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Anxiety trajectories in response to a speech task in social anxiety disorder: Evidence from a randomized controlled trial of CBT

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Abstract

The subjective experience of anxiety plays a central role in cognitive behavioral models of social anxiety disorder (SAD). However, much remains to be learned about the temporal dynamics of anxiety elicited by feared social situations. The aims of the current study were: 1) to compare anxiety trajectories during a speech task in individuals with SAD (n = 135) versus healthy controls (HCs; n = 47), and 2) to compare the effects of CBT on anxiety trajectories with a waitlist control condition. SAD was associated with higher levels of anxiety and greater increases in anticipatory anxiety compared to HCs, but not differential change in anxiety from pre- to post-speech. CBT was associated with decreases in anxiety from pre- to post-speech but not with changes in absolute levels of anticipatory anxiety or rates of change in anxiety during anticipation. The findings suggest that anticipatory experiences should be further incorporated into exposures.

Keywords

social anxiety disorder; behavioral assessment test; latent growth curve modeling; cognitive behavioral therapy

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Social anxiety disorder (SAD) has the second highest 12-month prevalence rate of anxiety, mood, and substance use disorders at 6.8%, just behind specific phobia at 8.7% (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). SAD is associated with social and occupational impairment and considerable comorbidity with other psychiatric conditions, resulting in tremendous personal, economic, and societal costs (Acarturk et al., 2009; Aderka et al., 2012; Stein & Kean, 2000). Although cognitive behavioral therapy (CBT) for SAD has been shown to be generally effective, many people with SAD do not benefit from CBT (e.g., Acarturk, Cuijpers, van Straten, & de Graaf, 2009; Gordon, Wong, & Heimberg, 2014; Mayo-Wilson et al., 2014). A better understanding of the basic mechanisms underlying SAD is necessary to enhance existing treatments and to better tailor treatments to the needs of different individuals. In the present study, we focus on elucidating the temporal course of subjective anxiety experienced in response to feared social situations before and after CBT.

Anxiety Reactivity in SAD and its Treatment: Theoretical Expectations

Cognitive behavioral models of SAD focus on the antecedents and consequences of anxiety (Clark & Wells, 1995; Heimberg, Brozovich, & Rapee, 2014; Hofmann, 2007). Common among these models is the notion that anxiety acts in a self-perpetuating manner. Anxious thoughts, physiological sensations, feelings, and behaviors interact with one other and contribute to negative interpersonal outcomes, thus confirming negative schemas and reinforcing the cycle. In contrast, less attention has been paid to characterizing the temporal course of subjective anxiety response as an individual with SAD anticipates, confronts, engages in, and terminates a highly feared social situation and how this temporal course of anxiety reactivity to a highly feared situation changes following a behavioral treatment intended to reduce subjective anxiety.

These are important areas of study for at least two reasons. First, the subjective experience of anxiety holds a prominent theoretical role in the maintenance of SAD. High anticipatory anxiety is believed to lead to avoidance, which is perpetuated by negative reinforcement (Foa & Kozak, 1986). Similarly, high anxiety during social situations may contribute to a greater reliance on safety behaviors to manage one's anxiety, which may prevent safety learning and have unintended negative interpersonal consequences (Clark & Wells, 1995). Likewise, high anxiety after social situations is thought to be associated with more negatively biased post-event processing and perceived confirmation of failure, thus increasing the likelihood of future avoidance (Heimberg et al., 2014). Nevertheless, we do not yet know whether and how individuals with SAD differ from non-anxious individuals in their patterns of anxious responding to feared social situations. The primary difference may be in the absolute levels of anxiety (e.g., mean or peak anxiety). Individuals with SAD may experience consistently elevated state anxiety throughout anticipation, performance, and termination, showing little to no decline or habituation in anxiety. Alternatively, SAD may be associated with greater reactivity, such as steeper inclines in anxiety during anticipation and/or performance. It is conceivable that high reactivity to social situations could be more detrimental than high but static anxiety responses. Drastic shifts in anxiety could be associated with more intrusive physiological sensations, which may lead to greater self-

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focused attention (see a similar conceptualization of generalized anxiety disorder by Newman and Llera, 2011).

Second, illuminating the temporal course of anxiety may inform treatments for SAD. Theoretical models suggest that effective exposure therapy should be associated with decreases in state anxiety to feared situations. Therapeutic exposure is thought to provide instances of safety learning, which is believed to compete with fear associations in memory (Bouton, 2002; Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014). These decreases in anxiety are believed to combine with motivational factors to reduce avoidance. Although these theories do not make specific predictions about whether absolute levels of state anxiety or rates of change in anxiety will be modified during treatment, the study of such state anxiety trajectories could provide useful information. For example, if the greatest distinguishing characteristic between the anxiety trajectories of individuals with SAD and non-anxious individuals is the rates of change in anticipatory anxiety, and if exposure-based therapy does not affect rates of change in anticipatory anxiety, this would suggest treatment might be improved by increasing focus on anticipatory processing, such as facilitating the client's use of field perspective imagery during anticipation (M. Brown & Stopa, 2007).

Anxiety Reactivity in SAD: Empirical Findings

Although no study has compared the temporal course of anxiety in individuals with SAD to the course in non-anxious individuals, there are several relevant studies that begin to inform this question. These studies have used behavioral assessment tests (BATs; McNeil, Ries, & Turk, 1995), or laboratory-based social interactions or performance situations, often a speech task. These studies and the current study should be differentiated from studies that have assessed patterns of anxiety response during exposures (e.g., Hayes, Hope & Heimberg, 2008). Whereas the purpose of the latter is to examine how anxiety/fear changes during a therapeutic context that is designed to promote emotional processing (e.g., habituation), corrective learning, or both, the purpose of BATs is to assess anxiety severity and treatment progress.

Compared with non-anxious individuals, individuals with SAD have reported higher mean anxiety, higher peak anxiety, and higher anxiety at multiple time points during different phases of a BAT (Beidel, Rao, Scharfstein, Wong, & Alfano, 2010), although the effects have sometimes been limited to individuals with comorbid depression (Yoon & Joormann, 2012). These studies suggest that SAD is associated with elevated *absolute levels* of anxiety during BATs, but they do not address whether the groups differ in their anxiety *reactivity* (i.e., rates of change). Relevant to anxiety reactivity, two studies have provided evidence that the generalized subtype of SAD is associated with greater increases in subjective anxiety in response to feared social situations relative to controls, but neither study distinguished between reactivity during the anticipatory and performance phases of the BAT (Hofmann, Gerlach, Wender, & Roth, 1997; Levin et al., 1993).

More recently, two studies have provided a window into the temporal course of anxiety in SAD, although neither included a control group. In a hierarchical cluster analysis examining patterns of anxious responding in SAD, four pattern clusters emerged, with differential

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levels of elevation during anticipation and performance and differential rates of change during anticipation and performance (Coles & Heimberg, 2000). Notably, fear and avoidance of social interaction and social performance situations were most closely associated with levels of anxiety during the performance phase of the BAT; however, they were not closely associated with levels of anxiety during the anticipation phase, or rates of change in anticipatory or performance anxiety. Further, the class characterized by steep increases in anticipatory anxiety and high levels of performance anxiety fared better in CBT than a class with lesser increases in anticipatory anxiety and moderate levels of anxiety during performance. This finding suggests greater increases in anticipatory anxiety may be predictive of better response to CBT. In a second study, which used growth curve analyses of anxiety responses during a BAT before and after CBT, anxiety increased from baseline through anticipation and performance. Notably the rate of increase was highest during early anticipation, lesser from later anticipation to confrontation of the stressor, and lesser still from confrontation to the speech performance (Price & Anderson, 2011). Taken together, these studies indicate that there may be important individual differences in how individuals with SAD subjectively experience anxiety prior to, during, and immediately following feared social situations, and that the delineation of these patterns has the potential to illuminate mechanisms that might be better targeted during treatment.

Effect of Treatment on Anxiety Reactivity in SAD: Empirical Findings

Evidence to date is mixed with regard to the effects of treatment on anxiety responses during BATs in SAD. In a recent study comparing CBT to Acceptance and Commitment Therapy (ACT) and a waitlist control, decreases in mean state anxiety from pre- to post-treatment/ waitlist did not differ across the three conditions (Craske, Niles, et al., 2014). This suggests that time or repeated exposure to the BAT context was sufficient to result in lower absolute levels of anxiety. However, several other studies have provided support for the effects of treatment on anxiety during the BAT. In one study, enhanced CBT (augmented with several theoretically derived treatment techniques such as attention retraining), but not traditional CBT, resulted in lower post-speech anxiety compared to a non-specific stress management treatment (Rapee, Gaston, & Abbott, 2009). In a second study, treatment with phenelzine or cognitive behavioral group therapy (CBGT) was associated with reductions in mean performance anxiety during an idiographic BAT from pre- to post-treatment as compared to an educational-supportive control condition and pill placebo (Heimberg et al., 1998). Neither active treatment was associated with greater reductions in mean anticipatory anxiety compared to the control treatments. Similarly, Price and Anderson (2011) found minimal attenuation of increases in anticipatory anxiety but significant decreases in anxiety during the course of performance at post-treatment, but these results should be interpreted with some caution given the lack of a control group. Finally, one study has provided evidence that absolute levels of anxiety and rates of change in anticipatory anxiety are reduced following treatment. Compared to those in an educational supportive control condition, patients with SAD who received CBGT exhibited lower anxiety at each of three anticipatory and each of five performance time points at post-treatment, controlling for pre-treatment (Heimberg et al., 1990). Moreover, at post-treatment, participants in the control treatment

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showed an increase in anxiety during anticipation, whereas those in the CBGT group showed no increase in anxiety during anticipation.

The Present Study

Anxiety responses to feared social situations play a prominent theoretical role in cognitive behavioral models of SAD. Nevertheless, no study has examined whether the pattern of anxious responding among individuals with SAD differs from that of non-anxious individuals. Also, although there is fairly consistent evidence that CBT is associated with lower absolute levels of anxiety during performance, absolute levels of anxiety during anticipation has been shown to be unchanged following CBT (Heimberg et al., 1998) or lower following CBT (Heimberg et al., 1990). Likewise, whereas one study indicates minimal pre- to post-treatment changes in rates of change of anxiety during anticipation and notable reductions in anxiety during performance (Price & Anderson, 2011), another suggests notable decreases in anxiety during anticipation and performance (Heimberg et al., 1990). Given that the evidence regarding changes in patterns of anxiety response appears to be partially dependent on the inclusion of a control group, and that the educational supportive control group of the Heimberg et al. studies may contain some active components, we compared the effects of CBT to a waitlist control condition. We also chose to use a speech rather than a social interaction or an idiographically-selected task because it is an almost universally feared situation among persons with SAD and we wished to provide a uniform feared stimulus to all participants.

In the current study, we used latent growth curve modeling to examine: 1) patterns of anxious responding from anticipation through completion of a speech task in individuals with SAD compared to non-anxious, healthy control individuals, and 2) the effects of CBT versus a waitlist control condition on these patterns of anxious responding in individuals with SAD. Based on prior literature, we hypothesized that for those with SAD at baseline, anxiety would increase from baseline through the anticipation phase, peak at the point of confrontation (i.e., immediately pre-speech; latent intercept), and then plateau from confrontation to completion (i.e., immediately post-speech). For non-anxious controls, we hypothesized an attenuated trajectory in which rates of incline in anxiety during anticipation (i.e., slope) were significantly less steep, resulting in a significantly lower absolute level of anxiety at the point of confrontation. No specific predictions were made regarding between group differences in the rates of change (i.e., slope) in anxiety from confrontation to completion of the speech. For the second aim, we hypothesized that, compared to a waitlist control condition, CBT for SAD would result in a modest reduction in the rate of change in anxiety during anticipation of the speech (i.e., less steep positive slope), a lower absolute level of anxiety at the point of confrontation (i.e., lower intercept), and a decline in anxiety from confrontation to completion of the speech (i.e., steeper negative slope).

Method

Participants

Participants consisted of 135 individuals who met *Diagnostic and Statistical Manual for Mental Disorders, Fourth Edition* (DSM-IV; American Psychiatric Association, 1994)

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criteria for a principal diagnosis of generalized SAD and 47 healthy control (HC) individuals who did not meet DSM-IV criteria for any current or past psychiatric disorders (see Table 1 for demographic and clinical variables). All participants with SAD were recruited as part of one of two randomized controlled trials (RCTs) for the treatment of SAD at Stanford University: (1) individual CBT versus a waitlist (WL) condition (as reported in Goldin et al., 2012), or (2) mindfulness-based stress reduction (MBSR) versus an aerobic exercise condition (as reported in Jazaieri, Goldin, Werner, Ziv, & Gross, 2012). For analyses of the baseline speech task (i.e., Aim 1), just over half of the SAD participants were from the Goldin et al. study (n = 75) and the remaining participants were from the Jazaieri et al. study (n = 60). All measures were completed prior to randomization to treatment condition in both trials. Therefore, the SAD sample in the present study was comprised of all individuals who completed the speech task, regardless of whether they completed all baseline assessments or were eventually randomized to treatment. For analyses of the effect of CBT on anxiety during the BAT (i.e., Aim 2), participants were from the Goldin et al. sample and completed the BAT a second time at post-treatment (n = 32) or post-waitlist (n = 32).

To determine eligibility for the respective RCTs, all participants were administered the Anxiety Disorders Interview Schedule for the DSM-IV-Lifetime version (ADIS-IV-L; Di Nardo, Brown, & Barlow, 1994). For inclusion in the RCTs, participants with SAD were required to meet diagnostic criteria for a principal diagnosis of generalized SAD. Participants were excluded for comorbid psychiatric disorders other than secondary diagnoses of generalized anxiety disorder, obsessive compulsive disorder, panic disorder, agoraphobia without a history of panic attacks, specific phobia, major depressive disorder, or dysthymia. In the Goldin et al. sample, participants were also excluded for current major depressive disorder or obsessive compulsive disorder, or previous CBT experience. In the Jazaieri et al. sample, participants were excluded for previous completion of an MBSR course or regular meditation or exercise practices. HC individuals were eligible if they did not meet DSM-IV criteria for any current or past psychiatric diagnoses as assessed by the ADIS-IV-L.

In addition to these exclusion criteria, all participants were required to be 21–55 years of age and speak fluent English. Because all data were collected as part of larger neuroimaging studies, participants were required to be right-handed as assessed by the Edinburgh Handedness Inventory (Oldfield, 1971), pass a magnetic resonance imaging (MRI) safety screen, and be free of current pharmacotherapy or psychotherapy, history of medical disorders, head trauma, and neurological disorders. All participants provided informed consent in accordance with the Institutional Review Board at Stanford University. Participants with SAD were offered free treatment. HC participants were provided modest financial compensation.

Interview and Self-Report Measures

Diagnostic Interview—The *Anxiety Disorders Interview Schedule for the DSM-IV-Lifetime version* (ADIS-IV-L; Di Nardo et al., 1994) is a reliable, semi-structured interview for the diagnosis of anxiety and related disorders (T. Brown, Di Nardo, Lehman, & Campbell, 2001). Specifically, in a sample of patients with a range of anxiety disorders it

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has shown excellent reliability for a principal diagnosis of SAD (κ = .77, T. Brown et al., 2001). The ADIS-IV-L was administered by experienced clinicians who held at least master's degrees in clinical psychology. They were trained to conduct the interview according to standards set forth by T. Brown et al. (2001) by watching training tapes and completing training interviews.

Self-Report Symptom Measures—The *Liebowitz Social Anxiety Scale-Self-Report version* (LSAS-SR; Fresco et al., 2001; Liebowitz, 1987; Rytwinski et al., 2009) was administered to assess social anxiety in a range of social and performance situations. The 24-item questionnaire measures how much fear one experiences and how often one avoids a particular situation during the past week on separate 4-point scales (e.g., 0 = "*none/never"*, 3 = "*severe/usually"*). Sample items include '*going to a party*,' '*speaking up in a meeting*,' and '*resisting a high pressure sales person.*' The total score is the sum of the 24 fear ratings and 24 avoidance ratings. The LSAS-SR has shown excellent internal consistency in previous studies (α = .95; Fresco et al., 2001), as well as in the present sample (HC: α = .92, SAD: α = .92). It has also demonstrated good convergent validity with other self-report measures of social anxiety as well as strong discriminant validity among socially anxious individuals (Fresco et al., 2001).

The *Beck Depression Inventory-2nd Edition* (BDI-II; Beck, Steer, & Brown, 1996) is a 21-item self-report instrument designed to assess the severity of DSM-IV symptoms of depression, including cognitive, affective, and somatic components. The respondent rates the severity of each symptom over the past two weeks on a scale of 0 to 3, with higher scores indicating greater severity. Beck et al., (1996) reported high internal consistency of the BDI-II among college students, α = .93, and outpatients, α = .92, with high one-week test-retest reliability among outpatients, *r* = .93, as well as good convergent and discriminant validity. Internal consistency in the present study was strong, HC: α = .84, SAD: α = .93.

Behavioral Assessment Test (BAT)

The BAT protocol was identical for all participants and involved the delivery of a brief speech in front of an experimenter. During the consenting process, participants were informed of a "behavioral session" during which they would be audio- and video-recorded, although they were not explicitly informed of a speech. After they completed two computer tasks not reported in the current study, they were asked to give two video-taped, two-minute speeches in front of the experimenter, although only data from the first speech were analyzed in the current study. [1] Participants were then provided more thorough instructions for the first speech. Specifically, they were informed that they should give a speech introducing themselves. They were told they would be given two minutes to prepare for the speech, during which time the experimenter remained in the room and began video-recording. After the preparation period, the experimenter instructed the participant to begin the speech. If the participant was unable to speak for the entire two minutes, he or she was

[1]Following the first speech, participants were provided instructions about and completed the second speech, in which they discussed a recent social anxiety-provoking situation. Data from the second speech are not presented in this study because the second speech was administered immediately after the first. For this reason, there was only one anxiety assessment administered during the "anticipatory" phase of the second speech. This precluded a similar growth curve modeling analysis of the second speech.

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informed that the camera would continue recording for the entire two minutes. Experimenters were instructed that while recording the speech, they were to look at the participant, hold a gentle smile, and not maintain a fixed stare.

Participants' anxiety during the BAT was assessed using the *Subjective Units of Distress Scale* (SUDS; Wolpe, 1969), a Likert-type scale ranging from 0 (no fear, anxiety) to 100 (extreme fear, anxiety). Participants were familiarized with the scale during their diagnostic interview at a prior appointment and re-familiarized at the beginning of this session. Individual SUDS ratings were elicited verbally by the experimenter four times: (1) at the beginning of the appointment (SUDS1), (2) immediately after being informed of the speech (pre-preparation; SUDS2), (3) following the two-minute preparation period and immediately prior to initiating the speech (pre-speech; SUDS3), and (4) immediately after the speech (post-speech; SUDS4). No assessments were collected during the speech. The SUDS scale is commonly used in CBT and in a variety of BATs (Fairbank & Keane, 1982; Rowe & Craske, 1998).

CBT RCT for SAD

Participants from the Goldin et al. sample who were randomized to immediate treatment received CBT for SAD based on the therapist manual by Hope, Heimberg, and Turk (2006). The individual treatment was completed over the course of 16 weeks in 1-hr weekly sessions, with one 1.5-hr session for the first in-session exposure. Treatment consisted of five phases: a) psychoeducation, approximately 4 sessions, b) cognitive restructuring training, 2–3 sessions, c) in-session and *in vivo* exposure, 8–9 sessions, d) advanced core beliefs work, 1–2 sessions, and e) treatment consolidation and termination, 1 session. Individuals received a CBT workbook (Hope, Heimberg, Juster, & Turk, 2000) and were asked to read the appropriate chapters prior to each therapy session and complete the relevant homework. Participants randomized to the waitlist condition were reassessed after a 16-week waiting period, after which they were offered the same treatment.

Procedure

All participants were recruited through referrals, web listings, or community flyers. After passing a preliminary telephone interview, participants were invited to an initial appointment at the laboratory to complete the ADIS-IV-L interview, a questionnaire assessing demographic information, the LSAS-SR, and the BDI-II. SAD participants completed additional questionnaires online and also completed the BAT appointment, during which they also completed computer tasks not presented in the current analyses, prior to treatment randomization. Participants in the Goldin et al. sample were re-administered the BAT after completing CBT or the waitlist condition (Time 2).

Data Analysis

Anxiety trajectories across the baseline and Time 2 BAT were assessed with separate latent growth curve models (LGCM) in Mplus v.7.11 (Muthén & Muthén, 1998–2013). Parameters were estimated using maximum likelihood parameter estimates with standard errors and a chi-square test statistic that are robust to non-normality and non-independence of observations (i.e., MLR). Model fit was assessed using the recommendations of Hu and

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Bentler (1999) and Hoyle and Panter (1995), such that acceptable fit was indicated by a comparative fit index (CFI) greater than 0.90, root mean square error of approximation (RMSEA) less than 0.10, and a standardized root mean square residual (SRMR) of less than 0.08. Given converging evidence of a tendency for RMSEA values to be inflated in smaller samples (e.g., Curran, Bollen, Chen, Paxton, & Kirby, 2003), we used the SWAIN correction (Boomsma & Herzog, 2013) for all reported RMSEA values. Models were compared using the Satorra-Bentler scaled chi-square difference test (S-B χ^2_d; Satorra & Bentler, 2001), Akaike Information Criterion (AIC; Akaike, 1987), and Bayesian Information Criterion (BIC; Schwarz, 1978). The AIC and BIC are not standardized and not interpreted for a given model but can be compared across models estimated from the same data set; the model with the smaller AIC or BIC is preferred.

For the models for both Aim 1 and Aim 2, an initial linear model was fitted to the data that included latent factors of intercept and slope with the four SUDS measurements from the speech task serving as indicators. Paths from the intercept factor to each indicator were set to one and paths from the slope factor to each indicator were initially set to −22, −2, 0, and 2 (time unit = minute) to estimate a linear growth model. Thus, the intercept reflects the average levels of anxiety at SUDS3 (immediately pre-speech) and the slope represents the linear rate of change in anxiety (from SUDS1-4). For the model corresponding to Aim 1 (SAD versus HC at baseline), SAD diagnostic group (0 = HC, 1 = SAD) was included as a predictor of the intercept and slope factors. For the model corresponding to Aim 2 (effect of CBT versus waitlist at post-treatment), treatment condition (0 = waitlist, 1 = CBT) was included as a predictor of the intercept and slope factors for the Time 2 speech. Based on the fit of the initial linear models, adjustments were made to obtain the model that best approximated the data. As a more stringent test of the effects of treatment on anxiety response to the speech, we also analyzed a second Time 2 model that controlled for Time 1 speech anxiety.

Results

Aim 1: SAD versus HC Differences in Anxiety Trajectories (*N* = 182)

An initial linear growth model would not converge. We then constrained the variances of SUDS1-4 to be equal, which allowed convergence but resulted in poor fit, S-B χ^2 (10) = 90.22; CFI = .869; RMSEA = .209 [90% CI = .170–.250]; SRMR = .053; AIC = 5838.46; BIC = 5864.09. Given that the change from SUDS1-4 was somewhat nonlinear for participants with SAD (see Figure 1), we then tested a quadratic slope. With variances equated for the purpose of model identification, this model demonstrated poor-to-adequate fit, S-B χ^2 (5) = 37.35; CFI = .947; RMSEA = .188 [90% CI = .134–.247]; SRMR = .024; AIC = 5792.33; BIC = 5833.98.

To attempt to improve model fit, we then examined a piecewise model which differentiated between rate of change from SUDS1-3 (baseline to immediately pre-speech; “anticipation phase”) and from SUDS3-4 (immediately pre- to immediately post-speech; “performance phase”). The “performance phase” of the speech included only two measurements, so we did not model this piece as a traditional latent slope given that three repeated measures is

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preferred “for at least a sizable portion of the cases” (p. 125; Curran, Obeidat, & Losardo, 2010). Therefore, to model change in anxiety from pre- to post-speech, we created a separate “performance reactivity” factor, representing the average change from SUDS3 to SUDS4. Factor loadings for the anticipatory slope (i.e., “slope”), were set to −22, −2, 0, and 0, for SUDS1-4, respectively, thus representing the linear rate of change in anxiety from SUDS1-3. This model failed to converge, even when equating variances of SUDS1-4. We then attempted to account for the non-linearity from SUDS1-3 by setting slope factor loadings for SUDS1, SUDS3, and SUDS4 at −1, 0, and 0, respectively, and freely estimating the loading for SUDS2. With residual variances for SUDS1-4 equated for model identification, this model demonstrated adequate fit, S-B χ^2 (4) = 11.92; CFI = .987; RMSEA = .103 [90% CI = .037–.174]; SRMR = .016; AIC = 5766.64; BIC = 5811.50. This model provided superior fit compared to the quadratic model, S-B $\chi^2_d(1)=38.89$, $p < .001$.[2]

Path estimates, means, and residual variances for this LGCM are presented in Figure 2. In the whole sample, mean anxiety increased from baseline to confrontation with the speech and did not change from pre- to post-speech. The effect of SAD diagnostic status on the latent intercept and slope factors was significant, indicating that compared to HCs, individuals with SAD reported higher anxiety during anticipation (i.e., intercept/SUDS3) and showed greater increases in anticipatory anxiety (i.e., steeper slope from SUDS1 to SUDS3) [HC simple slope $B = 8.08$, $z = 5.91$, $p < .001$; SAD simple slope $B = 27.83$, $z = 18.23$, $p < .001$]. The effect of SAD diagnostic status on the performance reactivity factor was not significant, indicating that the groups did not differ in the degree of change in SUDS from immediately pre- to post-speech.[3]

Aim 2: Effect of CBT versus Waitlist on Anxiety Trajectories (*N* = 65)

Model-building for the Time 2 speech progressed in a similar fashion. An initial linear growth model demonstrated poor fit, (variances not equated) S-B χ^2 (7) = 22.90; CFI = .870; RMSEA = .183 [90% CI = .100–.272]; SRMR = .088; AIC = 2064.82; BIC = 2088.73. Given the non-linear change, we then tested a model with a quadratic slope, which would not converge, even with variances equated. We then examined a piecewise model, again distinguishing between SUDS1-3 versus SUDS3-4 by modeling a linear slope from SUDS1-3 with factor loadings of −22, −2, 0, 0 for SUDS1-4, respectively. We again modeled a separate performance reactivity factor that represented the average change from SUDS3 (immediately pre-speech) to SUDS4 (immediately post-speech). With SUDS1-4 variances constrained for model identification, this model demonstrated poor-to-adequate fit, S-B χ^2 (5) = 9.94; CFI = .960; RMSEA = .119 [90% CI = .000–.233]; SRMR = .026; AIC = 2056.00; BIC = 2084.27.

[2]This conditional model also provided better fit than the same unconditional model (i.e., excluding SAD diagnosis as a predictor of the latent factors), S-B $\chi^2_d(3)=141.12$, $p < .001$. The unconditional model provided adequate fit, S-B χ^2 (3) = 8.90; CFI = .986; RMSEA = .104 [90% CI = .028–.186]; SRMR = .016; AIC = 5894.644; BIC = 5929.89. Residual variances for each of the latent factors of the unconditional model were significant, ps < .001, providing motivation for the addition of SAD diagnosis as a predictor.

[3]This pattern of results remained when participants with SAD with a current comorbid diagnosis of major depressive disorder ($n = 14$) were removed from the analysis [effect of SAD diagnosis on: intercept $\beta = 1.65$, $p < .001$; slope $\beta = 1.29$, $p < .001$; performance reactivity $\beta = .02$, $p = .92$].

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We then tested a second piecewise model that was identical to the best-fitting model for Aim 1. Specifically, we allowed for non-linearity in the slope from SUDS1-3 by setting slope factor loadings for SUDS1, SUDS3, and SUDS4 at −1, 0, and 0, respectively. The SUDS2 factor loading was set to −0.21, which was the loading that was freely estimated when running the baseline model in just the RCT sample. With variances of SUDS1-4 constrained to be equal for purposes of model identification, this model demonstrated excellent fit, S-B χ^2 (5) = 2.65; CFI = 1.000; RMSEA < .001 [90% CI = .000-.118]; SRMR = .016; AIC = 2046.37; BIC = 2074.64. This final piecewise model provided superior fit compared to the prior piecewise model according to the lower AIC and BIC. A chi-square difference test could not be conducted because the models were not nested (i.e., the estimated parameters in the final model are not a fixed/constrained subset of those in the piecewise model with the linear anticipatory slope, or vice versa).[4]

Path estimates, means, and residual variances for the final LGCM for the Time 2 speech are presented in Figure 3. On average, at post-treatment/waitlist, anxiety increased from baseline to confrontation with the speech and did not change from pre- to post-speech. There was no effect of CBT on the latent intercept or slope factors, indicating that CBT was not associated with lower anxiety levels at immediately pre-speech or in less steep increases in anticipatory anxiety compared to waitlist. There was, however, a significant effect of CBT versus waitlist on change in anxiety from pre- to post-speech. Compared to the waitlist condition, which showed no change in anxiety from pre- to post-speech [simple slope B = 0.12, z = 0.05, p = .96], CBT showed a decrease in anxiety from pre- to post-speech [simple slope B = −7.42, z = −3.09, p = .002].

To provide a more stringent test of the effects of CBT on Time 2 speech indices, we ran the same model controlling for the effects of the baseline speech (i.e., baseline slope predicting Time 2 slope, baseline performance reactivity predicting Time 2 performance reactivity). Prior to running this model, we tested whether the CBT and waitlist groups differed on any of the latent factors at the baseline speech by running the final piecewise model for Aim 1 in just the RCT subsample; the groups did not differ on any index (ps > .20). Results of the model of the effects of CBT on Time 2 speech indices controlling for the effects of the baseline speech were highly consistent with the model that did not control for the baseline speech. Overall model fit was poor-to-adequate, S-B χ^2 (27) = 43.37; CFI = .945; RMSEA = .089 [90% CI = .015–.142]; SRMR = .174; AIC = 4133.79; BIC = 4188.15. On average, anxiety increased across the anticipatory phase [M = 25.61, SE = 4.92, p < .001] and there were no group differences in either the latent intercept [β = −0.28, p = .27] or latent slope [β = −0.23, p = .40] factors, indicating no effect of CBT versus waitlist on absolute anxiety level at immediately pre-speech or on the increasing rate of anticipatory anxiety, respectively. On average, anxiety did not change from immediately pre- to post-speech [M = 2.50, SE = 2.47, p = .31]; however, the effect of CBT versus waitlist on change in anxiety

[4]This conditional model also provided marginally better fit than the same unconditional model (i.e., excluding treatment condition as a predictor of the latent factors), S-B $\chi^2_d(3)=7.74$, p = .052. The unconditional model provided excellent fit, S-B χ^2 (3) = 0.21; CFI = 1.000; RMSEA = .000 [90% CI = .000–.000]; SRMR = .006; AIC = 2050.34; BIC = 2074.26. Residual variances for each of the latent factors in the unconditional model were significant, ps < .001, providing motivation for the addition of treatment condition as a predictor.

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from pre- to post-speech was significant [$\beta = -0.68$, $p < .01$]. For the waitlist condition, anxiety did not change from pre- to post-speech [$B = 2.50$, $z = 1.01$, $p = .31$], whereas for CBT, there was a significant decrease in anxiety from pre- to post-speech [$B = -6.53$, $z = -2.66$, $p < .01$].

Discussion

The experience of anxiety is widely understood to be a core feature of SAD, and yet much remains to be learned about the temporal dynamics of anxiety experience during feared social situations. In the present study, consistent with hypotheses, we found that individuals with SAD reported greater levels of anxiety during anticipation and greater increases in anticipatory anxiety than non-anxious healthy individuals. Although anxiety was higher among individuals with SAD than healthy controls at post-speech, the groups did not differ in their degree of change in anxiety from immediately pre- to post-speech, which was not different from zero on the average. For the second aim, CBT was associated with a large effect on change in anxiety from pre- to post-speech compared to the waitlist condition. At post-treatment, anxiety decreased from pre- to post-speech in the CBT group, whereas it did not change in the waitlist group. Compared to the waitlist, CBT did not have an effect on either anxiety level immediately pre-speech or on rates of change in anticipatory anxiety.

Anxiety Reactivity in SAD Compared to Healthy Controls

Our finding of greater levels of anxiety during anticipation among individuals with SAD compared to healthy controls is consistent with previous research (e.g., Beidel et al., 2010). However, this finding has been less consistent in the literature than might be expected. One study showed that only those individuals with the generalized subtype of SAD reported higher anxiety than controls (Levin et al., 1993), and in a second study, only those individuals with comorbid depression reported higher anxiety than controls (Yoon & Joormann, 2012). The current study was not designed to disentangle these issues, although it is relevant to note that all of our participants met diagnostic criteria for the generalized subtype of SAD. Additionally, when the 10.4% of the SAD sample with comorbid major depressive disorder were removed from the analysis, individuals with SAD without major depression continued to report higher anxiety during anticipation and greater increases in anxiety during anticipation than control participants.

The greater increases in anxiety during the anticipation phase among our sample of individuals with SAD compared to controls is also generally consistent with the extant literature (Hofmann et al., 1997; Levin et al., 1993), though previous studies did not differentiate between rates of change in anxiety during the anticipatory and performance phases. Indeed, whereas the rate of increasing anxiety during anticipation was significantly greater among those with SAD in the current study, rates of change in anxiety from pre- to post-speech did not differ across the two groups and was not different from zero on average. Therefore, it appears that much of the anxiety that occurs in response to feared social situations occurs prior to confrontation with the situation, and that, on average, this anxiety neither increases nor decreases from immediately prior to immediately post engagement with the situation. In a treatment context, clients may benefit from being told that anxiety is

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unlikely to increase further once they have begun engaging in a feared situation. This knowledge may help the client to reappraise their ability to tolerate the anxiety and to engage in the situation with adequate social skill.

It should be noted that the present finding of no change in anxiety during the performance phase stands in contrast to the growth curve modeling results of Price and Anderson (2011) who observed anxiety during the performance phase of a speech to increase modestly among individuals with SAD. In their study, performance anxiety was indexed as change in anxiety from immediately pre-speech to a retrospective rating of peak anxiety during the speech that was made immediately post-speech. Therefore, had we assessed anxiety during rather than immediately after the speech, we may have observed an increase in anxiety during performance. Alternatively, it may be that the retrospective rating used in the previous study introduced a recall bias.

Effect of CBT on Anxiety Reactivity in SAD

Research on the effects of treatment on anxiety reactivity during BATs has been quite mixed. In the only other study to date that included a no-treatment control condition, uniform decreases in mean anxiety during a speech task were observed across CBT, ACT, and a waitlist control condition, suggesting decreases in anxiety are the result of mere exposure to the BAT environment (Craske, Niles, et al., 2014). Although our data appear to stand in contrast to these results, direct comparison is difficult given differences in operationalization of anxiety reactivity. Whereas Craske and colleagues indexed speech anxiety with an average anxiety rating that collapsed across the anticipatory and performance phases, our analysis examined three distinct anxiety indices that responded differentially to treatment. CBT did not result in differential levels of anxiety measured immediately pre-speech or in differential rates of change in anticipatory anxiety compared to a waitlist condition, but it did result in significantly greater decreases in anxiety from pre- to post-speech at post-treatment/waitlist. This pattern of findings is quite consistent with the results of Price and Anderson (2011), although there was no control group in that study. Therefore, our results strengthen the conclusion that CBT results in improvements in anxiety from pre- to post-speech above and beyond the effects of the passage of time and repetition of the speech task. Considered together with studies that used a single time point or mean indicators of performance anxiety (Heimberg et al., 1990; Heimberg et al., 1998; Rapee et al., 2009), there is relatively consistent evidence that CBT results in reduced anxiety during or immediately following the performance phase of feared social situations in SAD.

Less consistent support has been found for the effect of treatment on anxiety during the anticipatory period. Our results echo the results of Heimberg et al. (1998) who observed no effect of CBT or phenelzine, compared to a credible control treatment or pill placebo, on mean anticipatory anxiety. Only one study has found that treatment results in reduced anxiety during anticipation. Heimberg et al. (1990) reported that CBT resulted in significantly greater reductions in absolute anticipatory anxiety and in change in anxiety during anticipation compared to a credible control treatment. One noteworthy distinction between this study and the present one is the type of BAT employed. We used a nomothetic speech task whereas Heimberg and colleagues (1990) used personalized BATs, which

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arguably may have been more likely to feature in therapeutic exposures during treatment. Even though public speaking was likely a common and highly feared situation for the participants in our study, it is possible they did not complete exposures related to public speaking during their course of CBT depending on their unique treatment priorities. Nevertheless, it does not appear accurate to conclude that CBT results in reduced anticipatory anxiety during a BAT insomuch as the treatment involves exposures that match the content of the BAT. In the study by Price and Anderson (2011), exposures were primarily speeches, but post-treatment level of anxiety at confrontation did not differ from pre-treatment level of anxiety at confrontation.

Exposures during CBT typically focus on completion of the performance aspect of the situation as opposed to repetition of the anticipatory period. If thorough exposure to the performance phase of situations results in greater safety learning regarding one's ability to "handle" a situation once it has begun, then this would explain our pattern of results. What might enhance treatment response, then, is repeated exposure to the waiting and trepidation of the anticipatory period, with less time devoted to the thorough completion of the performance phase of exposures. Indeed, it is during the anticipatory period that the effects of anticipatory cognitive processing, such as negative self-imagery, self-focused attention, and rumination, take hold and contribute to increased anxiety, negative interpretation biases, and poorer social performance (Brozovich & Heimberg, 2013; Hinrichsen & Clark, 2003; Vassilopoulos, 2005; Wong & Moulds, 2011). What might enhance treatment is cognitive restructuring during the anticipatory period that assists the client in tolerating the escalating physiological sensations of anxiety, increasing awareness of and distancing from ruminative thinking, and transitioning away from negative self-imagery to more adaptive field perspective imagery.

Limitations and Future Directions

Our study had a number of notable strengths, including its inclusion of healthy control and no-treatment control groups, powerful statistical methods, and large baseline sample. However, there are also several limitations. Sample size for analyses of the effects of CBT on speech anxiety was larger than many previous studies but modest for a growth curve modeling approach (Kline, 2010). This may account for the marginal significance of the effect of treatment on change in anxiety during performance when controlling for Time 1 speech anxiety indices. Although modest sample size might have also contributed to difficulties with low power to detect an effect of CBT on absolute anxiety or change in anxiety during anticipation, these effects were relatively small. Sample size also precluded our ability to address the question of whether anxiety trajectories have utility with regard to predicting treatment response, or whether changes in such trajectories predict maintenance of treatment gains. Therefore, future studies are needed to examine prospective relationships between anxiety reactivity indices and symptom and functioning measures, both within and following treatment. We also focused on anxiety trajectories in SAD and it remains to be seen whether similar patterns would be observed across other anxiety and mood disorders, and their comorbidity. Finally, our assessment of change in performance anxiety could have been improved by obtaining repeated ratings of distress *during* the speech, as opposed to just before and after, to provide a more fine-grained assessment of how anxiety changes over

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time. Future studies should continue to make use of growth curve modeling to delineate individual differences in anxiety trajectories across the anticipatory, performance, and even recovery phases of BATs, to further our understanding of how individuals with SAD experience feared social situations before and after treatment.

These limitations notwithstanding, the results of our study help to clarify the role of anxiety in SAD and its treatment. More specifically, our findings support previous research indicating that individuals with SAD experience significantly elevated absolute anxiety during anticipation and greater increases in anxiety during anticipation of a feared social situation compared to non-anxious individuals. Conversely, anxiety did not change from pre- to post-speech either for those with SAD or controls, although following CBT, anxiety decreased from pre- to post-speech. This effect was the only observed effect of CBT compared to a waitlist control condition on speech anxiety indices. The finding highlights that repeated exposure to the performance phase of social situations during exposure-based therapy may indeed result in enhanced safety-learning during performance, and also suggests that CBT may be improved by integrating repeated exposures to the anticipatory period of feared social situations.

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Highlights

- We examined anxiety trajectories during a speech in social anxiety disorder (SAD).
- SAD participants reported higher levels of anticipatory anxiety than controls.
- SAD participants also reported greater increases in anticipatory anxiety.
- Following cognitive behavioral therapy, only anxiety during performance decreased.

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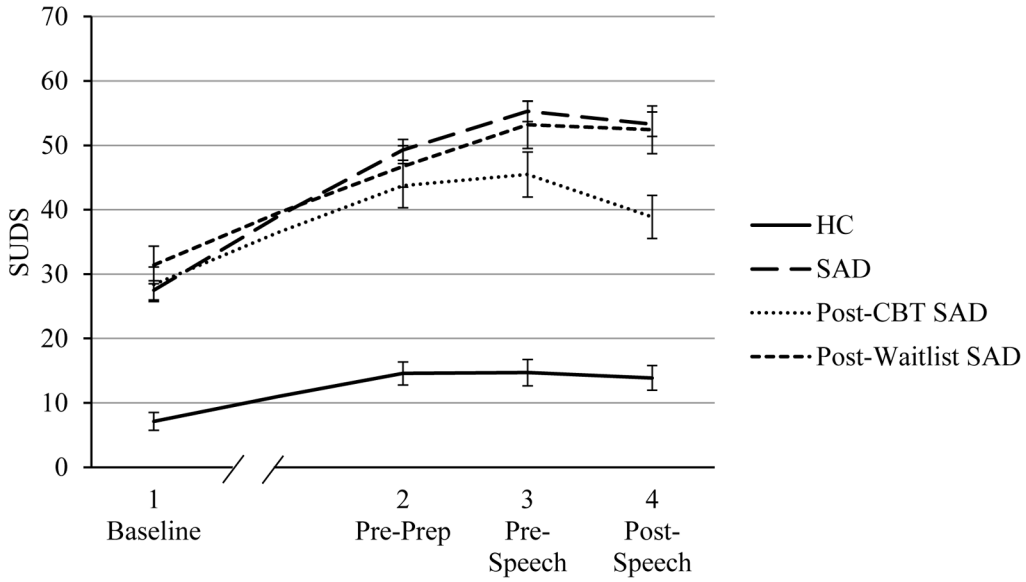

Figure 1.

Mean raw SUDS during the speech task at baseline and at Time 2 (i.e., post-treatment/ waitlist). Error bars are one standard error.

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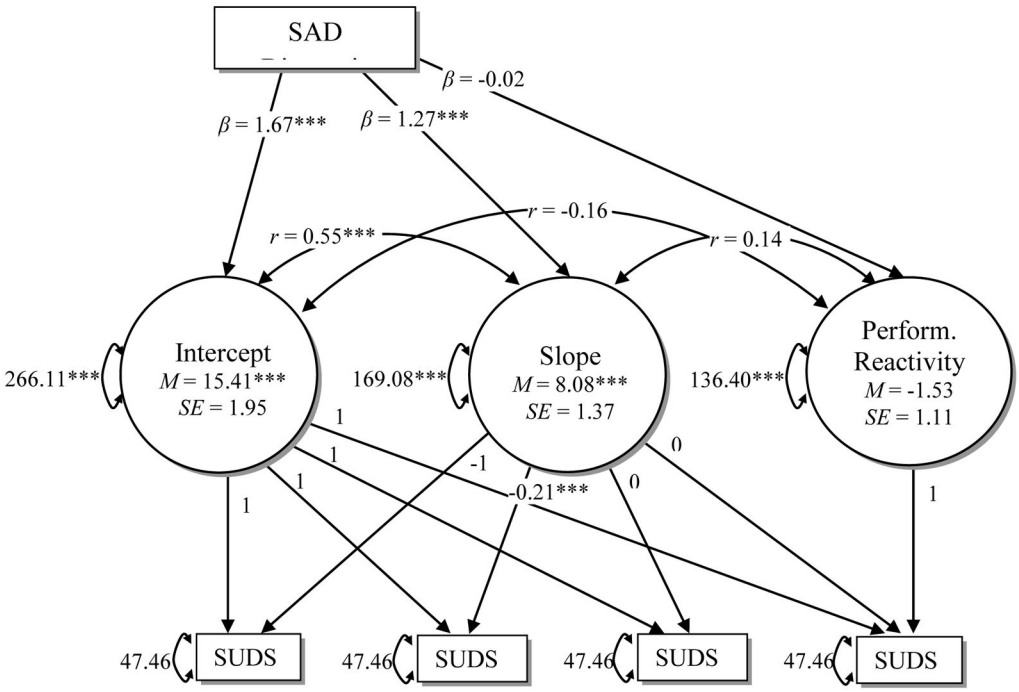

Figure 2.

The best fitting growth curve model for the baseline speech (N = 182). SAD diagnosis is a dichotomous variable (HC = 0, SAD = 1). β = standardized regression weights; r = standardized covariances (i.e., correlations). ***p < .001.

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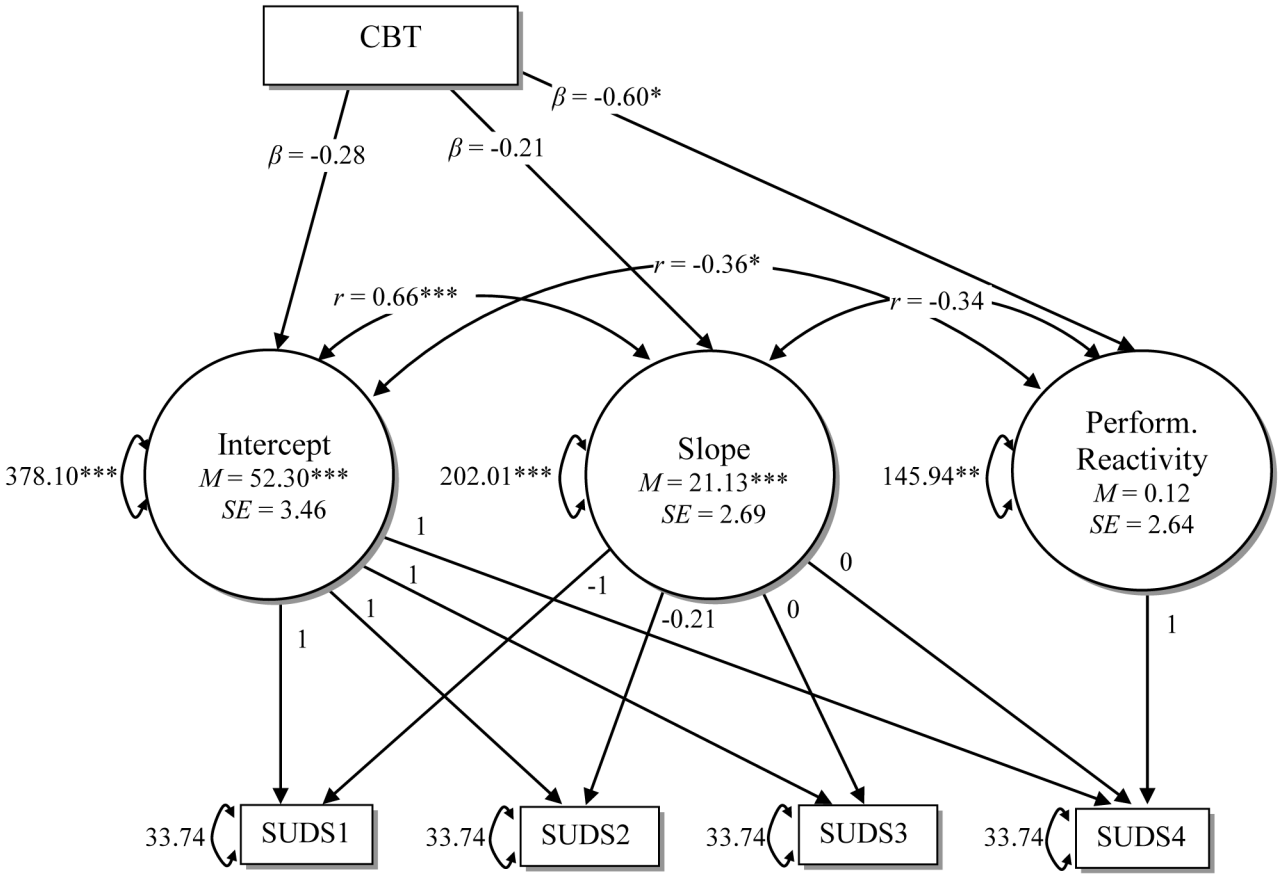

Figure 3.

The best fitting growth curve model for the Time 2 speech (N = 65). CBT is a dichotomous variable (waitlist = 0, CBT = 1). β = standardized regression weights; r = standardized covariances (i.e., correlations). * $p < .05$; ** $p < .01$; ***$p < .001$.

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Table 1

Demographic Information and Mean Self-Report Scores by Group

	SAD (*n* = 135)	HC (*n* = 47)	Test statistic
% Female	50.4	51.1	$\chi^2 = 0.01$
Age (mean years, SD)	33.4 (8.5)	33.5 (9.6)	$F = 0.01$
Education (mean years, SD)	16.7 (2.2)	17.4 (2.0)	$F = 3.32$
% Ethnicity			$\chi^2 = 6.84$
Caucasian	50.8	55.3	
Asian	33.9	34.0	
Hispanic	8.9	0.0	
African American	0.8	0.0	
Native Hawaiian/Pacific Islander	0.8	0.0	
More than One	4.8	10.6	
SUDS 1 (SD)	27.5 (17.3)	7.1 (9.5)	$F = 58.94^{***}$
SUDS 2 (SD)	49.3 (18.8)	14.6 (12.2)	$F = 138.80^{***}$
SUDS 3 (SD)	55.3 (18.1)	14.7 (14.1)	$F = 193.79^{***}$
SUDS 4 (SD)	53.3 (21.6)	13.9 (13.1)	$F = 137.68^{***}$
LSAS (SD)	85.2 (18.7)	15.8 (11.3)	$F = 559.50^{***}$
BDI-II (SD)	13.7 (10.5)	1.7 (2.9)	$F = 57.57^{***}$

Note: SAD = Social Anxiety Disorder; HC = Healthy Controls; SD = standard deviation; SUDS = Subjective Units of Distress Scale; LSAS = Liebowitz Social Anxiety Scale; BDI-II = Beck Depression Inventory, 2nd Edition.

$^{***}p < .001$.