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Research paper

Mapping network connectivity among symptoms of social anxiety and comorbid depression in people with social anxiety disorder

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ABSTRACT

Background: Social anxiety disorder (SAD) and depressive symptoms often covary. Yet, uncertainty still abounds vis-à-vis the individual symptom-to-symptom associations between these two disorders. Inspired by the network approach to psychopathology that conceptualizes comorbidity as a natural consequence arising from bridge symptoms that can transmit activation from one disorder to the other, we applied network analytic methods to characterize the associations among core symptoms of SAD—i.e. fear and avoidance of social situations—and comorbid depressive symptoms among 174 individuals with DSM-IV-TR criteria for SAD.

Methods: We first explored the general structure of these symptoms by estimating a regularized partial correlation network using the graphical LASSO algorithm. Then, we specifically focused on the symptoms' importance and influence. Of critical interest was the estimation of the unique influence of each symptom from one disorder to all symptoms of the other disorder using a new metric called bridge expected influence.

Results: The graphical LASSO revealed several cross-associations between SAD and comorbid depression. For each disorder, symptoms exhibiting the strongest cross-association with the other disorder were identified.

Limitations: Given our cross-sectional data, our findings can only suggest hypotheses about cause-effect relationships.

Conclusions: This study adds to a small but growing empirical literature revealing that the co-occurrence between two disorders is best portrayed as sets of symptom-to-symptom connections. As some individual symptoms show differential association in the co-occurrence between SAD and depression, those symptoms may be valuable targets for future research and treatment.

Social anxiety disorder (SAD) and depressive symptoms often covary; the lifetime comorbidity rates of major depression in patients with SAD range between 44% and 74.5% (Brown et al., 2001; Perugi et al., 2001; Schneier et al., 1992; Van Ameringen et al., 1991). Moreover, SAD often emerges before depressive symptoms. Indeed, SAD diagnosis predated any episode of mood disorders in 81.7% of patients with SAD (Van Ameringen et al., 1991). Likewise, in the general population, individuals with SAD had the highest likelihood of developing a major depression in the two years following the onset of SAD (Regier et al., 1998), especially in adolescents and young adults (Bittner et al., 2004; Stein et al., 2001). Moreover, longitudinal studies show that individuals with SAD are about three times more likely than those without SAD to develop depression (Beesdo et al., 2007; Stein et al., 2001).

The presence of comorbid depressive symptoms in people with SAD has clinical implications. First, comorbid depressive symptoms predict SAD persistence (Alpert et al., 1997; Stein et al., 2001) and recurrence (Bruce et al., 2005; Scholten et al., 2013). Second, depressive symptoms were associated with severity and generalization of the social fears and alcohol abuse among patients with SAD (Perugi et al., 2001). Third, patients with SAD and comorbid depressive symptoms are at elevated risk for attempting suicide (Cox et al., 1994; Sareen et al., 2005). Fourth, SAD patients with comorbid depressive symptoms are less likely to benefit from treatment for SAD in the short term (Ledley et al., 2005) or to maintain their gains over time (Marom et al., 2009). Especially, certain symptoms of depression such as insomnia and fatigue may interfere with CBT for SAD (e.g., Kushnir et al., 2014).

Although the presence of depressive symptoms in SAD is well documented, uncertainty remains regarding how core symptoms of SAD—i.e. fear and avoidance of social situations—are associated with

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depressive symptoms, and *vice versa*. Of clinical importance, distinct types of social fear may vary in terms of their debilitating nature. Theorists of SAD have argued that social fears involving interactions with strangers (e.g., Carron et al., 1999; Kagan, 2014; Kashdan and Wenzel, 2005) or authority figures (e.g., Gilbert, 2000; Swallow and Kuiper, 1988) may predict an especially inauspicious course, including risk for comorbid disorders. As a result, one cannot rule out the possibility that fear and avoidance vis-à-vis a given social situation may relate to different symptoms of depression. For instance, fear of talking to unfamiliar people may promote avoidance of going to a party, which, in turn, may influence depressive symptoms such as pessimism and self-dislike, worsening, in turn, the fear of talking to unfamiliar people.

In recent years, the theoretical (Borsboom, 2017; Borsboom and Cramer, 2013) and computational (e.g., Epskamp et al., 2012) advances in network analysis have opened up new vistas for understanding mental disorders as systems of interacting symptoms (Borsboom, 2017; Fried et al., 2016; McNally, 2016). Psychopathology networks comprise nodes (symptoms) and the edges (associations) connecting them. The network approach conceptualizes an episode of disorder as emerging from the pairwise interactions among symptoms. According to this perspective, symptoms possess independent causal powers that influence other symptoms (e.g., fear motivates avoidance; insomnia causes fatigue); they are not merely passive indicators of an underlying disease. Hence, symptoms are constitutive, not reflective, of disorder (Borsboom and Cramer, 2013).

Of critical importance, the network approach enables one to identify nodes that are central to the network based on the amount and direction of influence that flows from one node to other ones (Borgatti, 2005; Valente, 2012). Therefore, activation issuing from a node having strong connections to many other nodes can spread to other nodes, thereby producing a cascade of activation in the entire network (Borsboom and Cramer, 2013; Valente, 2012). Such highly influential nodes are thus especially important for the development, persistence, and remission of mental disorders (Borsboom and Cramer, 2013). By turning off such a node, one can affect other nodes both directly and indirectly (e.g., via paths through other nodes), thereby producing recovery from disorder (Hofmann et al., 2016; McNally, 2016; Valente, 2012).

If one accepts that symptoms and associations between them are what constitute a mental disorder, then the associations between symptoms of different disorders constitute pathways that can bridge those disorders (Borsboom and Cramer, 2013; Cramer et al., 2010). Traditionally, comorbid mental disorders have been understood as different underlying entities. Conversely, the network approach holds that the two disorders co-occur because of the mutual interactions among their symptoms (Cramer et al., 2010). Hence, comorbidity arises as a natural consequence of bridge symptoms—that is, a symptom that can transmit activation from one disorder to the other (Cramer et al., 2010; Fried and Cramer, 2017). Accordingly, bridge symptoms are key to disentangling co-occurrence between disorders (Bekhuis et al., 2016; Borsboom and Cramer, 2013; Fried and Cramer, 2017). Yet, uncertainty still abounds vis-à-vis the individual symptom-to-symptom association between SAD and depressive symptoms.

The purpose of our study was to apply network analytic methods to characterize the associations among core symptoms of SAD—i.e. fear and avoidance of social situations—and comorbid depressive symptoms in a convenience sample of individuals with a primary SAD. To accomplish this aim, we first explored the general structure of the network. Then, we specifically focused on the nodes' importance and influence. Of critical interest was the examination and identification of bridge symptoms—i.e. SAD symptoms that have strong associations with depressive symptoms, and *vice versa*.

1. Method

1.1. Participants

The sample consisted of 174 individuals (72% female) with a primary DSM-IV-TR diagnosis of SAD. In addition, 47 had a comorbid diagnosis of major depressive disorder, 26 had a diagnosis of depression (not otherwise specified), and 5 had a diagnosis of dysthymia. The participants constitute a convenience sample of individuals who were recruited for five other studies who had, as a result of participating in those studies, completed questionnaires measuring symptoms of social anxiety and depression used in the present network study (for full protocols, see Heeren et al., 2015a, 2015b, 2016, 2017a, 2017b). Each study included a nonprobability sampling approach and the different databases have been carefully checked to avoid potential multiple entries of the same participants.

To be eligible, individuals had to meet (a) DSM-IV-TR criteria for SAD, (b) have no current substance abuse or dependence, (c) no current neurological problems or use of psychotropic medications, and (d) no current psychological or psychiatric treatment. Participants were first screened via the self-report version of the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987). Eligible participants had to score above 56 on the LSAS (i.e., the cut-off score for probable diagnosis of SAD in the French version of the scale; Bouvard and Cottraux, 2010). Participants were then assessed by a clinical psychologist who used the screening version of the Mini-International Neuropsychiatric Interview (M.I.N.I; Sheehan et al., 1998). Each participant was tested individually in a quiet room. Each study was approved by the Ethical Committee of the Université Catholique de Louvain (UCL, Belgium) and conducted according to the Declaration of Helsinki. Participants' characteristics appear in Table 1.

1.2. Materials and measures

1.2.1. Measures of social anxiety symptoms

The LSAS is a 24-item scale that measures fear and avoidance of a range of social and performance situations (see Table 2). Participants rate each of the 24 social situations on a 4-point Likert-type scale, once for the intensity of fear (0, *None*; 1, *Mild*; 2 *Moderate*; 3 *Severe*) and once for frequency of avoidance of the situation (0, *Never*; 1, *Occasionally*; 2 *Often*; 3 *Usually)*. We used the validated French versions of this scale (Heeren et al., 2012). The internal reliability of LSAS was high in the current sample, with a Cronbach's alpha of .81 for the global scale score (.82 for the fear scale score and .80 for the avoidance scale score).

1.2.2. Measure of depressive symptoms

The Beck Depression Inventory (BDI-II; Beck et al., 1996) is a 21-item instrument designed to measure both the presence and severity of depressive symptoms. Each item consists of a group of four statements measuring the symptoms of depression (e.g., loss of interest) that range in intensity, each item being scored on a scale value of 0–3. We used the validated French versions of this scale (BDI-II; Beck et al., 1998). The

Table 1
Demographic and clinical measures for individuals with social anxiety disorder.

	Mean (*SD*)	Min-Max
Demographic measures		
Age	28.40 (11.26)	18–67
Educational level (in years)	10.78 (2.33)	0–15
Clinical measures		
BDI-II	12.86 (8.15)	1–37
LSAS	71.67 (13.69)	57–112

Note. Education level was assessed according to the numbers of years of education completed after finishing primary school. BDI-II = Beck Depression Inventory; LSAS = Liebowitz Social Anxiety Scale.