Research paper

Symptoms of social anxiety disorder and major depressive disorder: A network perspective

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ABSTRACT

Background: We used network analyses to examine symptoms that may play a role in the co-occurrence of social anxiety disorder (SAD) and major depressive disorder (MDD). Whereas latent variable models examine relations among latent constructs, network analyses have the advantage of characterizing direct relations among the symptoms themselves.
Method: We conducted network modeling on symptoms of social anxiety and depression in a clinical sample of 130 women who met criteria for SAD, MDD, both disorders, or had no lifetime history of mental illness.
Results: In the resulting network, the core symptoms of social fear and depressed mood appeared at opposite ends of the network and were weakly related; so-called "bridges" between these symptoms appeared to occur via intervening variables. In particular, the worthless variable appeared to play a central role in the network.
Limitations: Because our data were cross-sectional, we are unable to draw conclusions about the direction of these effects or whether these variables are related to each other prospectively.
Conclusions: Continued testing of these pathways using longitudinal data will help facilitate the development of more effective clinical interventions for these disorders.

Social anxiety disorder (SAD) and major depressive disorder (MDD) are among the most prevalent disorders worldwide (Kessler et al., 2005), and investigators have documented their high rate of co-morbidity (Beesdo et al., 2007; Brown et al., 2001; Kessler et al., 2005; Ohayon and Schatzberg, 2010; Stein et al., 2001).[4] Further, these disorders have been found to be associated with greater impairment when they co-occur than when either occurs alone (Norton et al., 2008). Despite these findings, however, we have only a limited understanding of the nature of the comorbidity of these disorders (Langer and Rodebaugh, 2014).

Traditionally, comorbidity of psychological disorders has been conceptualized as arising from a relation between the latent constructs of the disorders (e.g., Brown et al., 1998). Further, latent variable models are typically used under the theoretical and statistical assumption that any apparent relation between symptoms or items occurs because they are both indicators of the same underlying construct (Holland and Rosenbaum, 1986; Lord, 1953; McDonald, 1981), and not, for example, because a symptom such as *trouble sleeping* leads directly to the symptom of *fatigue*. Yet, it is likely that symptoms such as *trouble sleeping* and *fatigue* do have direct causal relations (Cramer et al., 2012). This assumption of symptom independence may contribute to the difficulties researchers often encounter when trying to fit the theorized

simple latent variable models to data (e.g., McCrae et al., 1996), such as poor fit and indicators that have nontrivial loadings on secondary factors (Church and Burke, 1994; McCrae et al., 1996; Savla et al., 2007). Given the theoretical limitations and the fit difficulties associated with latent variable models, it is important to examine alternative conceptualizations and techniques for investigating comorbidity.

Recently, models have been developed that account for direct symptom relations and conceptualize them as interacting elements, or *nodes*, within a causal network (Borsboom, 2008). These network models can account for relations between disorders on a symptom level and offer the potential to identify symptoms that *bridge* the relation between two disorders. A *bridge symptom* can be conceptualized as a stepping-stone in a pathway from one disorder to another; the presence of this symptom increases the likelihood that an individual will develop the secondary disorder. For example, researchers have found that when they modeled the symptoms of Generalized Anxiety Disorder (GAD) and MDD with a network approach, the symptoms of trouble sleeping and fatigue formed a bridge between the disorders (Borsboom and Cramer, 2013). Despite the advantages of using network models, research utilizing these approaches is in its early stages. In the context of the present paper, there is only one published paper examining symptoms of social anxiety and depression from a network perspective (Heeren et al., 2018).

Examining the comorbidity of SAD and MDD is important because we do not yet know which factors account for their significant association. Various pathways or moderating variables have been posited (Akiskal, 1990; Kessler et al., 1999; Mineka et al., 1998; Moitra et al., 2008; Swendsen, 1997), including positive affect (PA) and behavioral avoidance. The evidence supporting any one theory, however, is limited. Network analyses might help to clarify these potential pathways between the disorders by suggesting causal symptom relations. For example, one such pathway might begin with a person who becomes socially fearful, then starts avoiding social situations, and then develops a depressed mood as a result of the social isolation. In this example, social avoidance forms a bridge between the symptoms of social fear and depressed mood. Notably, latent variable analyses by design would typically obscure any such symptom pathways because they typically would not allow for direct symptom-level relations. Network analysis can also be informative in terms of the overall shape of the network, which can convey whether, for example, certain symptoms cluster together or are more diffuse.

To our knowledge, there are only two other examinations of a network model consisting of symptoms related to social anxiety and depression (Heeren et al., 2018; Piccirillo et al., 2018). Notably, the results of Piccirillo et al. discussed below are preliminary because that study has not been published and is still in preparation. Heeren et al. (2018) examined a network of social anxiety and depression symptoms in sample of participants with a primary diagnosis of SAD (a subset of the participants also met criteria for MDD). They found that symptoms of depression (items from the BDI-II) and symptoms of social anxiety (items from the Liebowitz Social Anxiety Scale; Liebowitz, 1987) tended to cluster together with some nodes showing strong relations with the network as a whole and with symptoms of the other disorder (i.e., some depression symptoms showed strong relations with social anxiety symptoms and vice versa). Of note, worthlessness was one of the depression items that was strongly connected to the network as a whole. Additionally, avoidance of participating in small groups was strongly connected to several depression symptoms, including worthlessness and loss of pleasure, suggesting that certain types of social avoidance may serve as a bridge from social anxiety symptoms to depression symptoms. Though Piccirillo et al. did not focus primarily on identifying bridge symptoms, their networks, which were based on a large data set of participants from mostly undiagnosed samples, suggest that measures of affect and personality serve as bridges between symptoms of social anxiety and depression. Taken together these findings provide some initial information about symptom level relations

relevant to MDD and SAD comorbidity. Whereas the findings from these previous samples are informative, no previous study has examined these relations in a sample recruited to capture all combinations of SAD and MDD comorbidity.

To address this gap in the literature, we examined a network of SAD and MDD symptoms in a dataset of women with all potential combinations of SAD and MDD symptoms: low and high levels of SAD symptoms fully crossed with low and high levels of MDD symptoms. This dataset ensures that the results of modeling cannot be attributed to the lack of a particular configuration of symptoms. The sample was restricted to women given the higher prevalence rates of mood and anxiety disorders in women than in men (Kessler et al., 1994). In addition to including variables that reflect diagnostic criteria, we selected variables based on theory regarding the relation between SAD and MDD, as well as on-going network analyses of depression and social anxiety (Piccirillo et al., 2018). Symptoms of social fear, social avoidance, depressed mood, PA, feelings of worthlessness, and mood instability formed the nodes within our network analyses. We selected hallmark symptoms of each disorder (American Psychiatric Association, 2013), including social avoidance, social fear, and depressed mood. We included PA based on prior evidence that it is related to both SAD and MDD and may be an intervening variable between SAD and MDD (Brown et al., 2007; Costa and McCrae, 1980; Joiner and Coyne, 1999; Rodebaugh, 2009; Schneier et al., 1994; Vittengl and Holt, 2000).

Finally, based on previous theory (Langer and Rodebaugh, 2014) and data (Piccirillo et al., 2018), we selected two additional variables that may play a bridge role or be a common vulnerability between SAD and MDD (Farmer and Kashdan, 2013; Thompson et al., 2012; Thompson et al., 2017). First, based on a formulation that feelings of inferiority serve as a common vulnerability between MDD and SAD (Langer and Rodebaugh, 2014), we included a variable representing feelings of worthlessness. Inclusion of worthlessness is also supported by Heeren et al.'s finding that this variable was strongly connected to a network of social anxiety and depression symptoms. Second, based on research suggesting that individuals with MDD or SAD have increased instability of negative affect (Farmer and Kashdan, 2013; Thompson et al., 2012; Thompson et al., 2017), we included a composite variable representing mood instability.

Given our theory that comorbidity between SAD and MDD is explained in part by shared relations with intermediary symptoms, we hypothesized that nodes representing social anxiety symptoms and depressive symptoms would be bridged by one or more intervening variables such as social avoidance or lower PA. As discussed above, a pathway involving social avoidance and lower PA is plausible given that both are likely to be consequences of social anxiety and to lead to depression.

1. Method

1.1. Participants

The sample for the current study came from a clinical study of women with diagnoses of MDD and SAD. Although the data from this sample are ideal to conduct network models analyzing social anxiety and depression symptoms, they were not originally collected for this purpose. Individuals were recruited from the community around Stanford, California, through advertisements posted online and at local agencies and businesses, to participate in a large study (i.e., Thompson et al., 2017). Participants were 130 women between 18 and 59 years of age ($M = 35.6$ years, $SD = 12.2$). Individuals were eligible to participate if they met eligibility criteria for one of four groups: (a) a current diagnosis of MDD (MDD group; $n = 35$); (b) a current diagnosis of generalized SAD (SAD group; $n = 31$); (c) current diagnoses of generalized SAD and MDD (SAD-MDD group; $n = 26$); or (d) no current or past history of any Axis I disorder (control group; $n = 38$). General

eligibility requirements included fluency in English, US citizen or green card, and age between 18 and 60 years. Exclusion criteria included a history of severe head injury or learning disorder, hearing impairment, or alcohol or substance abuse/dependence in the past 6 months. Two previous studies have examined subsets of the data stemming from the same overarching study (Kircanski et al., 2015; Thompson et al., 2017) but neither study was related to examining network relations.

Individuals were administered the *Structured Clinical Interview for DSM-IV Axis I Disorders* (SCID-I) by interviewers who had received extensive training. Inter-rater reliability kappas for a random subset of 15 interviews ranged from 0.92 to 1.0 for the above diagnoses.

1.2. Measures

Social Phobia and Anxiety Inventory (SPAI; Turner et al., 1989) is a 45-item measure containing 32 items that assess social anxiety symptoms across a range of social situations. Eleven items include separate ratings depending on the people with whom participants are interacting (strangers, authority figures, opposite sex, people in general). For these items, the ratings are averaged across the four different types of people. The SPAI has good test-retest reliability over 2 weeks, good internal consistency, and good ability to distinguish between individuals with and without social anxiety disorder (Turner et al., 1989). In the current study, internal consistency was high for the social phobia items ($\alpha = 0.99$).

NEO-Five Factor Inventory (NEO-FFI; Costa and McCrae, 1992) is a shortened version of the NEO Personality Inventory Revised that assesses the five basic dimensions of personality. Only neuroticism and extraversion were assessed in the current study (24 items). The NEO-FFI shows good internal consistency (Costa and McCrae, 1992). In the current study, internal consistencies for the extraversion ($\alpha = 0.87$) and neuroticism ($\alpha = 0.90$) subscales were high.

Beck Depression Inventory 2 (BDI-2; Beck et al., 1996) is a self-report measure containing a series of 21 groups of statements describing depressive symptomatology. Each symptom is rated based on endorsement of one of a series of statements arranged in order from least to most symptomatic. The BDI-2 has been validated in psychiatric and non-psychiatric samples (Steer et al., 1999). In the current study, internal consistency was excellent ($\alpha = 0.95$).

1.3. Procedure

1.3.1. Item selection

All items were standardized and, in the cases in which a node contained two items, the items were summed to form the node variable. Our decisions concerning how to combine items to form a node were made prior to conducting any network analysis and were based on theory about which constructs would be relevant to include in our network to best examine symptoms that might bridge the relation between social anxiety and depression. Further, combining items is a clear way to increase reliability of nodes and, thus, the replicability of networks.

1.3.2. Social fear and social avoidance

For the social fear node we selected two items from the SPAI: item 14 (*I feel anxious and I do not know what to do when in an embarrassing situation with …*) and item 22 (*I feel anxious when speaking in front of…*). Two items were combined because we wanted to capture both interaction and performance fears given that these are considered to be the two broad types of fears for individuals with SAD. We chose these particular items because they are included in a short version of the SPAI (the SPAI-18) that has strong psychometric properties and have been shown to load strongly on correlated factors representing distress in social situations and focus of attention in previous factor analyses (Osman et al., 1995; de Vente et al., 2013). For the social avoidance node, we selected item 7 from the SPAI (*I feel so anxious about attending*

social gatherings that I avoid these situations) because it was included as one of two items that loaded on an avoidance and escape factor in analyses of the SPAI-18 (de Vente et al., 2013) and had the stronger face validity of these two items.

Depressed mood was assessed with item 1 from the BDI-2, which consists of a ranking of how much sadness the respondent has been experiencing. We chose this particular item to capture depressed mood because it is the only item on this scale that assesses a low or down mood and we wanted to isolate depressed mood from other symptoms of depression. This item asks respondents to rate, on a 4-point scale, their level of sadness over the past 2 weeks.

Positive affect was assessed with two extraversion items from the NEO-FFI: item 4 (*I laugh easily),* and item 16 (*I am a cheerful, high-spirited person*). We selected these items because we wanted to capture positive emotionality rather than sociability as some of the other extraversion items do. Of the NEO-FFI items, these were the two that concerned positive emotionality.

1.3.3. Feelings of worthlessness (worthlessness)

We selected the item from the BDI-2 that assesses feelings of worthlessness (item 14). Respondents rate, on a 4-point scale, their level of feeling worthless over the past 2 weeks.

1.3.4. Mood instability (Moody)

We selected item 5 from the NEO-FFI (*When I'm under a great deal of stress, sometimes I feel like I' m going to pieces*) and item 17 from the BDI-2, which assesses irritability. We selected these items because they appeared to have the strongest face validity in terms of capturing difficulties with instability of negative mood. Notably, however, in our final network these items were modeled separately; see below.

1.4. Data analytic overview

Partially missing data occurred in 14 out of 130 participants (10.8%) and was addressed with multiple imputation using the missforest package in R (R Development Core Team, 2012), resulting in five multiply-imputed data sets. Network models were constructed in R using the qgraph package (Epskamp et al., 2012) and then network properties were obtained using the bootnet package (Epskamp et al., 2017). Because, to our knowledge, no procedure exists that handles network models generated from multiply-imputed data, we created and compared five network models.

Using bootnet, we first aimed to test the stability and accuracy of our five networks before proceeding with analysis in accordance with guidelines described by Epskamp et al. (2017). We used bootnet to create 1,000 nonparametric bootstrapped samples to first assess the accuracy of edges (connections between nodes) by allowing us to construct bootstrapped 95% confidence intervals around edge estimates. We interpreted wide bootstrapped confidence intervals around edge estimates as indicating greater variability (i.e., lower accuracy) and as being less likely to differ from other edges. We also used a case-dropping bootstrap in which a percentage of participants were removed at random and their centrality indices were correlated with the original sample to compute a stability coefficient. Simulations studies suggest that values above 0.5 are preferable for interpretation (Epskamp et al., 2017).

Following accuracy and stability analyses, we estimated a network for each imputation. Network models examine relations between nodes in a graphical format. The visual representation of a network allows researchers to investigate the overall structure and pattern of a group of symptoms in a way that would be much more difficult using traditional methodologies (Cramer et al., 2012). The glasso algorithm with EBIC model selection was used to generate the gaussian graphical networks models (GGMs) used for analysis. The network models generated are based on partial correlations between the nodes, allowing the researcher to examine the relation between two nodes with relations with

all other nodes partialled out. The glasso algorithm applies a penalty function that reduces any weak relations to zero to avoid spurious relations given the number of parameters estimated. The nodes are connected to each other by lines (i.e., edges). The weight of the edge represents the strength of the relation conditional on all other possible relations, and the color of the line represents whether it is a positive (blue, solid) or negative (red) relation. The interpretation of a network analysis comes from examining multiple features of the network, including the overall shape of the network, the distance between various nodes, the strength of the connections, and how close a node is to the center of the network. In addition to visual inspection, we used qgraph to calculate the following centrality indices: betweenness, closeness, and strength (Freeman, 1979; Opsahl et al., 2010). Betweenness measures the extent to which a node tends to funnel or control travel among all the other nodes and represents the number of shortest paths with which a node is involved. Closeness represents the inverse of the sum of distances to all other nodes. Strength captures the strength of the relations that a node has with other nodes and represents the sum of the weights of the relations with which a node is involved.

Finally, we used the *expectedinf* function from the R package *networktools* (Jones, 2017) to determine the one-step (EI1) and two-step (EI2) influence of each node. The expected influence index was first described by Robinaugh et al. (2016) and allows for an assessment of node influence that accounts for both positive and negative values of edge weights rather than absolute values. EI1 accounts for the summed edge weights of each node's directly connected neighbors and EI2 additionally accounts for the edge weights, or EI1, of those neighbors. Although both metrics (and particularly EI1) highly overlap with the strength centrality index, the expected influence metric allows us to take negative edges in the network into consideration when determining how that node might influence the rest of the network. In our proposed symptom network, positive affect is expected to have negative relationships with other symptom nodes because lower scores on this trait measure reflect psychopathology, whereas higher scores on all other traits indicate worsening symptoms.

2. Results

2.1. Participant symptom levels by group

Participant symptom levels by diagnostic group are presented in Table 1.

2.2. Network stability and accuracy

Using guidelines suggested by Epskamp et al. (2017), we determined that network stability was too low to confidently interpret results in our original 6-node model ($rs < 0.362$). We investigated other possible structures for the network, including a nine-node model in which none of the nodes were composited and a seven-node network in

Table 1
Mean and standard deviations (SD) of social anxiety, depression, and affect by diagnostic group.

	Social anxiety	Depression	Neuroticism	Extraversion
SAD group ($n = 31$)	130.67 (24.69)	13.00 (8.24)	3.57 (0.66)	2.91 (0.55)
MDD group ($n = 35$)	86.46 (32.04)	27.76 (9.80)	3.65 (0.56)	2.85 (0.69)
SAD-MDD group ($n = 26$)	145.13 (17.58)	33.79 (11.69)	4.05 (0.60)	2.52 (0.55)
Control group ($n = 38$)	44.74 (23.44)	2.05 (3.26)	2.21 (0.69)	3.60 (0.53)

Note. Mean totals by group with standard deviations in parentheses. SAD = Social Anxiety Disorder; MDD = Major Depressive Disorder

Table 2
Average (range across imputations) correlation stability indices for six and seven node network models.

	Strength	Betweenness	Closeness
6-node model	0.32 (0.21 - 0.36)	0.00 (0.00 - 0.00)	0.15 (0.13 - 0.21)
7-node model	0.45 (0.44 - 0.52)	0.01 (0.00 - 0.05)	0.28 (0.21 - 0.36)

Note. The nodes included in the six-node model include: social avoidance, depressed, moody, worthless, positive affect, and social fear. The nodes included in the seven-node model include: social avoidance, depressed, moody, irritable, worthless, positive affect, and social fear.

which moody was separated into two separate nodes (the NEO-FFI item [moody node] and the irritability item from the BDI-2 [irritable node]). We chose moody as the composited node to separate given the relatively low correlation between items ($r = 0.326$, CI[.163–0.472]). Both the nine- and the seven-node models resulted in improved stability indices; however, we used the seven-node model for subsequent analysis due to similarity to our initially theorized model. Stability indices for our final model ranged from 0–0.046 for betweenness; 0.208–0.362 for closeness; and 0.438–0.515 for strength, indicating that the closeness and strength are reasonably stable and, thus, interpretable. Centrality stability indices for the six and seven node models are depicted in Table 2.

In addition to examining accuracy, we performed a bootstrapped difference test of edges and the strength and closeness centrality indices using bootnet. Both the depressed-worthless edge and social avoidance-social fear edge were significantly stronger than 75–80% of the other edges in the network across imputations.

2.3. Network visualization and interpretation

The visualization of our final seven-node network model averaged across the five imputed datasets is presented in Fig. 1. The position of the nodes and their distance from each other is calculated in qgraph and node positions are averaged across the five imputed data sets (Epskamp et al., 2012). As shown in Fig. 1, the strongest relations (indicated by bolder lines and close proximity) are between social avoidance and social fear, and between worthless and depressed mood. As stated above, these edges were also found to be significantly stronger than the majority of other edges in the network according to results of the bootstrapped difference test. In terms of the overall shape of the network, there appears to be a cluster of depression symptoms that includes depressed mood, worthless, and irritability, as well as a cluster of social anxiety symptoms (fear and avoidance). The positive affect node appears furthest from the rest of the nodes. The social fear node and the depressed mood node appeared at opposite ends of the network and also had an edge estimated at 0 in all imputations. Thus, travel between these two hallmark symptoms of SAD and MDD, respectively, could only occur via the other variables.

We hypothesized that there would be social avoidance and positive affect edges that would signify the roles these intermediary symptom nodes play as bridges between social fear and depressed mood. To support that hypothesis, we would have expected to find that the only path between the social fear and depressed nodes would appear via strong depressed-positive affect, positive affect-social avoidance, and social avoidance-social fear edges. As hypothesized, the connection between social fear and depressed nodes were estimated at 0; however, the wide confidence intervals around the positive affect-social avoidance edge relation suggested that the estimated relation might not be accurate. In addition, both the positive affect and social avoidance nodes appeared peripherally in the networks, and results of a difference test of centrality for these nodes did not differ significantly from any other node other than worthless, which was significantly more central. Instead of our initially hypothesized bridge, we found support for a
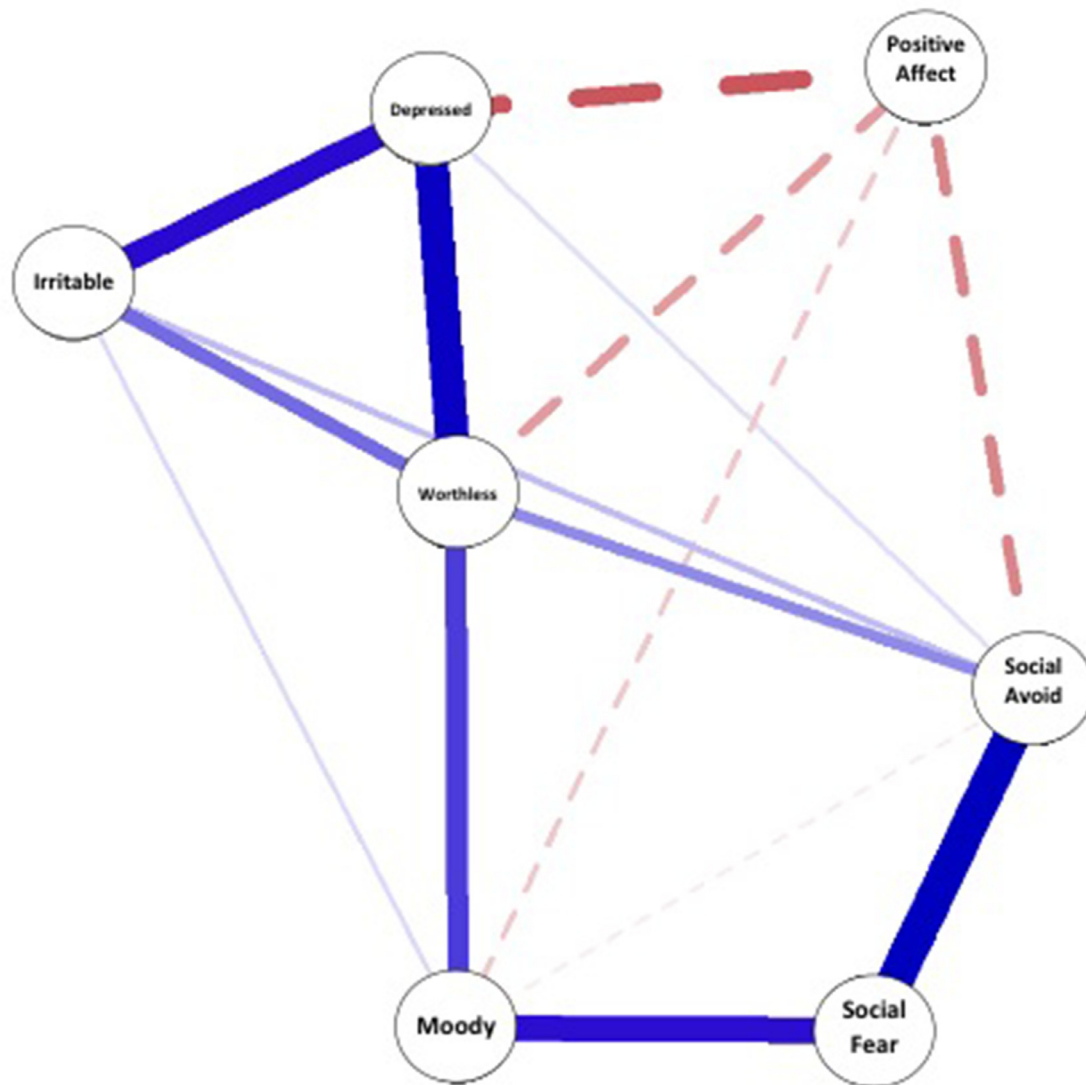
Fig. 1. Network model of social anxiety and depression symptoms. The figure represents the network averaged across the five imputations. The red dashed lines represent negative relations, whereas the blue solid lines represent positive relations. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 3.
Average Centrality indices across imputations for the nodes included in the final (i.e., seven-node) model.

	Strength	Betweenness	Closeness
Social avoidance	0.83	1.00	0.02
Depressed	1.01	7.00	0.03
Moody	0.70	3.00	0.03
Irritable	0.62	0.00	0.02
Worthless	1.12	7.00	0.03
Positive affect	0.56	0.00	0.02
Social fear	0.77	1.00	0.02

Note. The range of the centrality indices for each node across each of the imputed datasets were largely consistent and differed on the order of the hundredths decimal.

worthlessness bridge node: worthlessness, via a moody-worthless edge, appeared to serve as a bridge between social fear and depressed mood symptom nodes. Closeness centrality for the worthless node was significantly higher than irritability and social fear in all imputations (Table 3), and also higher than positive affect in three of the five imputations, indicating that paths between this node and other nodes

tended to be shorter. Notably, the depressed-worthless, worthless-moody, and moody-social fear edges were among the strongest across imputations.

Finally, we examined EI1 and EI2 values. EI1 indicated that positive affect (-2.22 to -2.23), followed by worthlessness (2.02–2.05) had the greatest expected influence on neighboring nodes across imputations. However, EI2 values indicated that worthlessness (7.23–7.43) followed by depressed (6.86–7.04) were the nodes with the greatest influence on the network. Taken together, although increases in positive affect may have a noticeable direct impact on neighboring nodes, worthlessness was more consistently influential on immediately neighboring nodes and nodes one step removed, suggesting broader network impact. Overall, our findings suggest a cluster of social anxiety symptoms and a cluster of depression symptoms, with the symptom of worthlessness forming the strongest pathway between social fear and depressed mood. In addition, the worthless node appeared at the center of the network, had the closest (i.e., shortest) relations with the other nodes, and had a noteworthy direct and indirect impact on the network.

2.4. Post-hoc analyses

Although we hypothesized specific nodes as being representative of certain communities (e.g., social avoidance as a symptom of social anxiety and moody as a symptom of depression), it is possible that symptom communities and the symptoms that bridge those communities do not exist as hypothesized. Consequently, we conducted a spinglass test using the function from the igraph package (Csardi and Nepusz, 2006) and a test of one- and two-step expected influence bridges using a function from the network tools package (Jones, 2018). The spinglass test is a data-driven approach to community identification. We conducted the test 1,000 times in each of the five imputations and examined the results to determine whether there were reliable communities. We found three such communities: social avoidance, social fear, and moodiness formed one community; depressed, irritable, and worthless formed a second community; and positive affect formed a third community. We then used these communities in conducting the bridge expected influence test. Bridge expected influence operates similarly to expected influence metrics calculated in the primary analyses, but differs in that edges are summed among nodes within the same community and compared to the sum of edges among nodes in other communities. Both one-step influence, or the direct effect of a node on a different community, and two-step influence, the indirect effect of a node on different communities, were strongest for positive affect (-0.56 to -0.54 and -0.98 to -0.99, respectively). Our original results converge with findings from the one-step expected influence analysis absent communities; once communities were identified, however, our two-step expected influence test result differed. Whereas worthlessness was most influential in our original results, when worthlessness is considered as part of the same community as depression, positive affect emerges as the strongest bridge symptom among *different* communities.

3. Discussion

Despite extensive research documenting the high rate of comorbidity between SAD and MDD, we have only a limited understanding of how individuals move from experiencing one set of symptoms to another. That is, for example, given that individuals have developed SAD, what might cause them to also develop MDD? In this context, the goal of this study was to elucidate potential pathways between these two disorders. To that end, we created a network of SAD and MDD symptoms. Our results indicated that comorbidity between SAD and MDD plausibly stems from direct relations between associated (as opposed to hallmark) symptoms of both disorders. For example, the relation between social fear and depressed mood, the two hallmark symptoms of each disorder, was one of the weakest in the network, whereas the other symptoms played more central roles in the network.

Consistent with previous research (e.g., Moitra et al., 2008), we hypothesized that social avoidance plays a key role in bridging between the two disorders; however, we found little evidence for social avoidance as a bridge symptom. Instead, we found stronger evidence for the role of worthlessness as a bridge symptom given its position in the center of the network as well as its strength of relations with other nodes and its level of closeness centrality. We have previously theorized that people diagnosed with MDD and SAD share feelings of inferiority (Langer and Rodebaugh, 2014). Based on these analyses, it appears that feelings of worthlessness are strongly related to depressed mood but not to social fear. Thus, the bridge role of worthlessness appears to arise from strong relations between depressed mood and worthlessness, worthlessness and mood instability, and mood instability and social fear, rather than being a direct relation between worthlessness and social fear. We also found that the roles of mood instability and positive affect in the network were limited: Mood instability was not connected to depressed mood once we accounted for the other relations, and positive affect had strong connections but limited impact (although see

discussion of post-hoc analyses below). This finding seems at least superficially inconsistent with prior research suggesting that individuals with MDD have increased instability of negative affect (Farmer and Kashdan, 2013; Thompson et al., 2012; Thompson et al., 2017); however, because prior researchers did not include all of the elements that we included in our model, it is also possible that mood instability would have been accounted for by other factors in those studies as well had those additional factors been included.

Our overall approach was driven by *a priori* selection of items based on our theory regarding the relation between social anxiety symptoms and depression symptoms. Nevertheless, we recognize there are alternative approaches, including more data-driven methods such as testing for communities of symptoms. Findings from our post-hoc analyses suggested that positive affect was most likely to bridge a community of social anxiety symptoms with a community of depression symptoms. Absent a test that would allow us to directly compare the fit of these two models of communities (our original results being driven by theory and the post-hoc results being data-driven), we decided to prioritize our original findings given that they were based on *a priori* theory and hypothesis. There are also a number of limitations to these analyses that further lead us to interpret them with caution. First, the small number of nodes may have limited our tests for communities. In addition, once formed, the structure of the communities may have advantaged positive affect as the most likely node to be found as a bridge between the social anxiety and depression communities because it existed outside of those communities. That is, once worthlessness was assigned to the depression community, it had a lower chance of being found as a bridge symptom because edges within communities do not contribute to the expected influence. Second, the expected influence function and the bridge expected influence function are both fairly new functions that would benefit from additional empirical and simulated samples to better understand how to interpret these functions, particularly when few potential communities exist. Nevertheless, it is worth noting that this method suggests a stronger role of positive affect; this is an alternative hypothesis that should be tested in future work.

Notably, interpreting our results as applying to the development of comorbidity requires a series of assumptions that may not hold. Many researchers would interpret such findings as indicating that these constructs are ripe for further testing, but it is important to realize that there are reasons to be pessimistic about this interpretation, which requires that cross-sectional relations are similar to longitudinal relations, and that what is true across people is also true within people. Indeed, cross-sectional relations may tell us little of importance about longitudinal relations (Cole and Maxwell, 2003; Gollob and Reichardt, 1987; Maxwell and Cole, 2007; Maxwell et al., 2011). Similarly, there are reasons to doubt that the differences between people who are and who are not depressed correspond to the changes that a person would undergo in the process of becoming depressed (Molenaar, 2007; Molenaar, 2013). What can be said without making such assumptions is that the bridge symptoms we identified here are symptoms that people with both MDD and SAD are more likely to endorse than are people who have only one, or neither, of the disorders. Therefore, at a minimum, the bridge symptoms we observed in these data might be helpful in screening participants for being more likely to have the combination of MDD and SAD.

We should note several limitations of this study. First, as alluded to above, although we discuss potential routes between the disorders, our data were cross-sectional. Even if one is inclined to think that the cross-sectional relations identified in this study are consistent with longitudinal relations, the direction of those associations remains in question. Any of the potential pathways between social anxiety symptoms and depression symptoms could plausibly function as bi-directional loops, wherein one might start with social anxiety symptoms, develop depression symptoms, and then experience subsequent increases in social anxiety symptoms as a result of the depression symptoms and so on. This loop could also start in the other direction, with depression

symptoms. Second, although our sample size was adequate given the number of nodes we included in our analyses (Epskamp et al., 2017), researchers may consider building on our findings by including a greater number of symptoms in a larger sample. Third, our analysis of centrality of our nodes of interest was conducted using only closeness and strength due to the potential instability of estimated betweenness centrality. In addition, our results should be extended to samples that include men to test for possible gender effects. We discussed above a number of limitations related to our post-hoc results, including the fact that our testing of communities and bridges was limited by the relatively small number of nodes and communities. Future research on social anxiety and depression symptom communities and potential bridge symptoms will benefit from including more items/nodes representing each symptom.

The results of this study suggest that comorbidity between SAD and MDD plausibly stems from direct symptom-level relations. Should future longitudinal research replicate our finding concerning the role of worthlessness as a bridging symptom between depressed mood and social fear, treatments for MDD and SAD may benefit from a greater focus on these symptoms. For example, clinicians may see more efficient improvement in their clients who are diagnosed with both SAD and MDD by focusing on interventions designed to target symptoms that are central to the overall symptom network. Indeed, targeting a symptom that appears at the center of the network may facilitate reductions in symptoms of both disorders.

Limitations

We should note four limitations of this study. First, although we discuss potential routes between the disorders, our data were cross-sectional. Second, our sample size limited the number of symptom nodes included; researchers should build on our findings by including a greater number of symptoms. Third, our analysis of centrality of our nodes of interest was conducted using only closeness and strength due to the potential instability of estimated betweenness centrality. Finally, our results should be extended to samples that include men to test for possible gender effects.

Authors' contribution

All authors contributed to the study design. J.K.L., T.L.R., N.A.T, and M.P. performed the data analysis and interpretation, and all authors contributed to the writing of the manuscript. All authors approved the final version of the paper for submission.

Conflict of interest

There are no conflicts of interest.

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