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

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A B S T R A C T

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.

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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
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 Tibe-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; Olahyn & 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 (κ = .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 = 42) 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
measures by group are provided for descriptive purposes in Table 1. The BDI-II is a 21-item self-report questionnaire measuring depression symptom severity (Cronbach’s alpha = .94). The SAASA is a self-report questionnaire assessing the degree of (a) avoidance and (b) anxiety from 34 social situations (e.g., “eating in public,” “being alone with a classmate of the opposite sex”). Avoidance and anxiety are rated separately on a 5-point scale from 1 – not anxious/never avoid to 5 – very anxious/almost always avoid (Cronbach’s alpha = .97). Separate avoidance and anxiety subscale scores are derived by summing the scores for each item. A total SAASA score is calculated as the mean of the anxiety and avoidance subscales. Thus, the maximum total score is 170. We chose the SAASA as a measure of social anxiety given the closeness in age of undergraduates to the adolescents on which the SAASA was validated, and our reasoning that the content of the items was a good match to the social environment of our young undergraduate sample (e.g., “Going to a party given by a classmate”: “performing, for the first time, a new task or role in front of classmates”).

2.2.3. Reading the mind in the eyes task (Eyes task: Baron-Cohen et al., 2001)

The Eyes task is a ToM decoding task consisting of 36 black-and-white photographs of the eye region of faces. Each pair of eyes was standardized to 15 x 6 cm and presented centered between four mental state adjectives located in each corner, equally spaced from the center of a computer screen (e.g., annoyed, horrified, hostile, preoccupied). Participants chose their response by pressing one of four keys (S, X, K, M), identified by stickers that were spatially analogous to the location of the adjectives. Participants’ responses and response latencies in milliseconds (ms) were digitally recorded. The Eyes stimuli include three emotional valence categories: positive (e.g. “Friendly”), neutral (e.g. “Reflective”) and negative (e.g. “Upset”) (see Harkness et al., 2005).

2.2.4. Animals task (Harkness et al., 2005)

The Animals task has been used in a number of previous studies (Harkness et al., 2005, 2010; Harkness, Washburn, Theriault, Lee, & Sabbagh, 2011; Lee et al., 2005) as a control to ensure that group differences on the Eyes task can be ascribed to differences in mental state decoding rather than to task demands or low-level perceptual processing. The task includes 12 black-and-white photographs of animals presented in a format similar to the Eyes task. Participants were required to choose the correct adjective from an array of four describing a trait of the animal (e.g., aloof, ferocious, timid, obedient). Responses and response latencies in milliseconds (ms) were digitally recorded.

2.2.5. Movie for the assessment of cognition (MASC; Dziobek et al., 2006)

The MASC is a ToM reasoning task in which participants watched a 15-min video of four actors interacting at a dinner party. The video was paused at 45 time points and participants were required to answer questions probing their understanding of the characters’ feelings, thoughts, and intentions at the point at which the film was stopped (e.g., “What is Betty feeling?”). For each question, participants chose from one of four possible answers in multiple-choice format. For each question, each of the four possible answers reflected either: (1) ‘correct’ ToM reasoning (i.e., an appropriate level of ToM reasoning), (2) excessive ToM, representing over-interpretative mental state reasoning, (3) insufficient, or reduced, mental state reasoning, and (4) no ToM ability, or non-mental state reasoning (i.e., physical causation). Percent correct responses, indicating appropriate ToM reasoning ability, were calculated as the total number of correct responses divided by 45 questions and multiplied by 100. Percent of each type of error was calculated as the number of items on which participants endorsed each error divided by the 45 questions and multiplied by 100. In addition to the 45 ToM questions, six questions were control items requiring non-social inferences (e.g., “How did Cliff likely behave when he was in Sweden?”). Performance on the control items was calculated as the number of correct responses out of the total number of control items (n = 6).

2.3. Procedure

After providing written informed consent, participants were seated at a computer and completed the Eyes and MASC tasks in counter-balanced order. For the Eyes task, participants completed a practice trial followed by the Eyes and Animals task trials randomly combined in a single block of 48 trials (36 trials for the Eyes and 12 Animals tasks). For the MASC, participants were told to follow the instructions that appeared on the screen. Participants were instructed to respond as quickly and accurately as possible. Participants then completed the SAASA, BDI-II, and SCID-I/P interview. Participants were compensated with course research credit or $20.

Table 1
Descriptive statistics of demographic variables, BDI, and SAASA scores stratified by group.

<table>
<thead>
<tr>
<th></th>
<th>Lifetime MDD (n = 40)</th>
<th>SAD (n = 12)</th>
<th>Comorbid (n = 24)</th>
<th>Control (n = 43)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age M (SD)</td>
<td>19.73 (4.43)</td>
<td>19.83 (4.11)</td>
<td>19.71 (2.81)</td>
<td>18.74 (1.71)</td>
</tr>
<tr>
<td>Sex: Female n (%)</td>
<td>28 (70)</td>
<td>7 (58.3)</td>
<td>18 (75)</td>
<td>28 (65)</td>
</tr>
<tr>
<td>Ethnicity (n/%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>28 (70)</td>
<td>5 (41.7)</td>
<td>15 (62.5)</td>
<td>29 (67.4)</td>
</tr>
<tr>
<td>Asian</td>
<td>11 (27.5)</td>
<td>6 (50)</td>
<td>9 (37.5)</td>
<td>11 (25.6)</td>
</tr>
<tr>
<td>Other</td>
<td>1 (2.5)</td>
<td>1 (8.3)</td>
<td>0 (0)</td>
<td>3 (7)</td>
</tr>
<tr>
<td>Range</td>
<td>1.00–46.00</td>
<td>2.00–18.00</td>
<td>0.00–38.00</td>
<td>0.00–29.00</td>
</tr>
<tr>
<td>SAASA M (SD)</td>
<td>92.85 (21.70)*</td>
<td>113.13* (20.12)</td>
<td>111.92* (19.91)</td>
<td>77.12* (18.49)</td>
</tr>
<tr>
<td>Range</td>
<td>48.50–136.00</td>
<td>78.50–136.00</td>
<td>78.00–147.50</td>
<td>45.00–130.50</td>
</tr>
<tr>
<td>Anxiety M (SD)</td>
<td>100.53 (23.39)*</td>
<td>122.00* (20.31)</td>
<td>120.42* (22.07)</td>
<td>82.91* (20.00)</td>
</tr>
<tr>
<td>Avoidance M (SD)</td>
<td>85.18 (21.56)*</td>
<td>104.25* (21.12)</td>
<td>103.42* (19.94)</td>
<td>71.33* (18.52)</td>
</tr>
</tbody>
</table>

Notes: BDI-II = Beck depression inventory; SAASA = Social anxiety and avoidance scale for adolescents.

* Group differences were significant at p < .01.

† Group differences were significant at p < .01.

& Group differences were significant at p < .01.
3. Results

3.1. Preliminary analyses

Descriptive characteristics of the sample stratified by diagnostic group are presented in Table 1. Groups did not differ significantly on age, sex, ethnicity, or education (all \(p > .35\)). As expected given that state depression symptoms are generally elevated in individuals with SAD (Coles, Gibb, & Heimberg, 2001) and in those with a history of MDD (Paykel et al., 1995), in the current sample the Lifetime MDD, SAD, and Comorbid groups scored significantly higher on the BDI-II than controls, \(F(3, 115) = 11.20, p < .001\), partial \(\eta^2 = .23\). Further, as expected, the SAD and Comorbid groups had significantly higher SAASA scores than the Lifetime MDD group, which scored significantly higher than controls, \(F(3, 115) = 20.12, p < .001\), partial \(\eta^2 = .34\). Neither Eyes task accuracy nor any of the subscales of the MASC were significantly associated with any of the demographic characteristics (all \(p > .31\)).

As expected, accuracy on the Eyes task was significantly positively correlated with accuracy on the Animals task, \(r(117) = .38\), \(p < .001\). Further, accuracy on the Eyes task was significantly positively correlated with total accuracy on the MASC task, \(r(117) = .21\), \(p = .02\). However, as shown in Table 2, correlations between BDI-II and SAASA scores and accuracy on the Eyes and MASC tasks, partialling variance accounted for by Animals task accuracy and MASC control item accuracy, respectively, were not significant.

3.2. Group differences in ToM decoding

Descriptive statistics for the primary study variables by group are presented in Table 3. Group differences in Eyes task accuracy were evaluated with a one-way Analysis of Covariance (ANCOVA) including Animals task accuracy and Eyes response latencies as covariates. Three a priori contrasts were specified as per the study hypotheses to compare the Lifetime MDD to the SAD only and Comorbid groups, and the Lifetime MDD group to the Control group.

The omnibus effect of group was significant, \(F(3, 113) = 3.26, p = .02\), partial \(\eta^2 = .08\). Consistent with hypotheses, the SAD only group performed significantly more poorly on the Eyes task than the Lifetime MDD only group, \(t(113) = 3.86, p < .001\). Contrary to hypotheses, the Comorbid group did not perform significantly more poorly on the Eyes task than the Lifetime MDD group, \(t(113) = 0.02, p = .89\), and the Lifetime MDD group did not perform significantly more poorly than the Control group, \(t(113) = .27, p = .47\).

To determine whether a relation of group and Eyes task accuracy was specific to stimulus valence, we conducted a 3 (valence: positive, negative, neutral) × 4 (group) mixed model ANOVA with valence as the within-group factor and group as the between-group factor. There was no evidence of a significant main effect of valence, \(F(2, 113) = .45, p = .64\), partial \(\eta^2 = .008\), nor was there a significant valence by group interaction, \(F(2, 113) = 2.33, p = .10\), partial \(\eta^2 = .04\).

Instead, replicating the overall accuracy results above, orthogonal contrast results indicated that the SAD only group performed more poorly than the Lifetime MDD group across the positive, \(t(115) = 2.22, p = .03\), negative (as a trend), \(t(115) = 1.72, p = .09\), and neutral eyes, \(t(115) = 2.15, p = .03\). Further, the SAD only group performed significantly more poorly than the Control group on the positive, \(t(115) = 2.66, p = .009\) and neutral eyes, \(t(115) = 2.00, p = .048\). As in the overall accuracy analyses, no other group comparisons were significant.

3.3. Group differences in ToM reasoning

We evaluated group differences in accuracy on the MASC task with a one-way ANCOVA controlling for accuracy on the Control items. The omnibus ANCOVA failed to reach statistical significance, \(F(3, 114) = 1.58, p = .20\), partial \(\eta^2 = .04\), although the orthogonal contrasts confirmed hypotheses that the SAD and Comorbid groups performed significantly more poorly than the Lifetime MDD group, \(t(115) = 1.79, p = .038\).

An analysis of group differences on MASC error types revealed that the SAD only group, \(t(115) = 2.66, p = .009\), and the Comorbid group, \(t(115) = 2.51, p = .052\), at a trend, made more excessive ToM errors than the Lifetime MDD group, and the Lifetime MDD group did not differ significantly from controls, \(t(115) = 1.16, p = .25\). No significant group differences were found for errors involving insufficient ToM, \(ts(115) < .54, ps > .60\), or no ToM, \(ts(115) < .83, ps > .40\).

4. Discussion

Consistent with hypotheses, individuals with SAD were significantly less accurate at decoding the subtle social features of eye expressions than were those with MDD. However, this effect was specific to non-comorbid conditions. Those with comorbid SAD and lifetime MDD did not differ significantly from MDD alone or from healthy controls. Very few studies have compared those with SAD alone to those with comorbid SAD and MDD, but those that have consistently find greater functional impairment in comorbid presentations (e.g., Adkerka et al., 2012). Therefore, we were surprised to find worse ToM decoding performance only in the non-comorbid SAD group. SAD generally precedes the onset of MDD, and longitudinal studies have shown that those with SAD who go on to develop MDD are characterized by higher levels of interpersonal over-sensitivity and social impairment than those who do not (e.g., Katz, Conway, Hammen, Brennan, & Najman, 2011; Starr, Hammen, Connolly, & Brennan, 2014). By definition, individuals who endorse high levels of interpersonal sensitivity think and care more about what others are thinking and feeling. This construct has, thus, been proposed as one explanation for the paradoxical findings linking some clinical conditions (e.g., subthreshold depression, borderline personality disorder) with enhanced theory of mind decoding abilities (see Dinsdale & Crespi, 2013; Franzen et al., 2011; Harkness et al., 2005, 2010, 2011; Harkness, Jacobson, Sinclair, Chan, & Sabbagh, 2012). That is, these groups may be significantly more accurate than others at decoding mental states because they are more sensitive to (i.e., care more about) what others may be thinking and feeling. Therefore, perhaps individuals with non-comorbid SAD who never develop MDD suffer from the central social avoidance that characterizes SAD generally, and that
would prevent an entraining of accuracy at decoding social stimuli, and they also lack the interpersonal orientation of depression vulnerability that could provide the motivation to override this central social-cognitive deficit. This explanation is very speculative at present, particularly given the small size of the non-comorbid SAD group. However, the current results highlight the need for more research specifically comparing comorbid and non-comorbid SAD on measures of social cognition and interpersonal outcomes.

Contrary to the results of Hezel and McNally (2014), the poor ToM decoding performance shown by those with non-comorbid SAD was not specific to eyes of a negative valence, but held across the positive, negative, and neutral valence categories. Indeed, in our sample, group differences were weakest for the negative eyes. In our previous work using the Eyes task in samples of individuals with depression we have also generally failed to find valence effects (e.g., Harkness et al., 2005, 2012; Lee et al., 2005). These results would appear to be inconsistent with the wealth of literature in the area of cognition more generally showing that individuals with SAD and MDD show biased attention to negative facial stimuli and valence-specific deficits in facial emotion recognition. However, the negative mental states included in the Eyes task are more complex and subtle than the basic emotions portrayed in studies of biased attention and facial recognition (sad, angry, fearful).

Further, the negative category in the Eyes task includes a wide range of mental states that represent sadness (“upset,” “regretful”), anger (“hostile,” “defiant”), and fear (“worried,” “nervous”). In studies of biased attention, individuals with SAD show a preferential bias to angry and threatening facial stimuli (Kiranski et al., 2014; LeMoulton & Jormann, 2012; Mogg & Bradley, 2002), whereas individuals with MDD show attentional biases preferentially to sad facial expressions (see Bourke, Douglas, & Porter, 2010). Therefore, it is possible that group differences due to valence effects would emerge more consistently in studies of mental state decoding if a more fine-grained approach to categorizing negative mental states were taken in future research.

Consistent with hypotheses, both the comorbid and non-comorbid SAD groups showed a pattern of excessive ToM reasoning on the MAS task relative to healthy controls. Further, the current results extend those of Hezel and McNally (2014) by showing that individuals with SAD also significantly differed in their ToM reasoning abilities to those with MDD alone. Specifically, those with SAD over-interpreted the thoughts, emotions, and intentions of others. As suggested by Hezel and McNally (2014), in contrast to individuals with autism spectrum disorder who fail to use ToM when reasoning about the scenes in the MASC, individuals with SAD are “over-mentaling;” and incorrectly attributing mental states to others when not appropriate to the context. This is certainly a ToM reasoning error, and one that would, in real-life contexts, potentially predict negative interpersonal outcomes (e.g., misunderstandings, conflicts, tensions, etc.), although perhaps different outcomes than those suffered by individuals with autism.

However, to over-mentalize, this suggests that individuals with SAD care about, and potentially have a motivation to, read into the minds of others. As such, it fails to account for the differential patterns of performance of the comorbid and non-comorbid SAD groups across the ToM decoding and ToM reasoning tasks. ToM decoding and reasoning are related, but also dissociable skills (Sabbagh, 2004), and, indeed, accuracy on the Eyes and MASC tasks was only modestly correlated in the current sample. There is also evidence that they are subserved by distinct neural mechanisms, and specifically that ToM reasoning involves higher-order prefrontal cortical areas and operates over a longer time course of neural activation relative to ToM decoding (e.g., Liu, Sabbagh, Gehring, & Wellman, 2009; Sabbagh, Mouison, & Harkness, 2004). Therefore, it is possible that higher-level cognitive biases, attributions, and other schematic processing that are applied to the more cognitively complex task of reasoning about others’ mental states operate similarly in SAD whether or not comorbid with MDD. Regardless of the exact mechanisms underlying the results reported here, they highlight the point made recently by Schaafsma, Pfaff, Spunt, and Adolphs (2015) that ToM is not a monolithic process, and instead is made up of multiple components operating at different levels of cognition. Further, they highlight the need for more research comparing comorbid and non-comorbid anxiety and depressive disorders across strong tasks of both ToM decoding and reasoning.

Contrary to hypotheses, we did not find evidence for significantly lower ToM decoding or reasoning accuracy in those with a non-comorbid lifetime MDD relative to healthy controls. As noted above, the current results may suggest that over-sensitive reasoning about the mental states of others in those with a lifetime diagnosis of MDD may be specific to those with a comorbid anxiety disorder characterized specifically by marked social fears and avoidance. However, neither the comorbid or non-comorbid lifetime MDD groups differed significantly from healthy controls in their mental state decoding. Previous studies have most consistently found deficits in ToM decoding among individuals in a current episode of MDD relative to controls. In contrast, while some studies have found lingering ToM deficits in severely depressed outpatients very recently remitted from their index episode (e.g., Inoue et al., 2004, 2006), other studies including undergraduate samples with a lifetime history of MDD similar to that assessed here have found that those with a history of MDD performed significantly more accurately on the Eyes task than never-depressed controls. Similarly, studies with undergraduate samples have found that those with current subthreshold symptoms, such as many in the current sample, also display an enhanced pattern of ToM decoding accuracy relative to controls (e.g., Harkness et al., 2005). In the current sam-

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Group means and standard deviations for the eyes and MASC tasks.</th>
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<tbody>
<tr>
<td></td>
<td>Lifetime MDD (n = 40)</td>
</tr>
<tr>
<td>----------------</td>
<td>----------------------</td>
</tr>
<tr>
<td>Eyes Accuracy</td>
<td>72.57 (8.21)</td>
</tr>
<tr>
<td>Positive Eyes</td>
<td>72.50 (18.32)</td>
</tr>
<tr>
<td>Negative Eyes</td>
<td>74.55 (13.93)</td>
</tr>
<tr>
<td>Neutral Eyes</td>
<td>71.25 (10.94)</td>
</tr>
<tr>
<td>Eyes Response Latency</td>
<td>4105.34 (114.55)</td>
</tr>
<tr>
<td>Animals Accuracy</td>
<td>72.71 (13.87)</td>
</tr>
<tr>
<td>MASC Accuracy</td>
<td>77.94 (8.29)</td>
</tr>
<tr>
<td>MASC Errors</td>
<td></td>
</tr>
<tr>
<td>Excessive ToM</td>
<td>11.94 (6.89)</td>
</tr>
<tr>
<td>Low ToM</td>
<td>6.61 (3.81)</td>
</tr>
<tr>
<td>No ToM</td>
<td>2.67 (2.48)</td>
</tr>
<tr>
<td>MASC Control</td>
<td>75.00 (14.12)</td>
</tr>
</tbody>
</table>

Notes: MASC = Movie for the Assessment of Social Cognition; ToM = Theory of mind.


ple, we did not have sufficient power to further stratify our Lifetime MDD group into those in a current MDD episode and those with past MDD. A direct comparison of these groups, taking into consideration the role of comorbid SAD, remains an important question for future research.

A further interesting and important area for future research regards the relation of dimensions of symptoms or affect to theory of mind skill. In the current study, continuous measures of depression and social anxiety symptoms failed to show significant associations to Eyes task and MASC task accuracy. On the one hand, this may suggest that a categorical syndrome approach is the best way to capture individual differences in theory of mind skill. On the other hand, this may suggest that a dimensional relation would be better captured by (a) using instruments that more clearly separate depressed versus anxious affect and/or symptoms; and (b) acknowledging that the relation of symptoms to theory of mind skill may be non-linear (e.g., Harkness et al., 2005).

Our results should be interpreted in the context of the following limitations. The size of the non-comorbid SAD group was small. Perhaps not surprisingly, this group was very difficult to find and recruit into the study. Therefore, replication with a larger sample is required. Given the small size of the sample, we were unable to examine other potential moderators of our effects. For example, although not statistically significant, the non-comorbid SAD group had the highest proportion of men and individuals of non-White ethnicity (see Table 1), two variables which affect ToM performance as well as face perception (e.g., Krach et al., 2009; Rehman & Helitz, 2006). While the hypothesized effects remained significant over and above the influence of sex and ethnicity, this does not rule out moderating effects. There was insufficient statistical power to examine sex and ethnicity as moderators of the relation of ToM ability and diagnostic status and, thus, this remains an important question for future investigation. Further, the sample consisted of undergraduate volunteers, and, thus the results reported here may not generalize to patient or general community samples. Nevertheless, all participants met DSM-IV criteria for psychiatric diagnosis according to a structured clinical interview. Finally, this study measured social anxiety symptoms using the SAASA. While this measure has strong psychometric properties and includes direct evidence of diagnostic validity, there was a significant negative effect on the prognosis of psychopathology and quality of life (Trompenaars, Masthoff, Van Heck, De Vries, & Hodiamont, 2007). Therefore, our results also suggest the need for treatment strategies for SAD that consider individual differences in social cognition. Fostering an optimal level of social sensitivity may lead to improvements in functioning that ultimately alleviate the interpersonal impairments that strongly characterize this chronic disorder.

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References


