior work (Jones, Ma, et al., 2017), the clusters of interconnected nodes were defined a-priori so that the symptom clusters corresponded with depression and anxiety symptoms. The ER-COO network was separated into three clusters: a cluster of the emotion regulation strategies (i.e., repetitive negative thinking and positive reappraisal), a cluster of depression symptoms (i.e., the 21 BDI-II items), and a cluster of anxiety symptoms (i.e., the 21 BAI items). In this way, the bridge expected influence values for positive reappraisal or repetitive negative thinking reflected their connectivity with the clusters of depression and anxiety symptoms.

Of note, it is possible that unequal variances of positive reappraisal, repetitive negative thinking, as well as the BDI-II and BAI items affect the centrality of the nodes, thereby influencing the observed network structure (Terluin, de Boer, & de Vet, 2016). Therefore, correlations between (bridge) expected influence values and standard deviations (*SD*s) of the individual nodes were examined. The expected influence values were not significantly related to the nodes' *SD*s for the ER-DEP (ρ=.17, p=.431) and ER-ANX (ρ=-.05, p=.806) networks. Furthermore, the bridge expected influence values were not significantly related to the nodes' *SD*s in the ER-COO network (ρ=.14, p=.358). This indicates that differential variability of the nodes in the estimated networks did not drive their centrality.

Network stability

The robustness of the network estimates was examined using the R package *bootnet* (Epskamp, Borsboom, & Fried, 2018). The stability of the edge weights was examined by

constructing a 95% confidence interval (CI) around each edge using non-parametric bootstrapping with 1000 samples and by computing bootstrapped difference tests for edge weights. Furthermore, the stability of the centrality metrics was examined using case-dropping subset bootstrapping with 1000 samples and by computing bootstrapped difference tests for expected influence and bridge expected influence values. This method draws samples from subsets from the original data and re-estimates the centrality metric for each subset. Correlation stability (CS) coefficients were calculated to quantify the stability of the (bridge) expected influence metrics. The coefficient should not be below 0.25 and preferably above 0.5 (Epskamp et al., 2018). For both the ER-DEP and ER-ANX networks, the CS coefficient of expected influence was 0.75. For the ER-COO network, the CS coefficient of bridge expected influence was 0.59. Note that all CS-coefficients were above the recommended threshold for stable estimation (Epskamp et al., 2018). The results of the stability analyses for the ER-DEP, ER-ANX, and ER-COO networks are provided in supplement S3.

To further investigate the stability of the estimated centrality metrics, correlations were examined between the centrality metrics of the networks estimated using the wave 1 data and networks using the data from four subsequent waves that were part of this twenty-wave longitudinal study (the waves were separated by one-week time intervals). For the ER-DEP network, the expected influence values at wave 1 correlated .932 with the values at wave 2, .907 with wave 3, .900 with wave 4, and .921 with wave 5. For the ER-ANX network, the expected influence values at wave 1 correlated .885 with the values at wave 2, .946 with wave 3, .916 with wave 4, and .946 with wave 5. Lastly, for the ER-COO network, the bridge expected influence values at wave 1 correlated .944 with the values at wave 2, .941 with wave 3, .889 with wave 4, and .904 with wave 5. These correlations suggest stability of the relative importance of the individual nodes in the different networks over the course of five weeks.

Results

Sample characteristics

Participants' BDI-II and BAI scores represented almost the full range of symptom severity. On the BDI-II (*M*=15.50, *SD*=12.62), a total of 234 respondents reported minimal (range: 0–13), 78 reported mild (range: 14–19), 79 reported moderate (range: 20–28), and 77 reported severe (range: 29–58) depressive symptoms. On the BAI (*M*=12.51, *SD*=10.44), a total of 221 respondents reported minimal (range: 0–9), 84 reported mild (range: 10–15), 101 reported moderate (range: 16–24), and 62 reported severe (range: 25–52) anxiety symptoms. The means and standard deviations for each of the symptom items of the BDI-II and BAI are provided in supplement 2. This table shows that there was sufficient and comparable variability in the item scores of both the BDI-II and BAI, similar to prior research (Bos et al., 2018).

Furthermore, the scores on the repetitive negative thinking subscale of the RTQ (*M*=70.90, *SD*=26.66; range 27-135) and the positive reappraisal subscale of the CERQ (*M*=14.23, *SD*=4.10; range: 4-20) covered the full range of emotion regulation strategy use. Together, the distributional characteristics of the variables allowed the present investigation to estimate the strength of the associations among individual differences in repetitive negative thinking, positive reappraisal, and individual symptoms of depression and anxiety.

The ER-DEP network

The glasso ER–DEP network structure is depicted in Figure 1A. Various edges between emotion regulation strategies and depressive symptoms survived the conservative regularization procedure. Repetitive negative thinking was most strongly related to guilty feelings (BDI-II item 5), changes in appetite (BDI-II item 18), agitation (BDI-II item 11), self-criticalness (BDI-II item 8), and sadness (BDI-II item 1). Positive reappraisal was most strongly and negatively related to pessimism (BDI-II item 2). Repetitive negative thinking and positive reappraisal were only weakly related.

To examine the overall connectivity or centrality of the individual nodes, expected influence values were computed for all nodes in the network (see Figure 1B). Among the most central nodes in the network were worthlessness (BDI-II item 14), loss of pleasure (BDI-II item 4), repetitive negative thinking, self-dislike (BDI-II item 7), loss of interest (BDI-II item 12), and sadness (BDI-II item 1). Positive reappraisal had the lowest value on expected influence of all variables in the network. The centrality difference test (see Figure S3-4A in supplement 3) suggested that the expected influence value for repetitive negative thinking was significantly greater than the value for positive reappraisal. This indicates that repetitive negative thinking was significantly more connected to depressive symptoms than positive reappraisal in the ER-COO network.

The ER-ANX network

Figure 2A presents the glasso ER–ANX network structure. Several edges between emotion regulation strategies and anxiety symptoms survived the glasso procedure. The strongest edges between repetitive negative thinking and anxiety symptoms were found for fear of losing control (BAI item 14), fear of worst happening (BAI item 5), unable to relax (BAI item 4), and nervous (BAI item 10). Positive reappraisal was negatively related to fear of worst happening BAI item 5). Repetitive negative thinking and positive reappraisal were not related.

The expected influence values for the nodes in the ER-ANX network (see Figure 2B) were inspected to examine their relative importance. The five most central nodes in the network were: shaky/unsteady (BAI item 13), fear of worst happening (BAI item 5), terrified or afraid (BAI item 9), difficulty in breathing (BAI item 15), and faint/lightheaded (BAI item 19). Repetitive negative thinking had a significantly higher expected influence value than positive reappraisal (see Figure S3-4B in supplement 3), suggesting that repetitive negative thinking was significantly more connected to anxiety symptoms than positive reappraisal.

The ER-COO network

Figure 3A depicts the network structure and 3B the centrality plot for the ER-COO network. The values of the bridge expected influence metric were examined for repetitive negative thinking and positive reappraisal. Repetitive negative thinking had the highest value of bridge expected influence of all nodes in the ER-COO network. By contrast, positive reappraisal has the lowest value of all nodes in the network. The difference test (see Figure S3-4C in supplement 3) showed that the bridge expected influence value for repetitive negative thinking was significantly higher than the value for positive reappraisal.

Inspecting the strongest edges, repetitive negative thinking connected the depressive symptoms of guilty feelings (BDI-II item 5), changes in appetite (BDI-II item 18), and self-criticalness (BDI-II item 8) with anxiety symptoms of fear of losing control (BAI item 14), fear of worst happening (BAI item 5), and nervousness (BAI item 10). Positive reappraisal was (negatively) related to pessimism (BDI-II item 2) and weakly (negatively) related to fear of worst happening (BAI item 5).

Discussion

Employing network analysis, this study aimed to reveal the pathways by which repetitive negative thinking and positive reappraisal connect various affective, cognitive, and somatic symptoms that may characterize depression and anxiety disorders. Inspecting associations between emotion regulation strategies and individual symptoms, it was observed that repetitive negative thinking and positive reappraisal were differentially related to individual depression and anxiety symptoms (cf. Study aim 1). Regarding depression symptoms, repetitive negative thinking was positively related to guilty feelings, changes in appetite, agitation, self-criticalness, and sadness. Positive reappraisal was negatively related to pessimism. Regarding anxiety symptoms, repetitive negative thinking was positively related to fear of losing control, fear of worst happening, unable to relax, and nervousness. Positive reappraisal was negatively

related to fear of worst happening. The absence of uniform connections with individual symptoms suggests that repetitive negative thinking and positive reappraisal may not function as a central mechanism that is equally important to all symptoms of depression and anxiety. Instead, the role of repetitive negative thinking and positive reappraisal in depression and anxiety may be confined to specific affective, cognitive, and/or somatic aspects of these disorders. Through their relation with specific symptoms, individual differences in the use of repetitive negative thinking and positive reappraisal may be associated with variation in the clinical presentation of depression and anxiety disorders. Indeed, this finding adds to emerging research showing that psychosocial, cognitive, and biological risk factors differ considerably for individual symptoms (Fried, Nesse, Zivin, Guille, & Sen, 2014; Keller et al., 2007; Marchetti et al., 2018; Santos et al., 2017).

Of the specific regulatory strategy – symptom relations, the negative partial correlations between positive reappraisal and symptoms of pessimism and fear of worst happening are notable. This suggests that decreased use of positive reappraisal is related to more negative views and expectations about the future, which is a known risk factor for suicide and other mental disorders (Roepke & Seligman, 2016). Moreover, this study also revealed intriguing relations between repetitive negative thinking and the symptoms guilty feelings and self-criticalness. Whereas most research focused on the role of repetitive negative thinking in negative emotions such as sadness (Nolen-Hoeksema et al., 2008; Watkins, 2008), its relations with guilt and self-criticalness remain underexplored. Interestingly, recent research has linked self-criticism to greater negative thinking in response to a lab-stressor (Bernstein et al., 2017). Similarly, it seems plausible that feelings of guilt may fuel and characterize the content of repetitive negative thinking. These findings warrant further research exploring the etiological significance of these pathways.

Determining their relative importance in separate symptom networks, this study found that repetitive negative thinking had significantly higher value on expected influence compared to positive reappraisal (cf. Study aim 2). This suggests that repetitive negative thinking was more strongly interconnected to symptoms of depression and anxiety than positive reappraisal. Thus, individuals who frequently engage in repetitive negative thinking also experience a larger number of depression and anxiety symptoms. By contrast, individuals who are using positive reappraisal less frequently do not necessarily experience a wide range of depression and anxiety symptoms. This difference in connectivity with individual symptoms may account for the stronger relation between total scores of depression/anxiety (which capture the shared variance of all symptoms) and forms of repetitive negative thinking vs. positive reappraisal (Aldao et al., 2010). Furthermore, it is to note that repetitive negative thinking was among the most important nodes in the depression (ER-DEP) network but not in the anxiety (ER-ANX) symptom network. This suggests that repetitive negative thinking may be a particularly relevant mechanism in understanding the coherence among depression symptoms and the clinical presentation of depression as a disorder. While these findings suggest that repetitive negative thinking and positive reappraisal differ in their connectivity with individual symptoms, it should be emphasized that this does not mean that positive reappraisal is unimportant. Positive reappraisal was connected to clinically important symptoms in the separate networks of depression and anxiety symptoms.

In examining whether regulatory strategies act as bridges connecting depression and anxiety symptoms (cf. Study aim 3), it was found that repetitive negative thinking had a higher bridge expected influence value than positive reappraisal. This indicates that repetitive negative thinking was relatively more important than positive reappraisal in connecting clusters of depression and anxiety symptoms. In particular, repetitive negative thinking connected several symptoms of depression (e.g., guilty feelings, changes in appetite, and self-criticalness) and

anxiety (fear of losing control, fear of worst happening, nervousness). By contract, positive reappraisal was (negatively) related to the depression symptoms pessimism and weakly related to fear of worst happening. These findings suggest that repetitive negative thinking could particularly important as mechanism explaining the high co-occurrence between depression and anxiety symptoms.

Together, the findings of this study suggest that repetitive negative thinking and positive reappraisal explain the relations between individual symptoms of depression and anxiety. As such, this study elucidates potential pathways through which these regulatory strategies cause and/or be caused by symptoms of depression and anxiety disorders. Indeed, these novel findings highlight how including hypothesized risk factors may enrich symptom networks to gain a precise understanding of processes operating in mental disorders and their comorbid forms (Bernstein et al., 2017; Heeren & McNally, 2016; Jones, Heeren, et al., 2017).

Understanding how putative risk factors such as repetitive negative thinking and positive reappraisal are related to individual symptoms of depression and anxiety may provide important clues for treatment. Indeed, targeting those risk factors that are strongly connected to individual symptoms of a disorder and/or clusters of symptoms belonging to multiple disorders holds potential to effectively treat (comorbid) mental disorders. The results of this study suggest that interventions focusing on repetitive negative thinking could be effective at reducing various symptoms of depression and anxiety as well as their co-occurring forms. If causally linked, reducing repetitive negative thinking may result in improvements in affective (e.g., sadness, guilty feelings, fear of losing control/worst happening), cognitive (e.g., self-criticalness), and somatic (e.g., changes in appetite, agitation, unable to relax, nervousness) symptoms. Of note, increasing the use of positive reappraisal may still be important (e.g., in terms of suicide risk reduction). If patients display symptoms related to negative expectations about the future (e.g., pessimism, fear of the worst happening), then treatments may adopt a symptom-focused

strategy and increase positive reappraisal use (e.g., through cognitive restructuring) to obtain relief in these specific symptoms. Again, caution about these clinical implications is required because longitudinal research has yet to establish the temporal relations among repetitive negative thinking, positive reappraisal, and individual symptoms to understand the importance of the regulatory strategies as part of a causal system of interacting depression and anxiety symptoms (Rodebaugh et al., 2018).

Several limitations to this study point to future directions. First, the cross-sectional data utilized to construct the emotion regulation strategy – symptom networks preclude claims about causality. As such, the present study cannot rule out whether repetitive negative thinking or positive reappraisal influence and/or are influenced by symptoms of depression and anxiety. Intensive longitudinal data with repeated assessments of emotion regulation strategies and symptoms are better suited to clarify the temporal precedence of emotion regulation strategies and symptoms of anxiety and depression.

Second, this study recruited a general population sample of individuals reporting a variety of symptom severity levels which may limit the generalizability of the findings to clinical samples. Yet, the dimensional approach of this study is be particularly suited to cast light on varying degrees of problems in emotion regulation along the continuum of symptom severity. Indeed, a considerable portion of the participants reported severe levels of depression and anxiety symptoms. This approach complies with the Research Domain Criteria (Insel et al., 2010) and may help to better understand the heterogeneous nature of conventional diagnostic categories (Fried & Cramer, 2017). Note that this study did not explore differences in network structure between low vs. high symptom levels because using the conventional cutoffs for the BDI-II and BAI restricts the variability in the network variables, which may impact the network structure. Therefore, future work should replicate the present findings in clinical samples of depression and anxiety disorders.

Third, the analyses utilized data from single item self-report measures of depression and anxiety symptoms. It is possible that these measures imperfectly capture the clinical phenomena. Future network studies should use multiple items and methods to measure each symptom. In this respect, there is research suggesting symptom networks based on (single item) self-report vs. clinician-report data may be highly similar (Moshier et al., 2018). This challenges the notion that network methods produce unreliable results due to estimations consisting primarily of measurement error.

Fourth, this study was limited by its focus on two prominent emotion regulation strategies in depression and anxiety disorders: repetitive negative thinking and positive reappraisal. Other emotion regulation strategies (Aldao et al., 2010) as well as related factors such as information-processing biases (Everaert et al., 2017) and personality variables (Stanton, Rozek, Stasik-O'Brien, Ellickson-Larew, & Watson, 2016) may be important in understanding the connections between symptoms of depression. Therefore, future studies could integrate a broader set of variables in symptom networks of depression and anxiety.

Finally, it is possible that the observed associations among emotion regulation strategies and psychopathology symptoms are specific to the depression and anxiety questionnaires utilized for this study. Self-report instruments of depression and anxiety often differ in the set of symptoms that are measured. This restricts the relations that can be observed in network models. However, the questionnaires employed in this study are widely-used self-report measures that were carefully selected based on their psychometric properties and the variety in common cognitive, affective, and somatic symptoms assessed. Therefore, the current study contributes to knowledge of how emotion regulation strategy use may be related to common symptoms of depression and anxiety. The findings may serve as an impetus for future studies that use other questionnaires of depression and anxiety to determine the robustness of these initial observations.

Despite these limitations, this study advances the understanding of the complex relations among repetitive negative thinking, positive reappraisal, and symptoms of depression and anxiety in important ways. Using network analysis, this study observed that (a) repetitive negative thinking and positive reappraisal were differentially related to individual depression and anxiety symptoms; (b) repetitive negative thinking was more strongly connected to symptoms of depression and anxiety than positive reappraisal; and (c) repetitive negative thinking was relatively more important than positive reappraisal in connecting clusters of depression and anxiety symptoms. Collectively, the results provide cues to the pathways through which repetitive negative thinking and positive reappraisal may influence symptoms of depression and anxiety.

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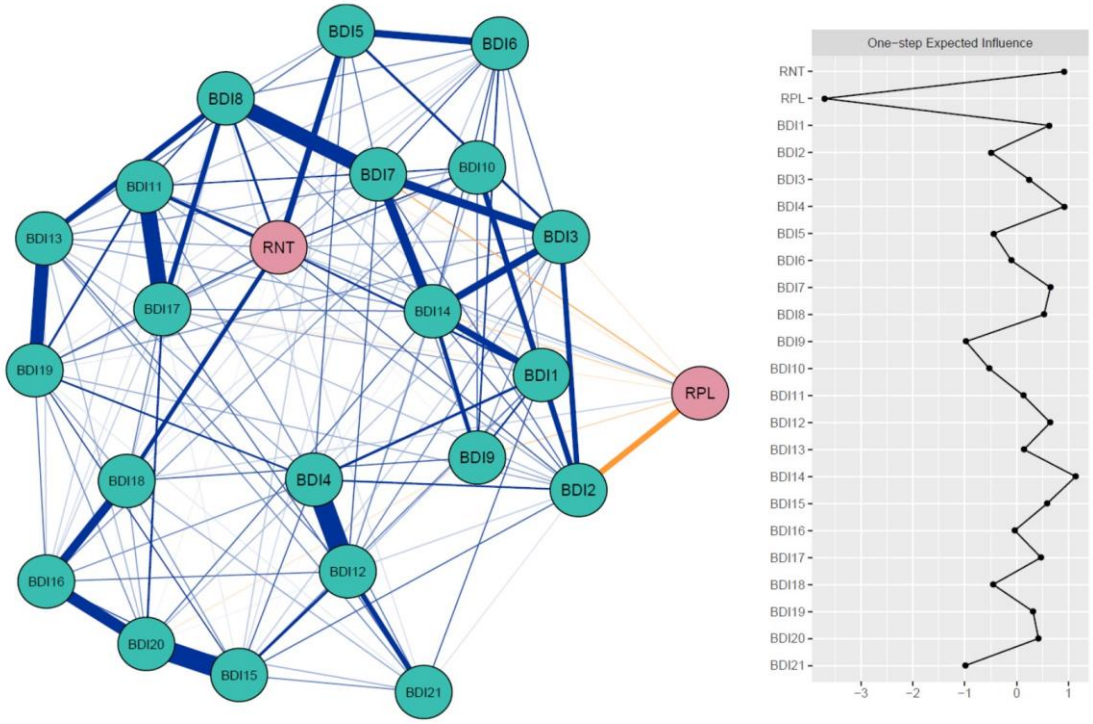
Figure 1. *ER-DEP network*.

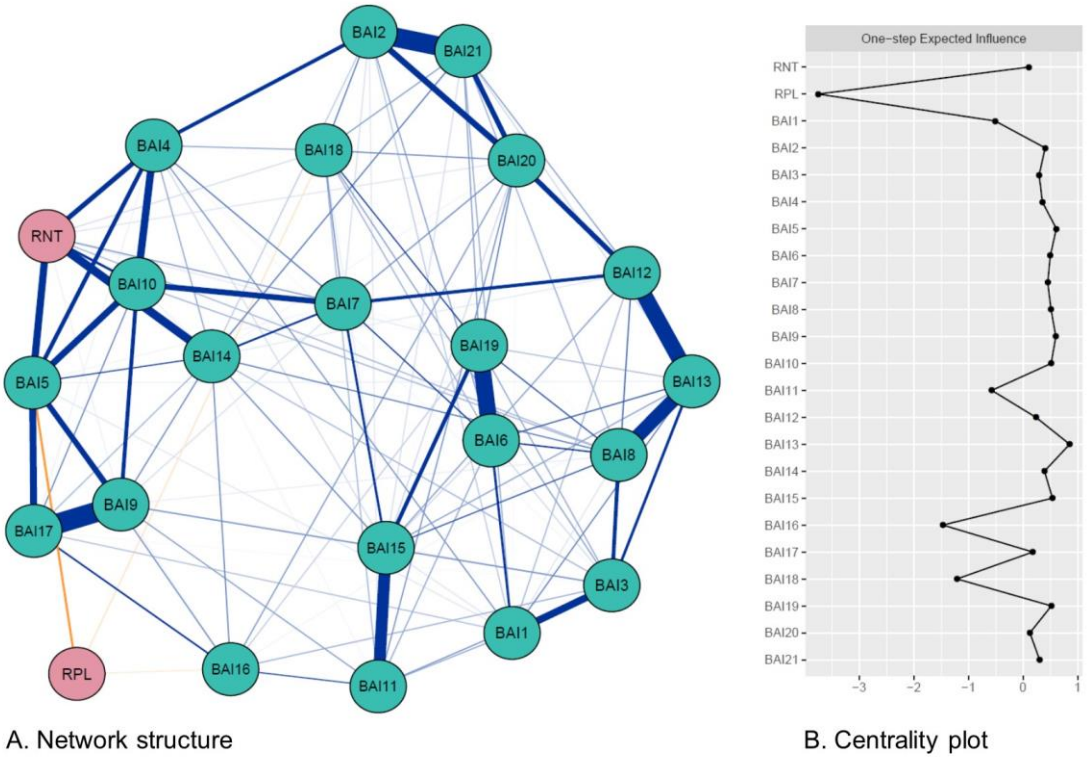


A. Network structure

B. Centrality plot

Note. RNT=Repetitive negative thinking; RPL=Positive reappraisal; BDI-II-1=Sadness; BDI-II-2=Pessimism; BDI-II-3=Past failure; BDI-II-4=Loss of pleasure; BDI-II-5=Guilty feelings; BDI-II-6=Punishment feelings; BDI-II-7=Self-dislike; BDI-II-8=Self-criticalness; BDI-II-9=Suicidal thoughts or wishes; BDI-II-10=Crying; BDI-II-11=Agitation; BDI-II-12=Loss of interest; BDI-II-13=Indecisiveness; BDI-II-14=Worthlessness; BDI-II-15=Loss of energy; BDI-II-16=Changes in sleeping pattern; BDI-II-17=Irritability; BDI-II-18=Changes in appetite; BDI-II-19=Concentration difficulty; BDI-II-20=Tiredness/fatigue; BDI-II-21=Loss of interest in sex

Figure 2. *ER-ANX network*.

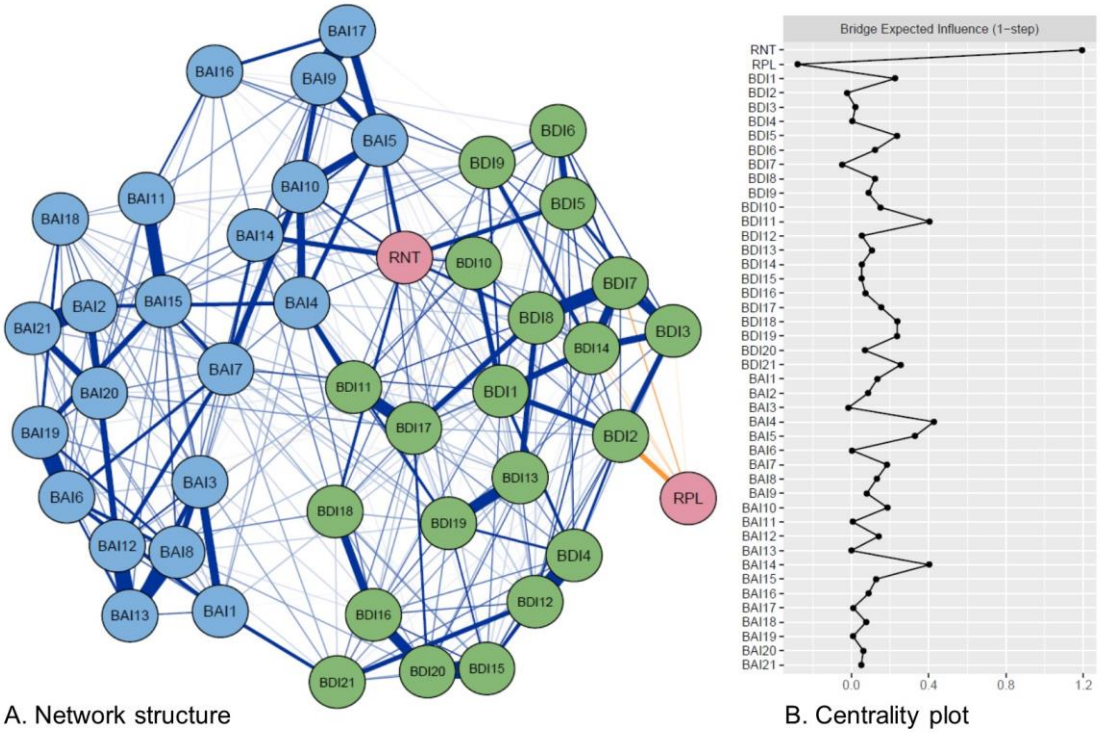


A. Network structure

B. Centrality plot

Note. RNT=Repetitive negative thinking; RPL=Positive reappraisal; BAI-1=Numbness or tingling; BAI-2=Feeling hot; BAI-3=Wobbliness in legs; BAI-4=Unable to relax; BAI-5=Fear of worst happening; BAI-6=Dizzy or lightheaded; BAI-7=Heart pounding/racing; BAI-8=Unsteady; BAI-9=Terrified or afraid; BAI-10=Nervous; BAI-11=Feeling of choking; BAI-12=Hands trembling; BAI-13=Shaky/unsteady; BAI-14=Fear of losing control; BAI-15=Difficulty in breathing; BAI-16=Fear of dying; BAI-17=Scared; BAI-18=Indigestion; BAI-19=Faint / lightheaded; BAI-20=Face flushed; BAI-21=Hot/cold sweats

Figure 3. *ER-COO network*.



A. Network structure

B. Centrality plot

Note. RNT=Repetitive negative thinking; RPL=Positive reappraisal; BDI-II-1=Sadness; BDI-II-2=Pessimism; BDI-II-3=Past failure; BDI-II-4=Loss of pleasure; BDI-II-5=Guilty feelings; BDI-II-6=Punishment feelings; BDI-II-7=Self-dislike; BDI-II-8=Self-criticalness; BDI-II-9=Suicidal thoughts or wishes; BDI-II-10=Crying; BDI-II-11=Agitation; BDI-II-12=Loss of interest; BDI-II-13=Indecisiveness; BDI-II-14=Worthlessness; BDI-II-15=Loss of energy; BDI-II-16=Changes in sleeping pattern; BDI-II-17=Irritability; BDI-II-18=Changes in appetite; BDI-II-19=Concentration difficulty; BDI-II-20=Tiredness/fatigue; BDI-II-21=Loss of interest in sex; BAI-1=Numbness or tingling; BAI-2=Feeling hot; BAI-3=Wobbliness in legs; BAI-4=Unable to relax; BAI-5=Fear of worst happening; BAI-6=Dizzy or lightheaded; BAI-7=Heart pounding/racing; BAI-8=Unsteady; BAI-9=Terrified or afraid; BAI-10=Nervous; BAI-11=Feeling of choking; BAI-12=Hands trembling; BAI-13=Shaky/unsteady; BAI-14=Fear of losing control; BAI-15=Difficulty in breathing; BAI-16=Fear of dying; BAI-17=Scared; BAI-18=Indigestion; BAI-19=Faint/lightheaded; BAI-20=Face flushed; BAI-21=Hot/cold sweats

Supplement

for

Emotion Regulation Difficulties Related to Depression and Anxiety: A Network Approach to Model Relations among Symptoms, Positive Reappraisal, and Repetitive Negative Thinking

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[2] Yale University, United States of America

Supplement 1: Demographic characteristics of the sample

Table S1. *Demographic characteristics*.

Age (*M*)	34.29 (*SD*=11.99)
Gender	*N*
Male	140
Female	328
Race	
White or Caucasian	367
Black or African American	35
American Indian/Alaska Native	1
Asian American	30
Hispanic American	8
Other	27
Education	
No high school degree	4
High school graduate	49
Some college	129
Two-year college graduate	50
Four-year college graduate	162
Master degree	59
Doctoral degree	11
Professional degree	4

Supplement 2: Descriptive statistics for the depression (BDI-II) and anxiety (BAI) symptom items.

Table S2. *Descriptive statistics for the individual items of the BDI-II and BAI.*

	BDI-II					BAI				
Item	Symptom	*M*	*SD*	Min	Max	Symptom	*M*	*SD*	*Min*	*Max*
1	Sadness	0.611	0.742	0	3	Numbness or tingling	0.509	0.748	0	3
2	Pessimism	0.795	0.828	0	3	Feeling hot	0.765	0.861	0	3
3	Past failure	0.850	0.916	0	3	Wobbliness in legs	0.378	0.661	0	3
4	Loss of pleasure	0.801	0.859	0	3	Unable to relax	1.224	0.990	0	3
5	Guilty feelings	0.652	0.769	0	3	Fear of worst happening	1.096	1.029	0	3
6	Punishment feelings	0.504	0.877	0	3	Dizzy or lightheaded	0.575	0.755	0	3
7	Self-dislike	0.853	1.008	0	3	Heart pounding/racing	0.720	0.844	0	3
8	Self-criticalness	0.889	0.912	0	3	Unsteady	0.462	0.705	0	3
9	Suicidal thoughts or wishes	0.263	0.564	0	3	Terrified or afraid	0.583	0.855	0	3
10	Crying	0.491	0.795	0	3	Nervous	1.197	0.968	0	3
11	Agitation	0.735	0.800	0	3	Feeling of choking	0.165	0.482	0	3
12	Loss of interest	0.784	0.879	0	3	Hands trembling	0.365	0.655	0	3
13	Indecisiveness	0.665	0.878	0	3	Shaky / unsteady	0.451	0.695	0	3
14	Worthlessness	0.639	0.907	0	3	Fear of losing control	0.562	0.834	0	3
15	Loss of energy	0.936	0.885	0	3	Difficulty in breathing	0.327	0.656	0	3
16	Changes in sleeping pattern	1.051	0.917	0	3	Fear of dying	0.338	0.700	0	3
17	Irritability	0.797	0.853	0	3	Scared	0.639	0.835	0	3
18	Change in appetitive	0.705	0.880	0	3	Indigestion	0.776	0.939	0	3
19	Concentration difficulty	0.688	0.815	0	3	Faint / lightheaded	0.412	0.682	0	3
20	Tiredness of fatigue	0.934	0.849	0	3	Face flushed	0.453	0.763	0	3
21	Loss of interest in sex	0.861	1.022	0	3	Hot/cold sweats	0.513	0.821	0	3

Notes. Means and standard deviations are provided for the full sample of *N*=468 participants. BDI-II: Beck Depression Inventory – II; BAI=Beck Anxiety Inventory; Both the BDI-II and BAI are rated on a four-point scale ranging from 0 to 3.

Supplement 3: Results of the network stability analyses

S3-1. Non-parametric bootstrapped 95% confidence intervals (CIs) for the estimated edge weights

Figure S3-1A. *Bootstrapped 95%-CIs for the ER-DEP network.*

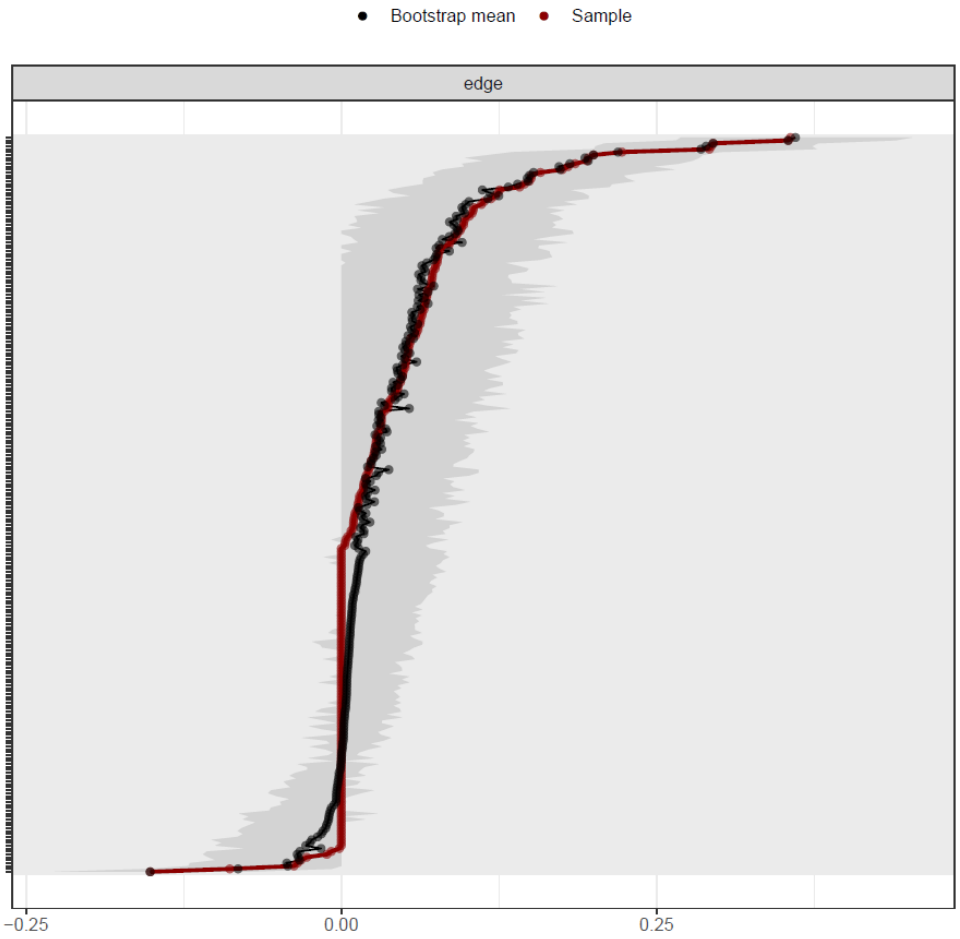


Figure S3-1B. *Bootstrapped 95%-CIs for the ER-ANX network.*

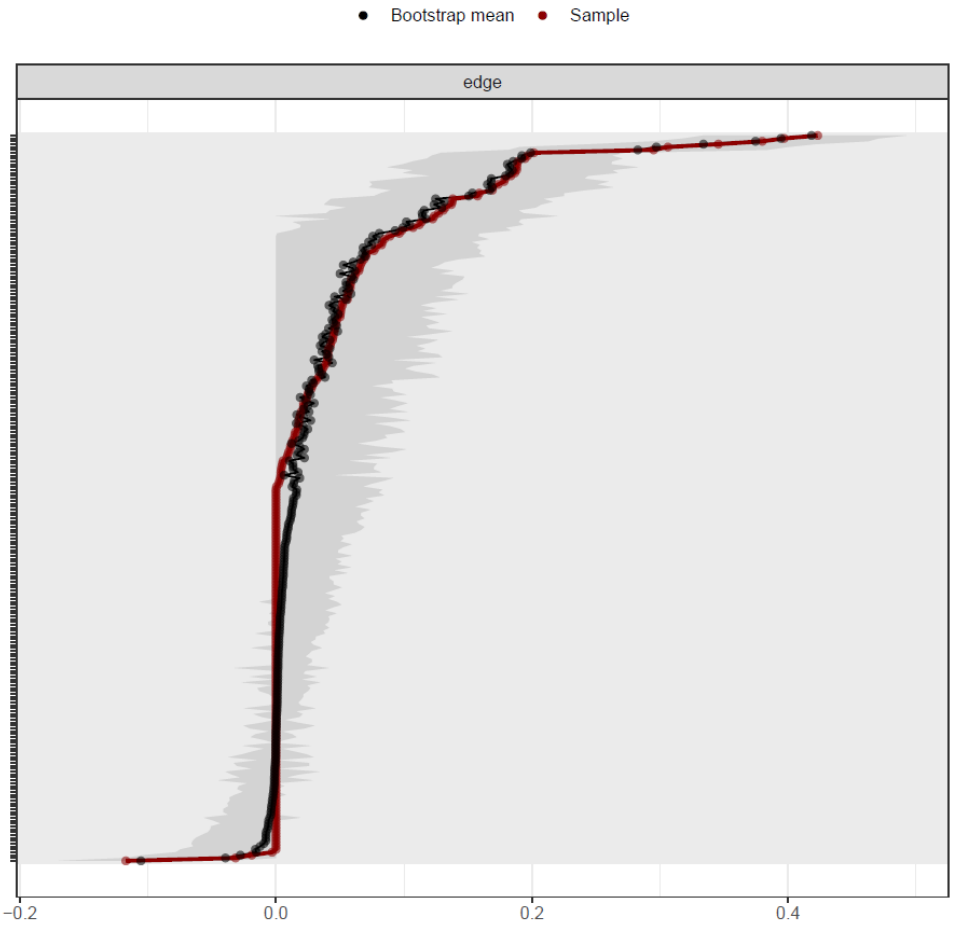
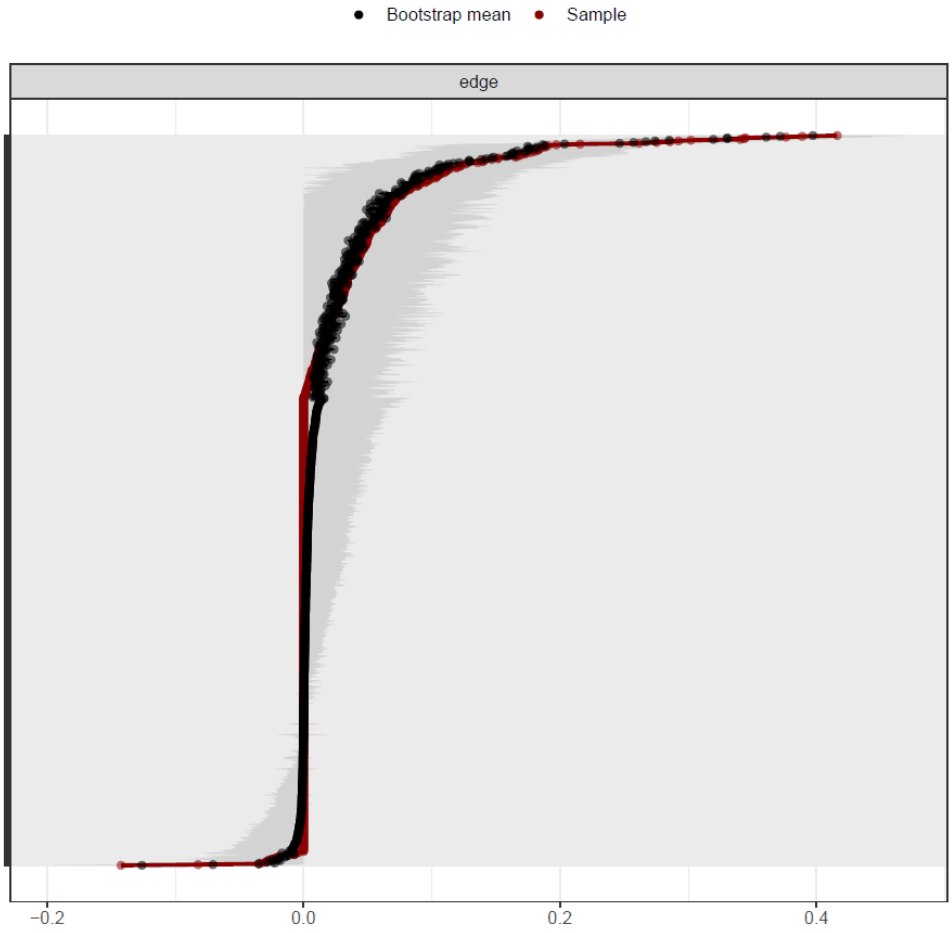


Figure S3-1C. *Bootstrapped 95%-CIs for the ER-COO network.*



S3-2. Non-parametric bootstrapped difference tests for edge weights

Figure S3-2A. *Edge weight difference test for the ER-DEP network.*

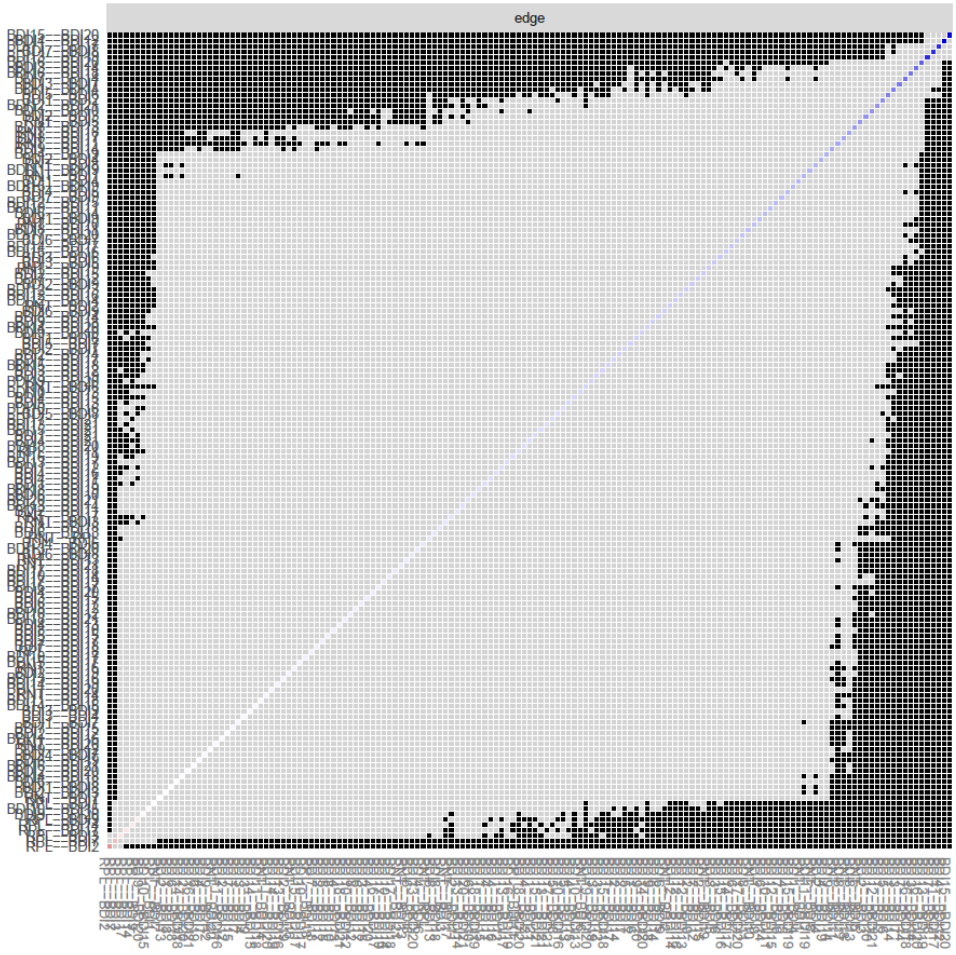


Figure S3-2B. *Edge weight difference test for the ER-ANX network.*

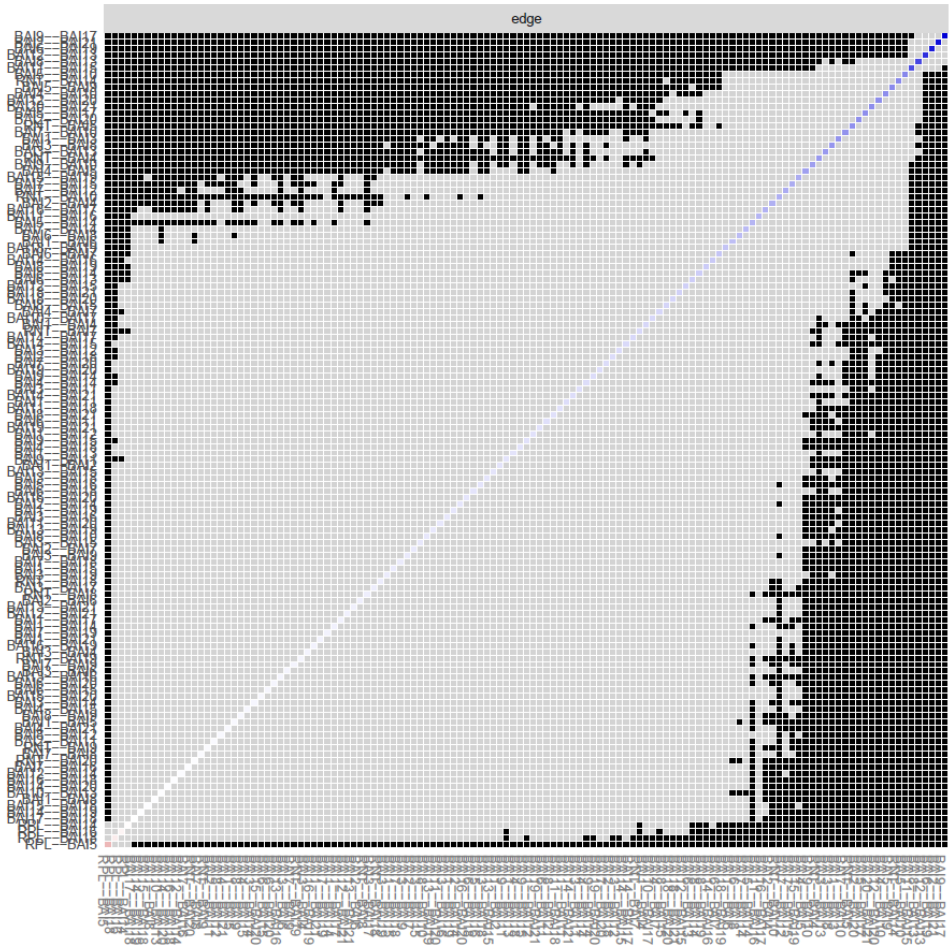
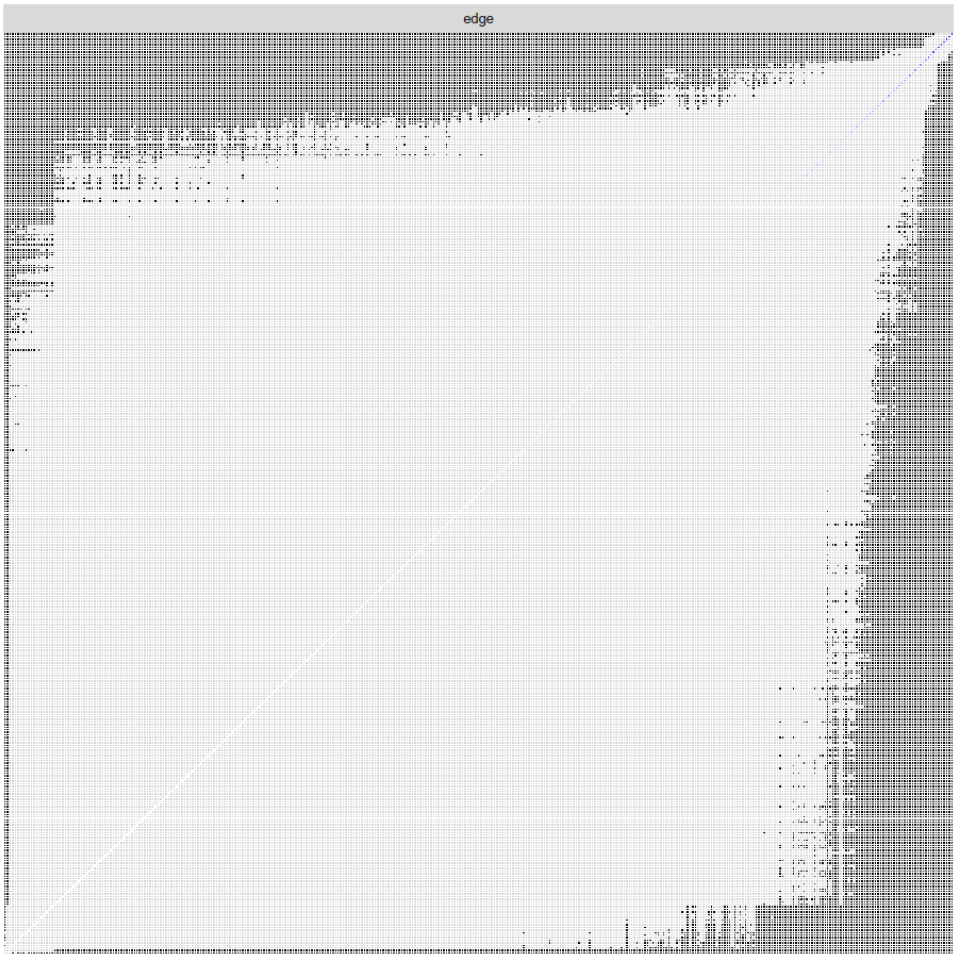


Figure S3-2C. *Edge weight difference test for the ER-COO network.*



S3-3. Person-dropping bootstrapped estimates of expected influence

Figure S3-3A. *Expected influence stability for the ER-DEP network.*

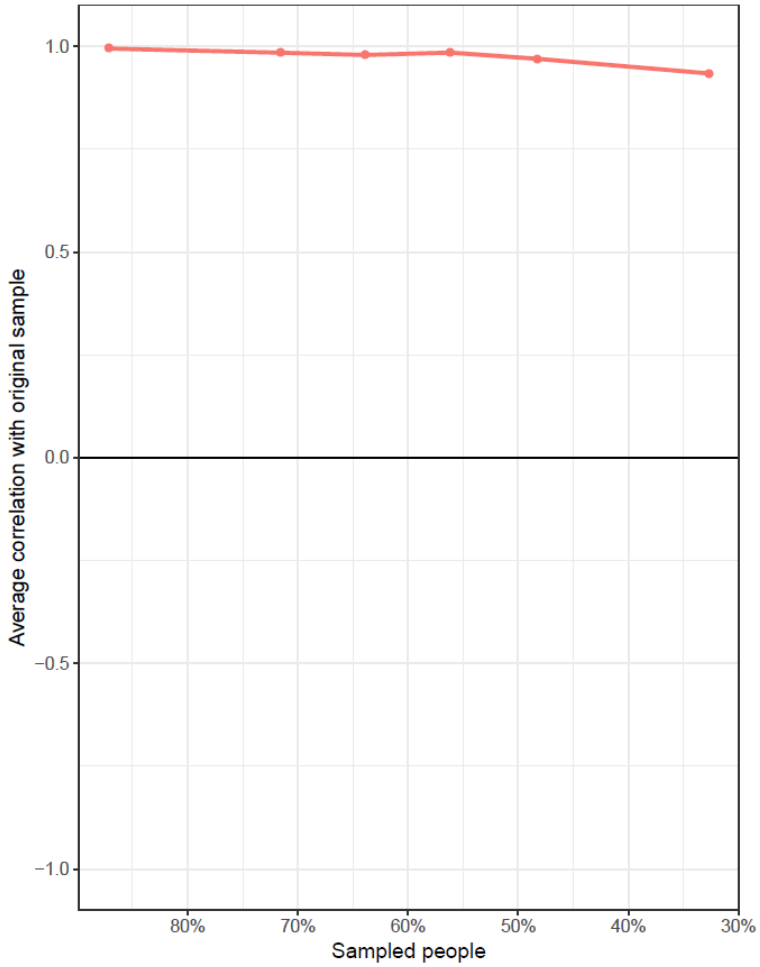


Figure S3-3B. *Expected influence stability for the ER-ANX network.*

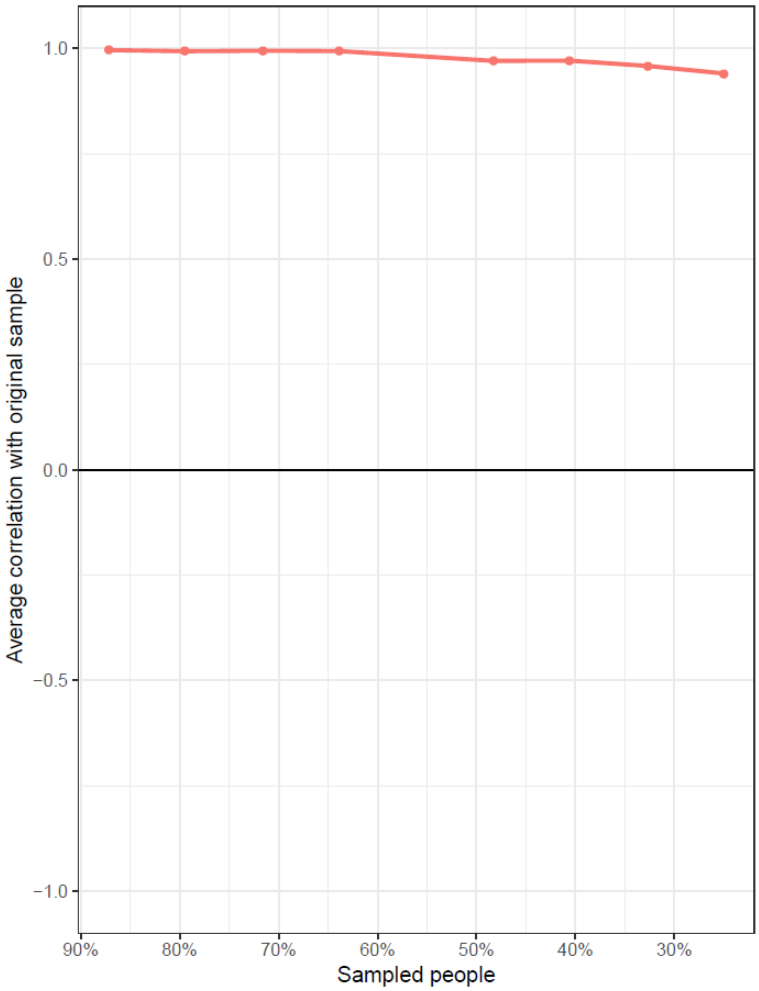
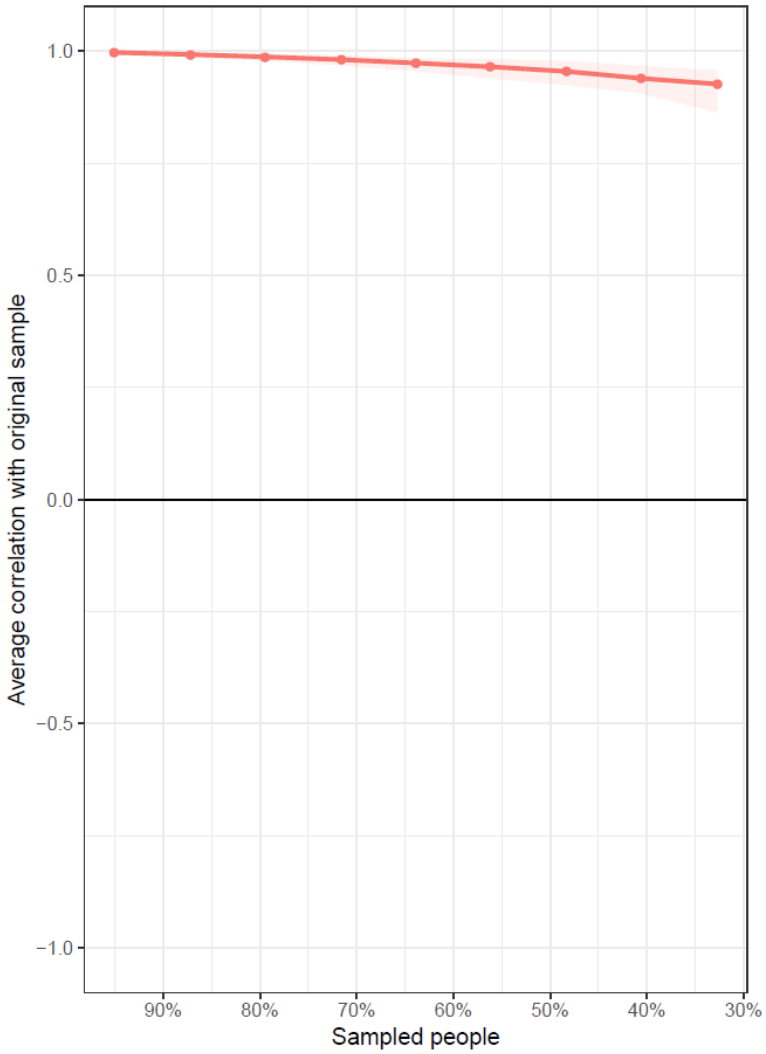


Figure S3-3C. *Bridge expected influence stability for the ER-COO network.*



S3-4. Non-parametric bootstrapped centrality difference tests

Figure S3-4A. *Expected influence difference test for the ER-DEP network.*

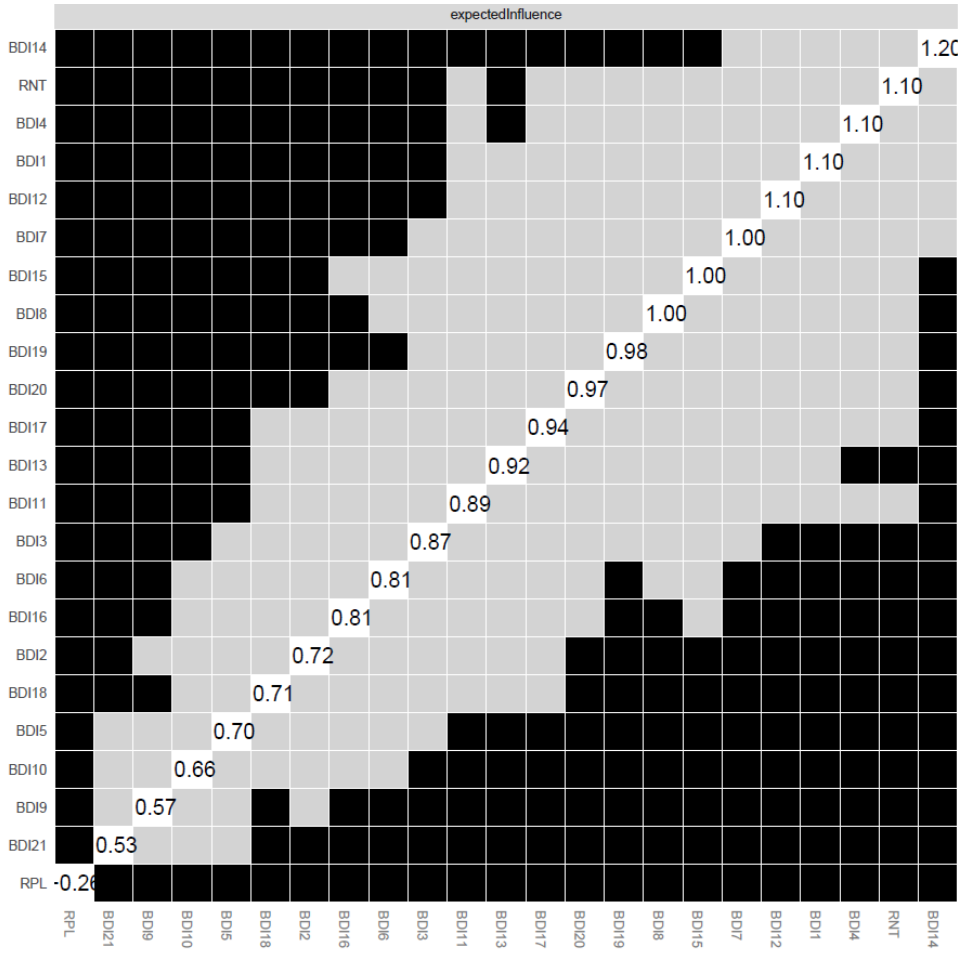


Figure S3-4B. *Expected influence difference test for the ER-ANX network.*

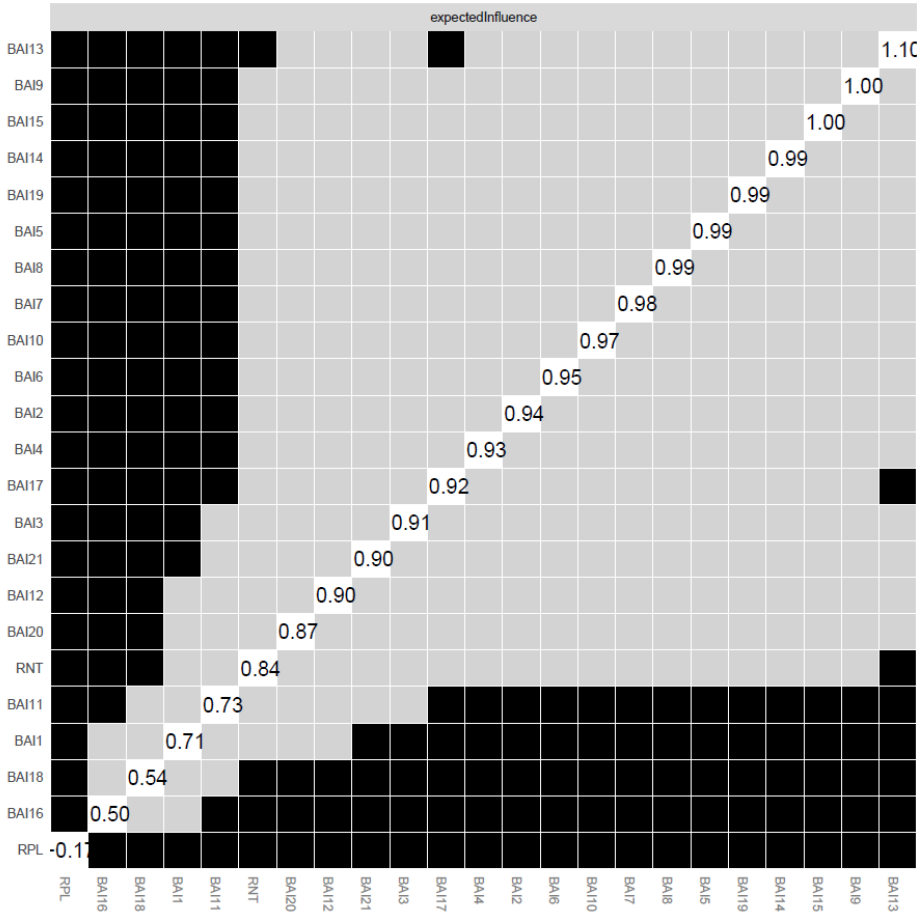
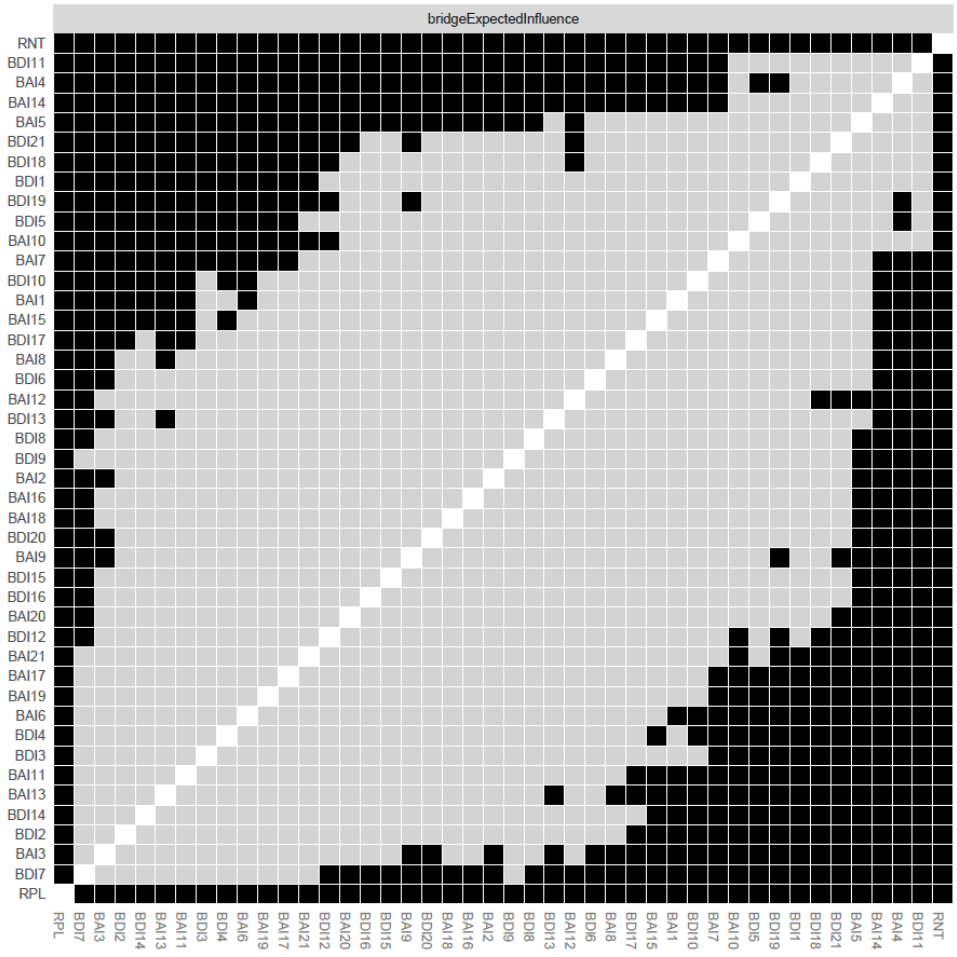


Figure S3-4C. *Bridge expected influence difference test for the ER-COO network.*



Supplement 4: Correlation matrices

See the appended file titled “CorMat_ER_DEP_ANX_COO_networks.xlsx” for the correlation matrices for each of the networks.

Supplement 5: R code

```
#-----------------------------------------------------
# Network estimation and inference
#-----------------------------------------------------

## 1. ER-DEP network

groups <- list('Emotion Regulation'=c(1,2), 'Depressive symptoms'=c(3:23))
CM_ERItemsBDI<-cor(dataBDI, method="spearman")
pcorERBDI <- qgraph(CM_ERItemsBDI, labels=colnames(dataBDI), groups=groups, graph = "glasso", tuning =
0.5, layout = "spring", sampleSize = nrow(dataBDI), legend.cex = 0.4, vsize = 5, esize = 15, palette = 'pastel',
posCol = "#003399", negCol = "#FF9933", borders = T)

cntrplt_ERDEP<-expectedInf(pcorERBDI)
plot(cntrplt_ERDEP)

## 2. ER-ANX Network

groups <- list('Emotion Regulation'=c(1,2), 'Anxiety symptoms'= c(3:23))
CM_ERItemsBAI<- cor(dataBAI, method="spearman")
pcorERBAI <- qgraph(CM_ERItemsBAI, labels=colnames(dataBAI), groups=groups, graph = "glasso", layout =
"spring", sampleSize = nrow(dataBAI), legend.cex = 0.5, vsize = 5, tuning = 0.5, esize = 15, palette = 'pastel',
posCol = "#003399", negCol = "#FF9933", borders = T)

cntrplt_ERANX<-expectedInf(pcorERBAI)
plot(cntrplt_ERANX)

## 3. ER-COO network

groups <- list('Emotion Regulation'=c(1,2), 'Depressive symptoms'=c(3:23), 'Anxiety symptoms'=c(24:44))
CM_ERItemsBDIBAI<- cor(dataBDIBAI, method="spearman")
pcorERBDIBAI <- qgraph(CM_ERItemsBDIBAI, labels=colnames(dataBDIBAI), groups=groups, graph =
"glasso", layout = "spring", sampleSize = nrow(dataBDIBAI), legend.cex = 0.5, vsize = 5, tuning = 0.5, esize =
15, palette = 'pastel', posCol = "#003399", negCol = "#FF9933", borders = T)

b<-bridge(pcorERBDIBAI, communities = list("Comm1"=c(1:2), "Comm2"=c(3:23), "Comm3"=c(24:44)))
plot(b, include =c("Bridge Expected Influence (1-step)"), zscore=F)

#-----------------------------------------------------------
# 2. Network stability
#-----------------------------------------------------------

## 1. ER-DEP network

npnBoot_ERDEP <- bootnet(dataBDI, default="huge", tuning = 0.5, nBoots = 1000, nCores = 8, type =
"nonparametric", statistics = "all")
pdBoot_ERDEP <- bootnet(dataBDI, default=" huge", nBoots = 1000, nCores = 8, type="case", statistics="all")

### Bootstrapped 95% CIs around the edge weights
plot(npnBoot_ERDEP, labels = F, order = "sample")

### Edge weight difference test
plot(npnBoot_ERDEP, "edge", plot = "difference", onlyNonZero = TRUE, order = "sample")
```

```
### Centrality difference test
plot(npnBoot_ERDEP, "expectedInfluence", plot = "difference")

### Plot centrality stability
plot(pdBoot_ERDEP, "expectedInfluence", labels = T, order = "sample")

### Compute CS coefficient
CorStabCG<-corStability(pdBoot_ERDEP, statistics=c('expectedInfluence'))

## 2. ER-ANX network

npnBoot_ERANX <- bootnet(dataBAI, default=" huge", tuning = 0.5, nBoots = 1000, nCores = 8, type =
"nonparametric", statistics = "all")
pdBoot_ERANX <- bootnet(dataBAI, default=" huge", nBoots = 1000, nCores = 8, type="case", statistics="all")

### Bootstrapped 95% CIs around the edge weights
plot(npnBoot_ERANX, labels = F, order = "sample" )

### Edge weight difference test
plot(npnBoot_ERANX, "edge", plot = "difference",  onlyNonZero = TRUE, order = "sample")

### Centrality difference test
plot(npnBoot_ERANX, "expectedInfluence", plot = "difference")

### Plot centrality stability
plot(pdBoot_ERANX, "expectedInfluence")

### Compute CS-coefficient
CorStabCG<-corStability(pdBoot_ERANX, statistics=c('expectedInfluence'))

## 3. ER-COO network

npnBoot_ERCOO <- bootnet(dataBDIBAI, default="huge", tuning = 0.5, nBoots = 1000, nCores = 8, type =
"nonparametric", statistics = "all", communities = list("Comm1"=c(1:2), "Comm2"=c(3:23),
"Comm3"=c(24:44)))
pdBoot_ERCOO <- bootnet(dataBDIBAI, default="huge", tuning = 0.5, nBoots = 1000, nCores = 8, type =
"case", statistics = "bridgeExpectedInfluence", communities =  list("Comm1"=c(1:2), "Comm2"=c(3:23),
"Comm3"=c(24:44)))

### Bootstrapped 95% CIs around the edge weights
plot(npnBoot_ERCOO, labels = F, order = "sample" )

### Edge weight difference test
plot(npnBoot_ERCOO, "edge", plot = "difference",  onlyNonZero = TRUE, order = "sample", labels=F)

### Centrality difference test
plot(npnBoot_ERCOO, "bridgeExpectedInfluence",, plot = "difference",labels = T, order = "sample" )

### Plot centrality stability
plot(pdBoot_ERCOO, statistics = "bridgeExpectedInfluence")

### Compute CS-coefficient
CorStabCG<-corStability(pdBoot_ERCOO, statistics=c('bridgeExpectedInfluence'))
```