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Attentional Blink Impairment in Social Anxiety Disorder: Depression Comorbidity Matters

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Abstract

Background and Objectives—Difficulties with attentional control have long been thought to play a key role in anxiety and depressive disorders. However, the nature and extent of attentional control difficulties in social anxiety disorder (SAD) are not yet well understood. The current study was designed to assess whether attentional control for non-emotional information is impaired in SAD when taking comorbid depression into account.

Methods—Individuals with SAD and healthy controls (HCs) were administered an attentional blink (AB) task in which they identified number targets in a rapid serial visual presentation stream of letters.

Results—Individuals with SAD and current comorbid depression exhibited reduced accuracy to identify a target that fell within the AB window after the presentation of a first target compared to individuals with SAD without current comorbid depression, as well as to HCs. The latter two groups did not differ from each other, and the three groups did not differ in accuracy for the second target when it was presented after the AB window.

Limitations—Although we included two clinical groups and the sample size for the noncomorbid SAD group was large, the comorbid SAD group was relatively small.

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Conclusions—These results suggest that impaired attentional control among individuals with SAD may be limited to those suffering from current comorbid depression.

Keywords

social anxiety disorder; attentional control; attentional blink; depression; comorbidity

1. Introduction

One mechanism thought to underlie social anxiety disorder (SAD) is attentional dyscontrol. In particular, heightened self-focused attention accompanied by undue capture of attention by social threat-relevant information in the environment is thought to contribute to difficulties maintaining attention on task-relevant goals in social situations (Clark & Wells, 1995; Heimberg, Brozovich, & Rapee, 2014). Moreover, post-event processing, or the ruminative review of one's actions and the reactions of others that occurs between social situations and that is typical of persons with SAD (Brozovich & Heimberg, 2008), has the potential to perpetuate attentional dyscontrol even outside of the context of social situations.

Attentional control theory (Eysenck & Derakshan, 2011; Eysenck, Derakshan, Santos, & Calvo, 2007) provides a useful framework for conceptualizing attentional processing in SAD. Attentional control theory posits that high levels of anxiety impair the goal-directed attentional system (i.e., attentional control) by increasing the influence of the stimulus-driven attentional system. This imbalance results in biased attention toward salient stimuli, typically defined in terms of central location in the visual field, but also in terms of threat relevance. Highly anxious individuals are purported to have difficulties inhibiting and shifting attention away from task-irrelevant stimuli, especially when such stimuli are threat-relevant (Eysenck et al., 2007). In light of the suggestion that attentional dyscontrol in SAD may extend into the relative calm that separates anxiety-provoking experiences, it is plausible that difficulties with attentional control may occur not only in the context of task-irrelevant threat distractors, but also in the context of neutral distracting information. Whereas there are many studies on the former hypothesis, albeit with sometimes inconsistent results, far less is known about the latter hypothesis. If the broader tenets of attentional control theory extend to SAD, this could illuminate a potential transdiagnostic mechanism involved in the maintenance of anxiety disorders. In what follows, we review the literatures on attentional control in the context of 1) emotional task-irrelevant (threat) distractors and 2) non-emotional task-irrelevant (neutral) distractors, attempting to unify the findings under the framework of attentional control theory.

Evidence of attentional dyscontrol in SAD in the context of emotional task-irrelevant stimuli comes primarily from studies of attention bias to threat. Meta-analytic results on the dot-probe task, spatial cuing task, and emotional Stroop task indicate a moderate between-group effect size of attention bias to threat in individuals with SAD compared to non-anxious individuals (d = .46; Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007). Biases toward threat in individuals with SAD or elevated social anxiety have also been reported in various other paradigms, such as the emotional antisaccade task (Wieser, Pauli, & Mühlberger, 2009), emotional attentional blink task (for review see Van

Bockstaele et al., 2014), and eye-tracking studies (e.g., Gamble & Rapee, 2010). Consistent with attentional control theory, observed biases have been attributed to biased engagement of attention with threat (enhanced stimulus-driven attention), difficulties disengaging attention from threat (impaired shifting away from threat), or both (e.g., Clarke, MacLeod, & Guastella, 2013).

Nevertheless, studies on attention bias to threat in SAD are not equivocal, with several studies reporting null results (e.g., Heeren, Mogoaşe, McNally, Schmitz, & Philippot, 2015) or attention biases *away* from threat (see Bögels & Mansell, 2004). These discrepancies are important, as they suggest the possibility of moderators of attention bias to threat. One likely moderator that is often neglected is depression comorbidity (Bar-Haim et al., 2007). Approximately 40-50% of individuals with a principal diagnosis of SAD also have major depressive disorder (MDD) or dysthymic disorder (Brown, Campbell, Lehman, Grisham, & Mancill, 2001), and depression has been associated with broad impairments in executive functioning (for a review, see Snyder, 2013). Indeed, attention bias toward threat in individuals with elevated social anxiety was nullified, or at least dampened, in those with comorbid depression (Grant & Beck, 2006; LeMoult & Joormann, 2012; Musa, Lépine, Clark, Mansell, & Ehlers, 2003).

In contrast to the vast attention bias literature, no study to date has examined whether attentional control in the context of neutral task-irrelevant stimuli in SAD is impaired. Three studies in undergraduate samples suggest this may be the case. In two studies, self-reported attentional control was negatively correlated with social anxiety, even after statistically controlling for the effects of depression (Moriya & Tanno, 2008; Morrison & Heimberg, 2013). In a third study, social anxiety was positively correlated with difficulty disengaging attention from non-emotional, task-irrelevant stimuli being held in working memory (Moriya & Sugiura, 2012). However, this effect was not moderated by working memory load. In theory, higher working memory load should be associated with a stronger association between anxiety and inhibitory difficulties, as attentional control resources are more consumed. Taken together, there is preliminary evidence of general attentional control difficulties in individuals with elevated social anxiety, but this research has been mostly limited to self-report studies, and the effects of depressive symptoms has only been considered through analyses of covariance, which may be inappropriate in this context (Miller & Chapman, 2001).[1]

To further our understanding of attentional dyscontrol in SAD, we sought to address the question of whether attentional control in the context of non-emotional stimuli is impaired in individuals with SAD, while also accounting for the often neglected and likely moderating effects of comorbid depression. Further, given criticisms of the psychometric properties of attention bias tasks often used in the anxiety disorders (e.g., Schmukle, 2005), we sought to use a well-established measure of attentional control, namely, the attentional blink (AB) task (see Martens & Wyble, 2010). The AB refers to the robust finding that accuracy to identify a

[1]Moriya and Tanno (2010, 2011) found that individuals high in social anxiety exhibited relatively *enhanced* attentional resources under high perceptual load. However, perceptual load is thought to modulate early attentional resources (e.g., at the level of visual cortex), so these findings do not reflect attentional control at the cognitive level (Berggren & Derakshan, 2013).

second target (T2) following a first target (T1) in a rapid serial visual presentation (RSVP) stream of non-targets is relatively reduced when the lag betweenT1 and T2 is short (200-500 ms) compared to when it is long (over 500 ms). The reduced accuracy for short-delay T2 is thought to result from a temporary loss of attentional control (Di Lollo, Kawahara, Ghorashi, & Enns, 2005).

In the current study, we examined AB performance in non-anxious, non-depressed healthy control participants (HCs) and in individuals with generalized SAD, either with or without current comorbid depression (MDD or dysthymic disorder). We hypothesized that, compared to HCs, individuals with SAD would (a) exhibit impaired attentional control (i.e., reduced accuracy for T2 presented within the AB window compared to a baseline condition) and (b) this relative impairment would be greatest for those with current comorbid depression.

2. Methods

2.1 Participants

Participants were 166 individuals who met Diagnostic and Statistical Manual for Mental Disorders, Fourth Edition (DSM-IV; American Psychiatric Association, 1994) criteria for a principal diagnosis of generalized SAD, as well as 37 healthy control (HC) participants who met no criteria for any current or past psychiatric disorders. Participants with SAD were recruited as part of one of two larger randomized controlled trials, either comparing cognitive behavioral group therapy (CBGT) with mindfulness-based stress reduction (MBSR) and a waitlist control condition (Goldin et al., in prep) or comparing MBSR to aerobic exercise (Jazaieri, Goldin, Werner, Ziv, & Gross, 2012). Of those with SAD, 26 (16%) met criteria for current comorbid MDD or dysthymic disorder (COM).

All SAD participants were excluded for any comorbid psychiatric disorders other than secondary diagnoses of generalized anxiety disorder, specific phobia, obsessive compulsive disorder, panic disorder, MDD, and dysthymic disorder. Additional exclusion criteria included previous completion of an MBSR course or regular meditation practice. For the Jazaieri et al. sample, individuals were also excluded for previous regular physical exercise practice, and for the Goldin et al. sample, past CBT for SAD. All participants were required to be between 21 and 55 years of age, speak fluent English, pass a magnetic resonance imaging safety screen, be right-handed, and report no current pharmacotherapy, current psychotherapy, or history of neurological or cardiovascular disorders. Participants provided informed consent in accordance with the Institutional Review Board at Stanford University. Participants with SAD were offered free treatment and HC participants were provided modest financial compensation.

2.2 Measures

The Anxiety Disorders Interview Schedule for the DSM-IV - Lifetime version (ADIS-IV-L; Di Nardo, Brown, & Barlow, 1994) is a semi-structured interview for the diagnosis of anxiety and related disorders. It has demonstrated excellent reliability for a principal

diagnosis of SAD (κ = .77) and good reliability for a current principal or additional diagnosis of MDD/dysthymia (κ = .63; Brown, Di Nardo, Lehman, & Campbell, 2001).

The *Beck Depression Inventory-2nd Edition* (BDI-II; Beck, Steer, & Brown, 1996) is a 21-item scale that assesses the symptoms of depression as listed in the DSM-IV. The BDI-II has demonstrated high internal consistency (α = .92), as well as good convergent and discriminant validity (e.g., Beck, Steer, Ball, & Ranieri, 1996).

The Liebowitz Social Anxiety Scale - Self-Report version (LSAS-SR) is a self-report version of the LSAS (Liebowitz, 1987). It includes 24-items that assess fear and avoidance in a range of social and performance situations. The LSAS-SR has shown excellent internal consistency (α = .95) and strong convergent and discriminant validity among individuals with a primary diagnosis of SAD (Fresco et al., 2001).

2.3 Attentional Blink (AB) Task

The AB task was programmed in E-Prime 2.0 (Psychology Software Tools, Pittsburgh, PA) and was a shortened version of the task reported in Slagter et al. (2007). Stimuli were presented in the center of a gray screen in black. On each trial, a fixation cross was presented for 1,780 ms, followed by an RSVP stream of 15 or 19 items. Letters (non-targets) were drawn randomly from the alphabet without replacement, excluding B, I, O, Q, and S. One or two numbers (targets) between 2 and 9 were drawn randomly and without replacement and presented within the RSVP. Each item was presented for 50 ms, with a 34-ms blank screen separating the items. On dual-target trials, the second target (T2) followed the first target (T1) by either 336 ms (short-delay) or 672 ms (long-delay). T2 was always presented 3-5 items from the end of the stream. On single-target trials, a blank screen replaced T2 at either a short- or long-delay. Excepting the last letter of the sequence and those immediately surrounding T1 and T2, each letter also had a 20% probability of being replaced with a blank screen, in order to reduce the salience of the blank screen. Following each RSVP stream, participants were asked to use the keypad to enter the target(s) in the order presented. Participants were told there would be one or two numbers and to guess if they were uncertain about a number's identity. If they were certain a single number was presented, they were instructed to enter “0” for the second number. The next trial began 200 ms after their response. Participants completed 51 trials, with the four trial types presented randomly, resulting in a variable number of each trial type per participant (*M* (*SD*) of each trial type: short-delay dual-target = 12.9 (3.4); short-delay single-target = 12.8 (3.3); long-delay dual-target = 12.6 (3.0); long-delay single-target = 12.9 (3.1)). T2 performance was conditioned on T1 performance (T2|T1).

2.4 Procedure

Participants were recruited through referrals, web listings, and community flyers. HC participants and SAD participants from the Goldin et al. study first completed an online screener that included the LSAS-SR. All participants completed a telephone interview and were invited for an in-person appointment to complete the ADIS-IV-L interview and BDI-II. SAD participants in the Jazaieri et al. sample completed the LSAS-SR during the in-person diagnostic appointment. All participants completed the AB task during a subsequent

behavioral session. SAD participants were randomized to their respective treatment conditions following completion of all baseline assessments.

3. Results

3.1 Preliminary Analyses

Demographic and clinical characteristics of participants are presented in Table 1. The three groups did not differ significantly in sex distribution, $\chi^2(2, N = 203) = 3.61$, $p = .17$, $V = .09$; age, $F(2, 200) = 0.37$, $p = .69$, $\eta_p^2 = .004$; years of education, $F(2, 194) = 1.38$, $p = .26$, $\eta_p^2 = .01$; or race/ethnicity composition (i.e., white versus non-white), $\chi^2(2, N = 187) = 2.32$, $p = .31$, $V = .08$. As expected, the groups differed significantly in their depression scores, $F(2, 183) = 37.53$, $p < .001$, $\eta_p^2 = .29$; the COM group had the highest BDI-II scores, followed by the SAD group, followed by the HC group, ps < .005. As expected, the groups differed significantly in their social anxiety scores, $F(2, 196) = 313.27$, $p < .001$, $\eta_p^2 = .76$; the HC group had significantly lower LSAS-SR scores than the SAD and COM groups, ps < .001, whose scores did not differ from each other, $t(160) = 1.03$, $p = .31$, $d = 0.16$.

3.2 AB Effect

An AB effect is reflected by contrasting short-lag T2|T1 accuracy with accuracy for a baseline condition (MacLean & Arnell, 2012). Most often, long-lag T2|T1 accuracy is used as the baseline; however, MacLean and Arnell (2012) explain that this baseline may underestimate the AB effect, particularly in samples with perceptual or cognitive impairment (e.g., Husain, Shapiro, Martin, & Kennard, 1997). Given findings of impaired executive functioning in depression, we used an additional baseline suggested by MacLean and Arnell (2012), that is, T1 accuracy.

A 3 group (COM, SAD, HC) X 3 target (short-delay T2|T1, long-delay T2|T1, T1) mixed model Analysis of Variance (ANOVA) on percent accuracy revealed a significant main effect of group, $F(2, 200) = 4.89$, $p < .01$, $\eta_p^2 = .05$, and a significant main effect of target, $F(2, 400) = 323.52$, $p < .001$, $\eta_p^2 = .62$. The signature AB effect is lower accuracy for short-delay T2|T1 relative to the baseline, which was true for all three groups relative to both long-delay T2|T1 as baseline [HC $t(36) = 5.71$, $p < .001$, $d = 0.94$; SAD $t(139) = 16.89$, $p < .001$, $d = 1.43$; COM $t(25) = 9.70$, $p < .001$, $d = 1.90$] and relative to T1 as baseline [HC $t(36) = 8.71$, $p < .001$, $d = 1.43$; SAD $t(139) = 25.27$, $p < .001$, $d = 2.14$; COM $t(25) = 16.00$, $p < .001$, $d = 3.14$]. See Table 2 for mean accuracy rates for each of the three target types by group.

The predicted interaction of group X target was also significant, $F(4, 400) = 2.52$, $p = .04$, $\eta_p^2 = .025$. See Figure 1. Follow-up tests contrasting short-delay T2|T1 accuracy to each of the baselines were first conducted. A 3 group X 2 target (short-delay T2|T1, long-delay T2|T1) ANOVA revealed an interaction that did not quite reach significance, $F(2, 200) = 2.41$, $p = .09$, $\eta_p^2 = .02$, whereas a 3 group X 2 target (short-delay T2|T1, T1) ANOVA yielded a significant interaction, $F(2, 200) = 3.87$, $p = .02$, $\eta_p^2 = .04$. Follow-up tests comparing groups

on each of the three targets was then conducted. A one-way ANOVA comparing the three groups' accuracy for short-delay T2|T1 was significant, $F(2, 200) = 6.42$, $p = .002$, $\eta_p^2 = .06$. The COM group was significantly less accurate than the other two groups (vs. HC: $t(61) = 3.44$, $p = .001$, $d = 0.88$; vs. SAD: $t(164) = 2.80$, $p = .006$, $d = 0.44$). In contrast, the SAD group did not differ from the HCs, $t(175) = 1.79$, $p = .08$, $d = 0.27$. Because the SAD and COM groups did not differ on the LSASSR, it is unlikely that the relatively poorer performance of the COM group was due to greater social anxiety. No significant group differences were found for either of the baseline conditions [long-delay T2|T1: $F(2, 200) = 1.19$, $p = .31$, $\eta_p^2 = .01$; T1: $F(2, 200) = 2.13$, $p = .12$, $\eta_p^2 = .02$], indicating that group differences in processing of targets was limited to targets occurring within the AB window.

4. Discussion

Individuals with SAD have been characterized as having difficulties maintaining attention on task-relevant goals when in the context of social threat (e.g., Heimberg et al., 2014). To examine whether these difficulties controlling attention (1) extend beyond contexts of immediate social threat and (2) are magnified in individuals with comorbid depression, we had HC participants and participants with SAD complete the standard AB task, in which attentional control is indicated by one's ability to accurately identify a second target that appears in close temporal proximity to a first target in an RSVP stream.

Individuals with SAD exhibited impaired attentional control relative to HCs *only* if they also had current comorbid depression. Those with SAD without current depression did not differ from HC participants in their accuracy for identifying the target in the AB window, whereas the comorbid SAD group exhibited reduced accuracy for this target relative to both HC participants and individuals with SAD without current depression. Comorbid SAD participants did not score higher on a measure of social anxiety than SAD participants without depression, suggesting the observed difference was not due to differences in social anxiety severity. Moreover, the groups did not differ in their accuracy for two baseline comparison conditions, namely, identification of a second target that fell beyond the AB window or identification of the first target, which supports an interpretation that impaired processing in the COM group was due to attentional control difficulties rather than broader perceptual or cognitive processing difficulties.

To the best of our knowledge, there are no studies examining this type of non-emotional AB effect in any anxiety disorder. With relevance to social anxiety, one study administered an emotional variant of the AB task to undergraduate students high or low in social anxiety (de Jong, Koster, van Wees, & Martens, 2009). Social anxiety group did not moderate the attenuated AB that occurred when T2 stimuli were emotional versus neutral facial expressions, although these results are not directly relevant to the current study given that stimuli were threat-relevant. More relevant to the current study is evidence from unselected samples that individual differences in personality traits predict AB magnitude. Greater negative affect and neuroticism are associated with a larger AB (i.e., reduced attentional control), whereas greater positive affect, extraversion, and openness are associated with a smaller AB (MacLean & Arnell, 2010; MacLean et al., 2010). Similarly, Rokke et al. (2002)

found that moderately-to-severely dysphoric college students exhibited impaired AB performance relative to nondysphoric and mildly dysphoric students, and the authors noted that these results could have been as likely due to group differences in state anxiety as to differences in depression.

Our finding of poorer AB performance in the comorbid group compared to the HC group aligns with this previous literature, but the similar performance between the non-comorbid SAD group and HC group is surprising, given that SAD has been generally characterized by both high negative affectivity/neuroticism and low positive affectivity/extraversion (for a review, see Naragon-Gainey & Watson, 2011). Several studies suggest, however, that whereas social anxiety is associated with low levels of multiple facets of positive affect/extraversion, depression is related to only one facet, that is, low positive emotionality (Naragon-Gainey, Watson, & Markon, 2009). Conversely, depression symptoms show a stronger relationship with neuroticism/negative affect than do social anxiety symptoms (Watson & Naragon-Gainey, 2014). Therefore, the current results suggest that particular *combinations* of personality traits may be a better predictor of AB performance than single personality traits, or that particular personality traits confer greater risk for poor AB performance than others. Future research will be needed to tease apart the unique and combined effects of specific temperaments and their facets on AB performance.

The current results may also inform research on attentional control in anxiety disorders. Our results are consistent with previous studies showing reduced attentional control in the context of non-threat distractors in socially anxious individuals (Moriya & Tanno, 2008; Moriya & Sugiura, 2012; Morrison & Heimberg, 2013). However, the current findings raise the possibility that previous findings were driven by the high degree of overlap between social anxiety and depression. Both of the self-report studies noted that statistically accounting for depression did not alter the negative relationship between social anxiety and attentional control, but the multiple groups method used in the current study is a more valid procedure for assessing the effects of comorbidity (Miller & Chapman, 2001). Of note, much of the research on attentional control in high trait anxious individuals suffers from this same limitation of overlooking anxiety-depression comorbidity. Determining whether anxiety contributes to attentional dyscontrol over and above depression represents an important research direction.

Considered in the context of attentional control theory, the current results suggest that elevated social anxiety is not sufficient to impair attentional control in the context of neutral task-irrelevant stimuli. Attentional control theory holds that elevated trait anxiety leads to (1) heightened capture of attention by salient stimuli and (2) reduced ability to shift attention away from or inhibit attention to distracting stimuli. Although evidence for both of these phenomena has been found in individuals with elevated trait anxiety (Eysenck et al., 2007), the relevance of this theory to social anxiety has been largely untested, except for attentional control in the context of threatening stimuli (i.e., attention bias to threat), which has been studied extensively. Even so, evidence for attention bias to threat in SAD has been mixed. Indeed, a recent review of the attention bias literature went so far as to conclude that "attentional bias in social anxiety is a relatively uncommon finding across different paradigms" (p. 695; Van Bockstaele et al., 2014). Together, results of the current study and

studies of attention bias to threat call into question the applicability of attentional control theory to elevated social anxiety per se.

The current study had a number of notable strengths, including the large clinical sample of individuals with SAD and a well-established measure of temporal attentional control. However, several limitations bear noting. Our comorbid SAD group was relatively small compared to our non-comorbid SAD group. Additionally, our AB task was relatively brief. It is conceivable that the duration of an attentional control task could alter results, as the performance of individuals with poor attentional control ability could deteriorate over longer periods as attention must be sustained or could improve due to practice effects. It is also difficult to know from behavioral studies, such as our own, whether the lack of difference in accuracy between the HC and non-comorbid SAD groups was due to differential exertion of effort, an argument made by Eysenck and colleagues in their attentional control theory. Finally, the AB task may assess a specific subcomponent of attentional control, which suggests that future studies should examine whether different aspects of attentional control are impaired in non-comorbid SAD. Such research will likely refine our understanding of mechanisms that maintain SAD, as well as point to more specific intervention targets for individuals with different comorbidities.

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Highlights

- We examined attentional control in social anxiety disorder (SAD) with the attentional blink task.
- SAD participants with comorbid depression exhibited reduced attentional control.
- SAD participants without comorbid depression did not differ from non-anxious, healthy controls.

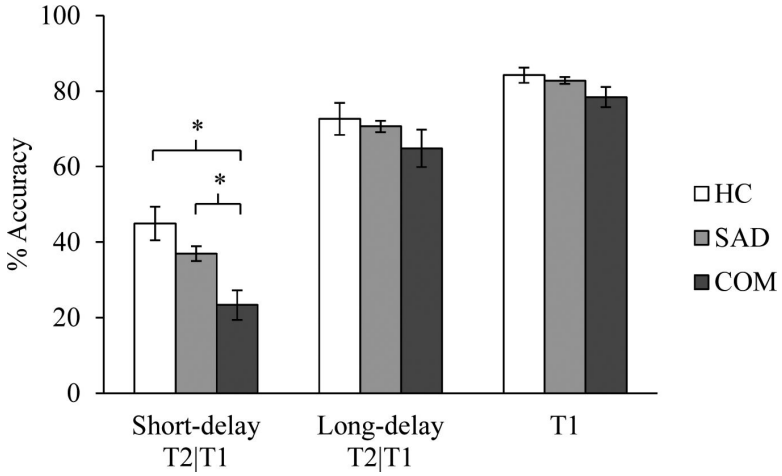

Figure 1.

Mean percentage of accurate T2|T1 responses for short-delay (336 ms) dual-target trials, T2|T1 responses for long-delay (672 ms) dual-target trials, and T1 responses in healthy control (HC) participants, participants with SAD without comorbid depression (SAD), and participants with SAD with comorbid depression (COM). Error bars are standard errors.

Table 1

Participant characteristics by diagnostic group

	HC (*n* = 37)	SAD (*n* = 140)	COM (*n* = 26)
Age, years (SD)	32.1 (8.6)	32.8 (8.2)	33.9 (7.8)
Education, years (SD)	17.4 (2.8)	16.7 (2.5)	16.4 (2.4)
Sex (% female)	59.5	57.9	38.5
% Race/Ethnicity			
Asian-American	33	41	31
Black/African-American	0	1	0
Hispanic/Latino	8	7	11
White/Caucasian	51	43	54
Other	8	8	4
LSAS-SR	14.2 (9.5)	89.0 (18.0)	93.0 (16.5)
BDI-II	1.5 (2.6)	10.0 (9.4)	21.4 (9.2)

Note: HC = healthy control group; SAD = social anxiety disorder and no current depression group; COM = social anxiety disorder and comorbid depression group; LSAS-SR = Liebowitz Social Anxiety Scale, Self-Report Version; BDI-II = Beck Depression Inventory, 2nd Edition. Standard deviations in parentheses.

Table 2

Mean percentage of accurate identifications of targets by target type and group

	HC	SAD	COM
Short-delay T2|T1	44.9 (27.3)	36.9 (23.2)	23.3 (19.9)
Long-delay T2|T1	72.6 (25.7)	70.7 (17.9)	64.8 (25.3)
T1	84.2 (12.2)	82.8 (10.7)	78.4 (13.6)

Note: HC = healthy control group; SAD = social anxiety disorder and no current depression group; COM = social anxiety disorder and comorbid depression group; T1 = first target in rapid serial visual presentation stream; T2 = second target. Standard deviations in parentheses.