Journal of Anxiety Disorders 37 (2016) 71–77

Contents lists available at ScienceDirect

Journal of Anxiety Disorders

Theory of mind in social anxiety disorder, depression, and comorbid conditions

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ARTICLE INFO

Article history:
Received 14 January 2015
Received in revised form 4 November 2015
Accepted 7 November 2015
Available online 22 November 2015

Keywords:
Social anxiety disorder
Theory of mind
Major depression
Comorbidity

ABSTRACT

Social anxiety disorder is characterized by marked interpersonal impairment, particularly when presenting with comorbid major depression. However, the foundational social-cognitive skills that underlie interpersonal impairment in comorbid and non-comorbid manifestations of SAD has to date received very little empirical investigation. In a sample of 119 young adults, the current study examined differences in theory of mind (ToM), defined as the ability to decode and reason about others' mental states, across four groups: (a) non-comorbid SAD; (b) non-comorbid Lifetime MDD; (c) comorbid SAD and Lifetime MDD; and (d) healthy control. The non-comorbid SAD group was significantly less accurate at decoding mental states than the non-comorbid MDD and control groups. Further, both the comorbid and non-comorbid SAD groups made significantly more 'excessive' ToM reasoning errors than the non-comorbid MDD group, suggesting a pattern of over-mentalizing. Findings are discussed in terms of their implications for understanding the social cognitive foundations of social anxiety.

1. Introduction

Social anxiety disorder (SAD) is characterized by a marked and persistent fear of social evaluation (APA, 2000). SAD is a chronic and debilitating psychiatric disorder that is associated with poor educational and occupational attainment, marked social and interpersonal impairment, and substantial psychiatric and medical comorbidity (Grant et al., 2005). Individuals with SAD display a number of negative interpersonal behaviors, including interpersonal dependency, conflict avoidance, and avoidance of emotion, that result in weak interpersonal ties and eventual social isolation (see Davila & Beck, 2002). Given the degree and chronicity of social and interpersonal impairment associated with SAD it is important to understand the underlying social-cognitive mechanisms.

Critical to successful social and interpersonal functioning is having a 'theory of mind' (ToM), or the ability to accurately decode and reason about the beliefs, intentions, desires, and emotions of others (Wellman, 1990). ToM is a universal human skill that involves two separate, but related components (Sabbagh, 2004). First, theory of mind 'decoding' involves the foundational skill of accurately labeling others' mental states (e.g., decoding that a conversation partner is 'interested' based on facial expression). Second, theory of mind 'reasoning' involves using others' mental states to make predictions about their future behavior (e.g., reasoning that one's conversational partner will continue the conversation based on one's judgment that he or she is interested). Deficits in ToM decoding and reasoning have been reported in a number of clinical conditions characterized by severe social and interpersonal dysfunction, including autism spectrum disorder (e.g., Baron-Cohen et al., 1999), schizophrenia (e.g., Frith & Corcoran, 1996), and major depressive disorder (MDD; e.g., Lee, Harkness, Sabbagh, & Jacobson, 2005).

Despite the fact that SAD presents with marked social and interpersonal dysfunction as a primary impairment, only two studies to our knowledge have examined ToM skill in individuals with social anxiety. First, in a community sample of adults, Samson, Lackner, Weiss, & Papousek (2012) found that higher scores on a measure of social anxiety were significantly related to lower enjoyment of humorous cartoons that involved resolving incongruity about others' false mental states. The researchers theorized from these results that humor that involves ToM reasoning may elicit negative affect in individuals with high levels of social anxiety, thus interfering with its enjoyment.

Second, Hezel and McNally (2014) found that individuals with a diagnosis of SAD were significantly impaired relative to healthy controls on tasks of ToM decoding and ToM reasoning. Specifically, individuals with SAD were significantly less accurate than controls at decoding the subtle mental states portrayed by photographs of eyes in the Reading the Mind in the Eyes task (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001), particularly if the eyes

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http://dx.doi.org/10.1016/j.janxdis.2015.11.004
0887-6185/

depicted mental states of a negative valence. Further, individuals with SAD were significantly impaired relative to controls at reasoning about the intentions and beliefs of characters presented in movie clips in the Movie Assessment of Cognition task (MASC; Dziobek et al., 2006). In particular, they showed a pattern of performance that was consistent with using excessive ToM. That is, they *over*-interpreted the mental states of the characters in the film clips. Hezel and McNally (2014) suggest from this latter finding that individuals with SAD may show "cognitive empathy" towards others, which could explain their tendency to "over-mentalize" others' perspectives (see also Tibi-Elhanany & Shamay-Tsoory, 2011). However, this interpretation is inconsistent with results showing that individuals with SAD performed more poorly than controls on the Eyes task, which also requires respondents to put themselves into the minds of others (see Harkness, Sabbagh, Jacobson, Chowdrey, & Chen, 2005). Therefore, further investigation of the differential pattern of performance across tasks of ToM decoding and ToM reasoning, and across different diagnostic groups is required. Nevertheless, these studies are important in suggesting that deficits in the foundational social cognitive skills of ToM decoding and reasoning may underlie the marked social and interpersonal impairment shown by individuals with SAD.

One critical question that remains unanswered from the studies cited above concerns the extent to which deficits in ToM in individuals with SAD can be accounted for by comorbid conditions that are associated with social-cognitive impairment. In particular, 20–37% of individuals with SAD also suffer from Lifetime major depressive disorder (MDD; Merikangas & Angst, 1995; Ohayon & Schatzberg, 2010). Similar to SAD, MDD is associated with excessive negative self-focused attention (Mor & Winquist, 2002), negative interpersonal behaviors such as avoidance, and marked interpersonal impairment (Alden & Taylor, 2004; Segrin & Abramson, 1994). Consistent with the hypothesis that deficits in the foundational social-cognitive skill of ToM underlie interpersonal impairment, several studies have documented deficits in ToM decoding and ToM reasoning in patients with MDD relative to healthy controls both when in episode (Lee et al., 2005; Wang, Wang, Chen, Zhu, & Wang, 2008; Kettle, O'Brien-Simpson, & Allen, 2008) and upon remission (Inoue, Tonooka, Yamada, Kanba, 2004; Inoue, Yamada, & Kanba, 2006; Harkness, Jacobson, Duong, & Sabbagh, 2010).

Individuals with comorbid SAD and MDD report even higher levels of avoidance of negative emotional stimuli, higher levels of social avoidance, and greater impairments in social functioning than those with SAD or MDD alone (Aderka et al., 2012; Dalrymple & Zimmerman, 2007; LeMoult & Joormann, 2012; Ottenbreit, Dobson, & Quigley, 2014). Therefore, there is reason to suspect that individuals with comorbid SAD and MDD may show lower levels of ToM performance than non-comorbid conditions. Indeed, given the strong comorbidity between MDD and SAD, and the presence in both conditions of significant social-cognitive and interpersonal impairment, it is important to clarify the extent to which deficits in performance associated with MDD or SAD shown in previous studies can be better accounted for by their comorbidity.

Individuals with SAD (either alone or comorbid with MDD) also differ from those with MDD alone on a number of variables that are of crucial relevance to social cognition. For example, they show significantly greater avoidance of social situations (Ottenbreit et al., 2014), and greater avoidance of negative emotional stimuli (e.g., angry faces; Kircanski, Joormann, & Gotlib, 2014). This prior literature suggests that individuals with SAD, whether or not comorbid with MDD, may show greater ToM deficits than those with MDD alone. Therefore, the goal of the current study was to compare ToM decoding and ToM reasoning accuracy across four diagnostic groups: 1. Those with a diagnosis of SAD and no comorbid lifetime diagnosis of MDD; 2. Those with a lifetime diagnosis of MDD and no comorbid SAD; 3. Comorbid SAD and lifetime MDD; and 4. Healthy controls with no history of a psychiatric diagnosis. We employed the Eyes task to assess ToM decoding and the MASC task to assess ToM reasoning. We hypothesized that the SAD only and comorbid groups would evidence significantly lower accuracy on the Eyes and MASC tasks than the Lifetime MDD only and control groups. Further, we hypothesized that the Lifetime MDD only group would evidence significantly lower accuracy on the Eyes and MASC tasks than the Healthy control group.

2. Materials and methods

2.1. Subjects

The General Research Ethics Board at Queen's University approved this study. All participants provided written informed consent. Participants included 119 individuals (81 females; ages 17–36, $M = 19.38$, SD = 2.87; 65% European–Canadian, 31% Asian, 4% Other ethnicity) recruited from an introductory psychology class. All students from the introductory psychology class completed the Beck Depression Inventory-II (BDI-II; Beck, 1996) and the Social Anxiety and Avoidance Scale for Adolescents (SAASA; Cunha, Pinto-Gouveia, & do Céu Salvador, 2008) during a prescreening session held at the beginning of the academic year. To increase our chances of recruiting individuals with a history of depression and social anxiety symptoms, we oversampled from this prescreen pool students with elevated scores on the BDI-II (over 10) or SAASA (over 70). Community advertisements also targeted socially anxious individuals. Exclusion criteria were a lifetime history of psychotic disorder, manic episodes, drug/alcohol dependence, or developmental disability.

All potential participants underwent an initial telephone screen performed by the first author that queried for exclusion criteria. This telephone screen also included the depressive disorder and the SAD modules of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P; First, Spitzer, Gibbon, & Williams, 2002). Potential participants were invited to participate in the study if they answered 'no' to the questions querying the exclusion criteria and met criteria based on the SCID-I/P modules for one of our study groups (lifetime MDD, SAD, comorbid lifetime MDD and SAD, or no history of MDD or SAD). An initial 121 individuals passed the phone screen and were invited to participate. Two participants were excluded because they scored more than two standard deviations below the sample mean on the MASC task, leaving a final sample of 119.

2.2. Measures

2.2.1. Diagnostic interview

The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P; First et al., 2002) was administered at the time of the study to confirm diagnoses by clinical psychology graduate and senior undergraduate students trained to reliability under the senior author's supervision ($\kappa = .71$–1.00). Participants were assigned to the study groups based on their clinical diagnoses as determined by the in-person SCID-I/P as follows: (a) SAD only ($n = 12$) consisted of those who met current DSM-IV criteria for SAD and did not have a history of MDD; (b) Lifetime MDD only ($n = 40$) included individuals with a current or past episode of MDD and no current diagnosis of SAD; (c) Comorbid SAD and Lifetime MDD ($n = 24$) included individuals diagnosed with both current SAD and a current or past episode of MDD; or (d) Healthy Control ($n = 43$) included those who had never met criteria for SAD or MDD or any other psychiatric diagnosis.

2.2.2. Depression and social anxiety measures

At the experimental session participants again completed the BDI-II and SAASA, and means and standard deviations on these