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An integrative network approach to social anxiety disorder: The complex dynamic interplay among attentional bias for threat, attentional control, and symptoms

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ABSTRACT

Cognitive models posit that social anxiety disorder (SAD) is associated with and maintained by biased attention allocation vis-à-vis social threat. However, over the last decade, there has been intense debate regarding whether AB in SAD results from preferential engagement with or difficulty in disengaging from social threat. Further, recent evidence suggests that AB may merely result from top-down attentional impairments vis-à-vis non-emotional material. Consequently, uncertainty still abounds regarding both the relative importance and the mutual interactions of these different processes and SAD symptoms. Inspired by novel network approaches to psychopathology that conceptualize symptoms as complex dynamic systems of mutually interacting variables, we computed weighted directed networks to investigate potential causal relations among laboratory measures of attentional components and symptoms of social anxiety disorder. Global and local connectivity of network structures revealed that the three most central variables were the orienting component of attention as well as both avoidance and fear of social situations. Neither preferential attention engagement with threat nor difficulty disengaging from threat exhibited high relative importance as predictors of symptoms in the network. Together, these findings suggest the value of extending the network approach beyond self-reported clinical symptoms to incorporate process-level measures from laboratory tasks to gain new insight into the mechanisms of SAD.

1. Introduction

Social anxiety disorder (SAD) is a common syndrome with a lifetime prevalence of more than 12% (e.g., Kessler et al., 2005). SAD is characterized by intense fear and avoidance of social situations causing considerable distress and impaired daily functioning. It has an early age of onset and tends to follow a chronic and debilitating course if untreated (e.g., Hayward et al., 2008). Moreover, SAD usually precedes the onset of other common comorbid anxiety, mood, and substance abuse disorders (e.g., Lampe, Slade, Issakidis, & Andrews, 2003; Randall, Thomas, & Thevos, 2001).

Although the personal and economic costs of SAD as well as its comorbidity with other disorders are well documented, uncertainty remains regarding factors responsible for the etiology and chronicity of this disorder. As highlighted by Hirsch and Clark (2004), a curious feature of SAD is that it persists even when sufferers perform naturalistic exposure to at least some feared social situations on a regular basis in their daily life. One possibility is that people with chronic SAD process information in ways that maintain their anxiety. Laboratory studies involving probe detection and probe discriminations tasks indicate that people with SAD respond faster to probes replacing social-threat stimuli, such as faces expressing anger or contemptuous disgust, or to words, such as *humiliation*, than to probes replacing neutral cues, thereby exhibiting an attentional bias (AB) for social threat that is absent in nonanxious individuals (for a meta-analysis, see Bantin, Stevens, Gerlash, & Hermann, 2016). Moreover, as argued by cognitive theorists, AB may causally contribute to increased anxiety proneness, and thereby figure prominently in the maintenance, and perhaps the etiology, of SAD (e.g., Heimberg, Brozovich, & Rapee, 2010; Rapee & Heimberg, 1997; for a review, see Wong & Rapee, 2016). Accordingly, AB may interfere with the ability to process external cues that disconfirm the negative beliefs about socially

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challenging situations held by people with SAD. Failure to disconfirm these beliefs may impede anxiety reduction, which, in turn motivates avoidance of social situations and worsens anxiety or least prevents it from extinguishing (e.g., Heimberg et al., 2010; Wong & Rapee, 2016). Therefore, reducing AB may yield clinical benefits (for a meta-analysis, see Heeren, Mogoaşe, Philippot, & McNally, 2015). Likewise, transiently fostering AB promotes anxiety proneness among nonanxious controls (e.g., Heeren, Peschard, & Philippot, 2012; MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002). Taken together, such findings are suggestive of a causal relation between AB and SAD.

To date, several explanations have been proposed to account for the maintenance of AB in anxiety disorders (e.g., Cisler & Koster, 2010; Eysenck & Derakshan, 2011; Heeren, De Raedt, Koster, & Philippot, 2013; Peers, Simons, & Lawrence, 2013). One of the most common explanations focuses on general attention control (AC), that is, the ability to voluntarily regulate the allocation of attentional resources. According to this account, AB may result from impaired AC. For example, Derryberry and Reed (2002) found that AB exhibited by individuals with elevated trait anxiety was moderated by AC. Individuals with lower AC exhibited stronger AB for threat in comparison to those with higher AC. Since this initial study, several replications of this effect have been reported across numerous paradigms and anxiety disorders (e.g., Bardeen & Orcutt, 2011; Reinholdt-Dunne, Mogg, & Bradley, 2009; Taylor, Cross, & Amir, 2016). However, despite increasing research linking AB and SAD symptoms, there are several limitations to these studies.

First, many studies on SAD failed to find a correlation between AB and severity of symptoms (e.g., Gotlib et al., 2004; Ononaiye, Turpin, & Reidy, 2007; Taylor et al., 2016). Likewise, although meta-analyses indicated a significant difference on AB between SAD and nonanxious participants, the effect size is small (for a meta-analysis, see Bantin et al., 2016). Moreover, modifying AB had only a very small effect – albeit significant – on reducing SAD symptoms (for a meta-analysis, see Heeren, Mogoaşe, Philippot et al., 2015). Likewise, the anxiolytic benefit resulting from AB reduction may be more complicated than initially thought as recent studies suggest that control procedures lacking a contingency between emotional cues and probes reduced anxiety just as much as AB modification procedures where probes reliably followed nonthreat cues (e.g., Carleton et al., 2015; Heeren, Coussement, & McNally, 2016; McNally, Enock, Tsai, & Tousian, 2013; Yao, Yu, Qian, & Li, 2015). Taken together, these findings seemingly challenge the claim that AB figures prominently in the maintenance of SAD (e.g., Clark & Wells, 1995; Rapee & Heimberg, 1997).

Second, there have been attempts to disentangle subcomponents of AB through the use of variants of the probe discrimination and detection tasks as well as through eye-tracking procedures to determine whether AB in SAD reflects facilitated attentional engagement with social-threat cues (e.g., Grafton & MacLeod, 2016; Klumpp & Amir, 2010) or impaired attentional disengagement from them (e.g., Amir, Elias, Klumpp, & Przeworksi, 2003; Buckner, Maner, & Schmidt, 2010; Schofield, Johnson, Inhoff, & Coles, 2012; Taylor et al., 2016). Some studies suggest that people with SAD, relative to nonanxious participants, exhibit increased attentional engagement with social-threat cues and impaired disengagement with them (for a meta-analysis, see Bantin et al., 2016). Yet it remains unclear how these biases interact with AC and with symptoms such as fear and avoidance. Moreover, researchers have usually tested only simple, unidirectional relationships among these variables. This is unfortunate as many of these may have reciprocal influences among them in SAD. For instance, facilitated attentional engagement with social-threat cues may influence fear of social situations, and fear of these situations may motivate avoidance that exacerbates fear. Hence, feedback loops among symptoms may foster maintenance of the disorder.

Third, prominent models of attentional systems postulate that AC is a multifaceted construct (e.g., Petersen & Posner, 2012; Posner & Rothbart, 2007), including at least three components: alerting (maintenance of alertness), orienting (selective engagement and disengagement with certain stimuli rather than others), and an executive component (top-down control of attention exemplified by maintenance of attention on certain stimuli and resisting distraction by other stimuli). However, most studies in the field of AB research have treated AC as a unitary construct. This is unfortunate as SAD is associated, in some studies, with the orienting component (e.g., Heeren, Maurage, & Philippot, 2015; Moriya & Tanno, 2009a, 2009b) whereas, in others, with the executive one (e.g., Judah, Grant, Mills, & Lechner, 2013; Sutterby & Bedwell, 2012). To date, no study has explored the relations between AB and all three components of attention in SAD.

To clarify the dynamics among AB components, the three components of AC, and the core symptoms of SAD, such as fear and avoidance of social situations and reactivity to social-evaluative challenge, we applied network analytic methods pioneered in the psychopathology field by Borsboom and his colleagues (e.g., Borsboom & Cramer, 2013; Cramer, Waldorp, van der Maas, & Borsboom, 2010) and increasingly used by others (e.g., Costantini et al., 2015; Hoorelbeke, Marchetti, De Schryver, & Koster, 2016; McNally et al., 2015; Robinaugh, Leblanc, Vuletich, & McNally, 2014). According to this approach, mental disorders are complex dynamic systems of interacting elements or "symptoms" in traditional psychiatric parlance (Borsboom & Cramer, 2013; Cramer et al., 2010). Based on graph theory (i.e., the branch of mathematics concerned with the study of networks), a network consists of nodes and edges that connect them. Such a network approach can be used to describe many kinds of phenomena, including social relations, biological structures, and information networks (Barabási, 2012).

Although several psychological studies have already explored the associations among the aforementioned processes of interest, network approaches can be employed to disentangle complex dynamic systems of such mutually interacting psychological processes (e.g., Hoorelbeke et al., 2016; Robinaugh et al., 2014). Particularly, as compared to mere correlational approaches, computational tools from graph theory can be used to examine the extent to which nodes are central to the network, based the amount and direction of (potentially) causal influence that flows from one node to other ones (Borgatti, 2005; Costantini et al., 2015). Moreover, aside from the local connectivity among nodes, one additional relevant feature of graph theory is the notion of modularity-based community detection, defined as the identification of subsets of nodes where there is a higher density of edges within these communities ("clusters") than between them (Boccaletti, Latora, Moreno, Chavez, & Hwang, 2006; Fortunato, 2010). Such communities can function as relatively independent modules of a network, playing distinctive roles just as organ systems do in the human body (Fortunato, 2010). Community detection algorithms can uncover major sub-networks that correspond to specialized functional modules (Boccaletti et al., 2006; Fortunato, 2010).

In the present study, we computed weighted and directed networks to investigate the dynamic interplay among laboratory measures of AB, attentional components, emotional reactivity to social-evaluative challenge, and core symptoms of SAD (i.e., fear and avoidance of social situations). Of primary interest was the elucidation of local connectivity between variables, and especially their centrality and the predictive relations among them. Aside from local connectivity, we also examined global connectivity by using modularity-based community detection methods. In this way, we tested whether these variables cohere as a single causal system of mutually interacting elements or constitute distinct functionally specialized communities of interacting elements.