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# INVESTIGATING AND TARGETING POTENTIAL MECHANISMS OF OUTCOME IN EXPOSURE THERAPY FOR SOCIAL ANXIETY DISORDER.

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# INVESTIGATING AND TARGETING POTENTIAL MECHANISMS OF OUTCOME IN EXPOSURE THERAPY FOR SOCIAL ANXIETY DISORDER

**BY**

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# DEDICATION

This dissertation is dedicated to my husband, Norbert, for his endless support and encouragement.

# Investigating and Targeting Potential Mechanisms of Outcome in Exposure Therapy for Social Anxiety Disorder

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Though exposure-based treatments for social anxiety disorder (SAD) are efficacious, not everyone benefits, and we still do not fully understand how and under what conditions this treatment works. Fruitful approaches to better understanding and improving exposure therapy 1) identify predictors of treatment outcome based on theory and 2) manipulate proposed predictors to improve outcomes. Theoretical models of exposure therapy implicate reduction in threat-related avoidance (or, increases in threat-related attention and approach) as a mechanism of fear reduction. The studies in this dissertation follow this theory-driven approach by testing the hypothesized relation between changes in implicit measures of threat-related attention and treatment outcome (Studies 1 and 2) and testing the efficacy of a strategy aimed to manipulate threat approach to improve exposure outcomes (Study 3). Specifically, in Study 1 we examined how social threat-related attentional biases change during exposure therapy for SAD, and attempted to relate these changes in attention bias with symptom change. Additionally, we tested a computational method used to yield attention bias scores that may be more reliable for

repeated measurement. Our results indicated that while social threat-related attention bias did decrease over the course of exposure therapy, these changes were not predictive of symptom change. Study 2 examines the language used during speech exposures (i.e., linguistic style) as a potential predictor of exposure outcome, relating these variables to SAD symptom severity a week after a single exposure session. We found that increased use of first-person plural pronouns during the public speaking exposure was predictive of superior exposure outcomes, in line with an hypothesis that first-person plural pronoun is indicative of threat-related attentional focus during exposure therapy. Finally, Study 3 tested whether a behavioral manipulation thought to temporarily increase endogenous testosterone (a hormone associated with social threat approach) would augment exposure therapy. We found no evidence to suggest that testosterone levels can be manipulated via our behavioral strategy and were thus unable to test whether increasing testosterone facilitates exposure therapy outcomes. We discuss some limitations to the research, as well as recommendations for future directions.

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# INTRODUCTION

Social anxiety disorder (SAD) is characterized by intense, persistent fear of negative evaluation in social situations, due to the belief that negative evaluation will result in humiliation and rejection (Clark & Wells, 1995). These fears often culminate in marked distress in social situations and avoidance of situations in which there is a perceived social threat (American Psychiatric Association, 2013). Due to this, SAD is associated with numerous impairments, including increased financial dependency (Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992), a higher unemployment rate (Wittchen & Beloch, 1996), and increased utilization of medical services (Schneier et al., 1992), in addition to overall reductions in quality of life (Eng, Coles, Heimberg, & Safren, 2005; Lochner, Mogotsi, du Toit, Kaminer, Niehaus, & Stein, 2003; Mendlowicz & Stein, 2000; Meyer, Rumpf, Hapke, & John, 2004; Schneier et al., 1992). SAD is one of the most prevalent mental health disorders, with an estimated 13% of American adults meeting criteria for a lifetime diagnosis (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012), as well as a highly comorbid disorder, with a recent study demonstrating that only 12.2% of their sample of individuals with SAD did not meet criteria for any other mental health disorders (Meyer et al., 2004; Kessler et al., 2012). Accordingly, treating SAD effectively is a public health priority.

## Exposure Therapy for SAD

Exposure therapy is a promising strategy for SAD, with one meta-analysis estimating a medium effect size (Hedges's $g = 0.62$) for randomized placebo-controlled trials of exposure-based therapy for SAD (Hofmann & Smits, 2008). Exposure therapy

involves gradual, repeated exposure to feared objects or contexts in the absence of their feared outcome (Abramowitz, Deacon, & Whiteside, 2012). The treatment of SAD, wherein the core fear is negative evaluation (and individuals tend to overestimate both the likelihood and the cost of encountering negative evaluation; Clark & Wells, 1995), involves gradual confrontation of situations that elicit this fear (e.g., public speaking).

Despite being an efficacious treatment for many, not everyone who receives exposure therapy for SAD evidences improvement, and among those who do, not all maintain those gains long-term (Blanco et al., 2010; Davidson et al., 2004). For example, in one trial of exposure-based treatment for SAD, only 5 of the 20 participants (25%) receiving exposure-based therapy met criteria for remission at the end of the trial (Otto, Pollack, Gould, Worthington, McArdle, & Rosenbaum, 2000). Clearly, there is room for improvement in exposure therapy for SAD.

Given that exposure therapy is effective, approaches to improving anxiety disorder treatment outcomes have largely focused on treatment combination strategies (rather than disregarding exposure); for example, combining anxiolytics or antidepressants alongside exposure in hopes that combining two effective strategies will yield a synergistic effect. Unfortunately, combining these types of pharmacological strategies has not been shown to be advantageous (Davidson et al., 2004; Otto, Smits, & Reese, 2005). However, newer combination approaches to improving exposure-based therapy for anxiety disorders have focused on identifying parameters critical to fear extinction, the mechanisms thought to underlie exposure efficacy, and developing novel augmentation strategies (both pharmacological and behavioral) that can manipulate these parameters. Accordingly,

research on exposure therapy mechanisms is vital to not only developing a thorough theoretical understanding, but also toward the development of advantageous clinical applications.

## Mechanisms of Exposure Therapy

Exposure therapy is based on fear extinction principles wherein a classically conditioned stimulus (i.e., a previously neutral stimulus which begins to elicit a conditioned response after it is paired with an unconditioned, aversive outcome) is repeatedly presented in the absence of the unconditioned aversive outcome until conditioned responding ceases (Myers & Davis, 2007). Foa & Kozak (1986) hypothesize that the reduction in the fear response seen in exposure therapy occurs via repeated activation of a "fear structure", or a set of information, memories or beliefs related to the feared stimulus. According to this theory, the fear structure must be activated in order for the individual to obtain and integrate corrective information about the relative safety of the feared stimulus. Accordingly, the primary goals of exposure therapy involve having individuals attend to (or approach) stimuli that they perceive as threatening in order to activate the fear structure and obtain corrective learning. In exposure therapy, fear structure activation is indexed by an increase in physiological and self-reported levels of fear. During exposure sessions, individuals are repeatedly asked about their subjective fear levels using a 100-point Likert scale fear rating called the Subjective Units of Distress Scale (SUDS; Wolpe, 1958). Therapists typically view successful exposure sessions as those in which there is an increase in SUDS (or fear activation), which decreases over time (either within the session or between sessions). Though between-session declines in fear are predictive of symptom change, there is a lack

of compelling evidence that the changes in fear within a single exposure session can be used to reliably predict therapeutic outcome (Craske, Kircanski, Zelikowsky, Mystkowski, Chowdhury, & Baker, 2008). Accordingly, recent approaches to understanding the mechanisms of exposure therapy have additionally focused on an inhibitory learning based approach rather solely relying on changes in fear levels (Craske et al., 2008).

Inhibitory learning is based on fear extinction research demonstrating that the original fear memory (i.e., the association between the neutral and aversive stimuli) is not eliminated or erased with extinction training, but rather a new, non-threat association forms that competes with the original association (Bouton, 1993). Inhibitory learning occurs as this new association develops and gains strength. Since concurrent anxiety levels do not necessarily reflect this learning, this could explain why within-session fear levels may not accurately predict symptom improvement (Craske et al., 2008). As we learn more about the precise mechanisms underlying exposure therapy, we can develop procedures and strategies to improve outcomes (e.g., by focusing on optimizing inhibitory learning rather than fear reduction).

## Improving Exposure Therapy Outcomes

### Consolidation Enhancement Approaches

As mentioned previously, there is now evidence that fear extinction, rather than involving an "unlearning" of an acquired fear, involves new safety learning which inhibits previous fear associations (Bouton, 1993; Myers, Ressler, & Davis, 2006). Accordingly, D-cycloserine (DCS), which had been shown to facilitate fear extinction retention in animal research by enhancing N-methyl-D-aspartate (NMDA) receptor

function (Laube, Kuhse, & Betz, 1998), was identified as a strategy to increase the consolidation of new "safety" learning with exposure therapy. In other words, DCS has been applied as a "cognitive enhancement" strategy for increasing retention of learning during exposure. The evaluation of this putative augmentation strategy has shown some success (Hofmann, Otto, Pollack, & Smits, 2015), with research demonstrating that DCS may improve CBT outcome for individuals with SAD (Smits et al., 2013a). Several other types of cognitive enhancement agents are currently being tested (for review, see Hofmann et al., 2011). Another example of a possible successful augmentation agent derived from the knowledge on fear extinction is yohimbine hydrochloride, a selective α2-adrenergic receptor antagonist, which increases norepinephrine in regions of the brain implicated in fear and fear extinction (Holmes & Quirk, 2010) and facilitates fear extinction in mice (Cain, Blouin, & Barad, 2004). Indeed, yohimbine has been found to successfully augment exposure therapy for SAD (Smits et al., 2014). Together, this corpus of research demonstrates that fear extinction is theoretically a fruitful model for exposure therapy, and thus can guide the development augmentation strategies for exposure therapy that target fear extinction consolidation.

**Acquisition Enhancement Approaches**

Because follow-up research appears to indicate that agents which target the enhancement of fear extinction consolidation may only be effective when applied to successful sessions (i.e., sessions in which there is a low end fear rating (Smits et al., 2013a; Smits et al., 2013b; Smits et al., 2014), it may be necessary to focus on the mechanisms underlying fear extinction learning (i.e., acquisition) instead of retention

alone. Clearly, consolidating information learned in an exposure session may not be useful if successful extinction learning has not taken place.

Behavioral strategies have already been developed and implemented into exposure therapy procedures that build on this idea of enhancing fear extinction acquisition. Given that theory (Foa & Kozak, 1986) and empirical research (Powers, Smits, Leyro, & Otto, 2007) underscore the importance of attention to and processing of (perceived) threat-relevant information during acquisition to exposure therapy outcome, exposure therapy has been optimized by the addition of strategies that enhance inhibitory learning by reducing or eliminating avoidance behaviors. These "safety behaviors" or "safety signals" may interfere with the development of inhibitory associations during exposure (Hermans, Craske, Mineka, & Lovibond, 2006). For example, an individual with SAD who avoids making eye contact during exposure sessions does not gather the information necessary to violate their expectancies about the audience's reaction, and thus will not develop new learning regarding safety. Indeed, several empirical studies have supported this theory by demonstrating that distraction (or avoidance) reduces exposure efficacy (Kamphuis & Telch, 2000; Sloan & Telch, 2002; Taylor & Alden, 2010). Interestingly, two of these studies (Kamphuis & Telch, 2000; Sloan & Telch, 2002) tested the differential effects of exposure with guided threat appraisal (where participants are specifically directed to focus on the perceived threat), exposure with distraction or safety behavior utilization, and exposure without guided threat appraisal. In both studies, individuals in the distraction group evidenced significantly more fear at post-treatment and follow-up, and exposure therapy outcomes were facilitated by guided threat reappraisal. The Taylor and Alden

(2010) study focused specifically on individuals with SAD, instructing individuals prior to exposures with either a general exposure rationale beforehand, or a safety behavior reduction rationale, describing how the participants should try to refrain from engaging in strategies that aim to prevent feared outcomes. Less utilization of less safety behaviors mediated a change in participant self-judgments (i.e., participants were less negative and more accurate in judgments in their performance) and future social predictions (i.e., participants had lower judgments about the likelihood of negative outcomes in future social events). Similarly, research studies also suggest that utilizing strategies to reduce the perceived threat of exposure tend to lead to less reduction in fear (Powers, Smits, & Telch, 2004; Rodriguez & Craske, 1993). From these studies, it appears evident that enhancing threat reappraisal and reducing avoidance leads to improved treatment outcomes. Given some findings that suggest that judicious use of safety behaviors may not interfere with exposure therapy outcomes (Deacon, Sy, Lickel, & Nelson, 2010; Milosevic & Radomsky, 2008; Rachman, Shafran, Radomsky, & Zysk, 2011; Sy, Dixon, Lickel, Nelson, & Deacon, 2011), Craske and colleagues (2014) theorize that avoidance may only be innocuous if it does not decrease the individual's expectation of feared outcomes within a given exposure session, suggesting that there needs to be a balance of inhibition (i.e., safety learning) and excitation. This is in line with Foa and Kozak's (1986) emotional processing theory. In sum, there is widespread consensus that avoidance should be eliminated during exposure therapy (Hermans et al., 2006).

## Targeting Avoidance in Exposure Therapy

One difficulty that arises while researching the potential effects of avoidance on exposure therapy outcomes is the sometimes covert nature of avoidance. Despite facing their fears by engaging in an exposure exercise, an individual may utilize attentional or behavioral strategies that undermine exposure therapy outcomes, which are difficult to assess. For example, an individual with SAD may use mental distraction during public speaking exposures or restrict their speech to non-emotional content to avoid their feared outcome. Rather than relying on the individual to report on their use of these strategies (which may even be outside their level of conscious awareness), it may be beneficial to instead focus on their use of strategies that are inconsistent with avoidance, such as increased attention or approach toward a threatening stimulus. This is also in line with the aforementioned theories suggesting that it is necessary to focus on both inhibitory and excitatory processes (Craske et al., 2014; Foa & Kozak, 1986).

### Attention in Exposure Therapy

It is important to understand the hypothesized role of attention dysregulation to the etiology and maintenance of SAD in order to understand how threat-related attention might play out within an exposure session. Cognitive theory suggests that individuals with SAD demonstrate biased attention, manifested as hypervigilance toward and/or avoidance of perceived social threatening stimuli, which is detrimental to their interpretations of situations (for review, see Morrison & Heimberg, 2013). Research supports this theory, with performance on computerized attention bias assessment tasks generally differentiating socially anxious and non-anxious populations (for review, see

van Bockstaele et al., 2014). However, we know relatively little about how performance on these tasks might predict threat-related attention during an exposure therapy session, or, alternatively, how exposure therapy might in turn impact attentional biases. Though attention bias tasks (for review, see van Bockstaele et al., 2014) and eye tracking technology (Horley, Williams, Gonsalvez, & Gordon, 2009; Wieser, Pauli, Alpers, & Mühlberger, 2009) have been used to study attention toward various computerized stimuli, we lack well-validated implicit tools for gauging attentional focus during exposure therapy for SAD (though eye tracking and virtual reality technology may be promising avenues toward this end; for review see Lindner et al., 2017).

**Approach in Exposure Therapy**

Though attentional control is one form of approach or avoidance in exposure therapy (and initial attention is necessary for either action), approach-avoidance behavior in response to threat is a broader construct describing preparation for action organized by underlying motivational systems (Roelofs, Elzinga, & Rotteveel, 2005). Research suggests that approach and avoidance behaviors in SAD are mediated (in part) by the hypothalamus-pituitary-adrenal (HPA) axis (Roelofs, van Peer, Berretty, de Jong, Spinhoven, & Elzinga, 2009), a neuroendocrine system. Accordingly, social approach motivation appears to be governed not only by neural activity, but is influenced by the endocrine system.

As previously described, extensive research has shown the potentially deleterious effects of avoidance on fear extinction. Alternatively, there is evidence that having individuals engage in "antagonistic actions", or actions that are inconsistent with fear and

avoidance (e.g., having a client with a fear of heights run towards a railing in a high place), may increase exposure outcomes (Wolitzky & Telch, 2009). Further, increased activity in brain areas associated with threat approach has been shown to predict increased exposure efficacy (McClure et al., 2007; Olatunji et al., 2014; Doehrmann et al, 2013). Accordingly, increased approach toward or engagement with fear-related stimuli may bolster fear extinction.

## Overview of Dissertation Studies

Recent approaches to improving exposure therapy for SAD have focused on 1) identifying predictors of exposure therapy outcome based on mechanisms of fear extinction, and then 2) attempting to manipulate these mechanisms to improve exposure therapy outcomes. Elimination of threat avoidance, or facilitated attention/approach toward threat, has emerged repeatedly as a reliable predictor of exposure therapy efficacy and, when targeted in exposure therapy, appears to facilitate exposure outcomes. Accordingly, the three studies comprising this dissertation have followed this focused on 1) measuring attention as a predictor of exposure therapy outcome using implicit measures, and 2) attempting to manipulate a hormone related to threat-related attention and approach in exposure therapy for SAD.

### Study 1: Do changes in attention bias predict changes in symptoms in exposure therapy for SAD?

The first study in this dissertation aimed to inform whether exposure-based treatment for SAD resulted in decreased attention bias and, if so, whether those changes were related to symptom change (Davis et al., 2016). Given that attention toward threat is theoretically necessary

for a successful exposure, and that individuals diagnosed with SAD appear to have bias threat-related attention, it is possible that one of the mechanisms of exposure therapy is via a reduction in threat-related attention bias. Accordingly, we measured attention bias with a spatial cueing task during and following exposure-based treatment for 39 individuals with SAD, testing whether changes in attention bias related to SAD symptom reduction. Additionally, we utilized a novel computational method (trial level bias scoring; TLBS) in an attempt to address the reliability issues noted with the traditional attention bias scoring approach.

**Study 2: Do linguistic measures of attention predict changes in symptoms during exposure therapy for SAD?**

Given noted weaknesses with computerized attention bias assessment tasks, in particular that such tasks may not be sensitive enough to discriminate individuals with differing levels of social anxiety symptom severity (for review, see Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoorn, 2007), the second study (Davis, Baird, Witcraft, Pennebaker, & Smits, in preparation) aimed to analyze language use, which has been hypothesized to measure attentional focus, during a public speaking exposure. Specific parameters of language, particularly first-person singular and plural pronoun use, have been used to successfully predict the subject and processing of attention (Gunsch, Brownlow, Haynes, & Mabe, 2000; Kowalski, 2000; Pasupathi, Mansour, & Brubaker, 2007; Simmons, Gordon, & Chambless, 2005). First-person plural pronoun use has been linked with attention towards others (versus the self) and engagement (Cegala, 1989; Pennebaker, Mehl, & Niederhoffer, 2003), as well as adaptive coping (Agnew, Van Lange, Rusbult, & Langston, 1998; Chung & Pennebaker, 2007; Rohrbaugh, Shoham, Skoyken, Jensen, & Mehl, 2012). Accordingly, we analyzed the language

used during public speaking exposures for SAD and tested first-person pronoun use (singular or plural) as a predictor of SAD symptom severity a week after exposure.

**Study 3: Can we manipulate endogenous testosterone behaviorally to improve treatment outcome in exposure therapy for SAD?**

Finally, the third study (Davis, Rosenfield, Roelofs, Kolb, Powers, & Smits, under review) attempted to target endogenous testosterone levels prior to exposure therapy, as testosterone is related to social threat-related approach behaviors (Eisenegger, Haushofer, & Fehr, 2011; Maner, Miller, Schmidt, & Eckel, 2008; Mazur & Booth, 1998; Mehta & Josephs, 2010; van Honk et al., 1999). We attempted to manipulate endogenous testosterone levels by having participants (N=73) hold postures associated with dominance (i.e., power pose) immediately prior to public speaking exposures. Power posing is a form of embodied cognition, which is based on the theory that behavioral movement can impact perception. Power posing has been linked to both increased subjective feelings of power, as well as brief increases in testosterone (Carney, Cuddy, & Yap, 2010). We also tested whether, if power posing results in increases in testosterone levels, individuals in the power posing condition (compared to those in a submissive posing or rest condition) evidenced superior exposure therapy outcomes.

If hypotheses are supported, the findings from this dissertation would 1) justify further studying attention and approach as predictors of exposure therapy outcomes, perhaps using implicit measures such as computerized tasks and linguistic analysis, 2) demonstrate that testosterone levels can be behaviorally manipulated, and 3) demonstrate that increased testosterone facilitates exposure therapy outcomes, highlighting social approach motivation as a useful target for augmentation strategies.

## STUDY 1: ATTENTION BIAS DYNAMICS AND SYMPTOM SEVERITY DURING AND FOLLOWING CBT FOR SOCIAL ANXIETY DISORDER[1]

Cognitive-behavioral theories implicate attention bias in the etiology and maintenance of social anxiety disorder (Clark & Wells, 1995; Hirsch & Clark, 2004; Morrison & Heimberg, 2013; Rapee & Heimberg, 1997). Attention bias has been conceptualized as dysregulation in attentional processing of emotional stimuli (Cisler, Bacon, & Williams, 2009; Mogg, Bradley, Williams, & Mathews, 1993), characterized by facilitated attention toward and/or difficulty disengaging attention from threatening cues (Amir, Elias, Klumpp, & Przeworski, 2003; Koster, Crombez, Verschuere, & Houwer, 2006; Mogg & Bradley, 1998). Attention bias is thereby thought to drive a variety of maladaptive processes implicated in social anxiety disorder (e.g., misinterpretation of threat; Clark & Wells, 1995; Heinrichs & Hofmann, 2001; Morrison & Heimberg, 2013).

There is a growing body of research focused on the role of attention bias in social anxiety disorder (for review, see Heeren, Reese, McNally, & Philippot, 2012; van Bockstaele et al., 2014). Cross-sectional research has shown some support for the hypothesized association between attention bias and social anxiety disorder. Specifically, performance on tasks adapted to measure attention bias (e.g., the Stroop, dot-probe, and spatial cueing paradigms) has, with some mixed evidence, generally tended to discriminate between socially anxious and non-anxious individuals. Yet, relatively little research has focused on how attention bias relates to social anxiety disorder symptoms over the course

1. Davis, et al (2016)

of cognitive behavior therapy (CBT), which aims to directly target factors related to attention bias (e.g., cognitive appraisal, avoidance; Clark & Wells, 1995; Hofmann, 2007). Pishyar and colleages (2008) used a composite measure of both attentional avoidance and hypervigilance and found that, relative to those assigned to a waitlist control condition, individuals receiving CBT evidenced reductions in both social anxiety symptoms and attention toward threat. Barry et al. (2015) demonstrated that difficulty with disengagement from threatening cues, but not facilitated engagement toward threatening cues, predicted CBT response in a mixed anxiety disorder sample that included individuals suffering from social anxiety disorder.

Building upon extant research, we subjected adults with social anxiety disorder to a repeated assessment schedule of attention bias and social anxiety disorder symptom severity during and following a brief exposure-based CBT program. In addition to examining the change in attention bias that occurs with CBT, we modeled the data such that we could make inferences with respect to directionality and causality – i.e., determine whether attention bias changes during CBT precede and lead to social anxiety reduction, whether social anxiety reductions during CBT precede and lead to reduction in attention bias, or both.

We also capitalized on emerging theory and findings positing that dysregulation in attentional processing of threat in social anxiety disorder may be better reflected by a dynamic process over time instead of static perspective on attention bias (Zvielli, Bernstein, & Koster, 2015a). Specifically, Zvielli et al. (2015a) proposed that attention bias is a dynamic process expressed in fluctuating, phasic bursts toward and away from

motivationally relevant stimuli over time. Accordingly, they introduced a novel computational procedure, Trial-Level Bias Scores (TLBS), that yields a series of repeated estimations of attention bias, toward and/or away, from trial-to-trial over time, per individual – rather than only a single aggregated mean static estimate of attention bias that collapses across time. Traditionally, bias is inferred from aggregated mean/median differences in reaction time (RT) between trial types in which emotional stimuli may interfere with (slow) *or* enhance (speed) attentional processing. Zvielli et al. (2015a) found that key features of the temporal dynamics of attention bias (e.g., mean and temporal variability in attention bias toward and away from motivationally-relevant stimuli) demonstrated higher split-half reliability as well as incremental predictive validity above and beyond conventional aggregated mean bias scores in discriminating between phobics and healthy controls. More recently, Yuval et al. (in press) found that the temporal dynamics of attentional bias (towards, away, and variability) to trauma cues predicted levels of posttraumatic stress symptom severity in refugees at elevated risk for trauma-related mental health problems; and that temporal variability in bias as well as attentional bias away but not towards trauma cues predicted behavioral avoidance of exposure to trauma stimuli; no effects were observed when bias was quantified traditionally. Schäffer et al (2016) found that, among German soldiers, bias dynamics to emotional information, at pre- and post-deployment, predicted higher levels of posttraumatic stress symptomatology after deployment as a function of number of traumatic experiences; conventional mean bias scores did not similarly prospectively predict posttraumatic stress at post-deployment. In anxious adults, Amir and colleagues (in press) found that not only

were features of covert and overt bias dynamics correlated, but that the real-time, dynamic expressions of overt and covert attentional processes were significantly coupled from trial-to-trial; again, conventional covert and overt bias scores were not associated. In related work, attention bias variability was examined with respect to PTSD (Iacoviello et al., 2014; Naim et al., 2015), providing further evidence for the utility of a dynamic process perspective on attention bias in psychopathology. Accordingly, in so far as this conceptual and computational approach better represents the nature of attentional dysregulation in the processing of threat theorized to be important in the maintenance of social anxiety disorder, this perspective may be key to help to elucidate the role(s) of attention bias in therapeutic change over the course of CBT for social anxiety. This study will be the first to model attention bias as a dynamic process measured repeatedly over the course of CBT, allowing us to examine temporal relations between dysregulation in attentional processing of threat and symptom change.

We predicted that modeling attention bias as a dynamic process, rather than using the conventional computation, would yield more reliable indices of attention bias. This is important for modeling attention bias measured repeatedly at multiple points in time over the course of therapy. Next, we predicted that attention bias toward threat (i.e., hypervigilance), attention bias away from threat (i.e., avoidance), and temporal variability in attention bias (i.e., attention dysregulation) would each decrease over time. Furthermore, we expected that greater regulation of attentional processing of threat (reduced attention bias dynamics) would lead to reduced social anxiety symptom severity, *and* that reduced symptom severity would lead to greater attentional regulation. Thus, we predicted that

(dys)regulated attentional processing of threat and social anxiety symptom severity would be reciprocally related. Finally, we examined whether these bias-anxiety relations would vary as a function of time (i.e., testing the stationarity assumption, or the often incorrect assumption that relations do not vary over the course of treatment).

## Method

### Design

The parent clinical trial, approved by the Institutional Review Board at Southern Methodist University, for this secondary analysis involved the random assignment of 40 adults with social anxiety disorder to a 5-session CBT protocol augmented with either Yohimbine hydrochloride, an alpha2-adrenergic receptor agonist, or pill placebo administered acutely 1-hour prior to sessions 2-5 (Smits et al., 2014). In this study, social anxiety symptoms declined significantly for participants in both conditions, with individuals receiving yohimbine-augmented CBT evidencing a faster rate of symptom decline than those receiving placebo-augmented CBT. Symptom severity and attention bias were assessed at baseline, before each weekly session, and at both one week and one month post-treatment. Prior to the session onset, symptom severity was assessed and was immediately followed by the attention bias task (and pill administration at weeks 2-5). Because we aimed to examine the relation between attention bias and symptom severity during and following CBT, we only used data from sessions 1 (immediately before the start of CBT) to 5, post-treatment and follow-up (e.g., 7 total data points).

### Participants

Of the 40 individuals participating in the study, 39 individuals completed attention bias assessment during at least one of the seven assessments (i.e., one participant did not complete any attention bias assessments due to early dropout). Informed consent was obtained from all participants at Southern Methodist University. Participants had a DSM-IV diagnosis of social anxiety disorder, as diagnosed by the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1998) and evidenced no psychiatric and medical comorbidities that interfered with the safety of participating in the study (see Smits et al., 2014 for list of study entry criteria). Sample characteristics (from baseline and the first attention bias assessment task at week 1) are reported in Table 1.

| | Total | Yohimbine | Placebo |
|---|---|---|---|

| Variable | N | Mean | SD | N | Mean | SD | N | Mean | SD |
|---|---|---|---|---|---|---|---|---|---|
| Age | 39 | 37.31 | 13.13 | 20 | 35.50 | 14.02 | 19 | 39.21 | 12.19 |
| LSAS-SR | 39 | 70.67 | 23.78 | 20 | 69.55 | 21.52 | 19 | 71.84 | 26.49 |
| $TLBS_{away}$ | 36 | -72.15 | 39.54 | 19 | -70.59 | 24.85 | 17 | -73.90 | 52.14 |
| $TLBS_{toward}$ | 36 | 67.97 | 31.61 | 19 | 62.14 | 24.98 | 17 | 74.49 | 37.39 |
| $TLBS_{variability}$ | 36 | 90.29 | 41.94 | 19 | 88.85 | 28.78 | 17 | 91.89 | 53.95 |
| $AB_{conventional}$ | 36 | -7.09 | 42.16 | 19 | -4.81 | 42.49 | 17 | -9.64 | 42.95 |
| | N | % | | N | % | | N | % | |
| Male | 25 | 64.1 | | 15 | 75.0 | | 10 | 52.6 | |
| Female | 14 | 35.9 | | 5 | 25.0 | | 9 | 47.4 | |

*Note.* For $TLBS_{away}$, lower (negative) scores reflect increased attention bias away from threat. For $TLBS_{toward}$ and $TLBS_{variability}$, higher (positive) scores reflect increased levels of each of these biases.

**TABLE 1.1** BASELINE CHARACTERISTICS.

**CBT**

Treatment consisted of a 5-week CBT protocol for social anxiety disorder (Hofmann, 2004), which has been employed in previous and ongoing studies examining pharmacological augmentation of CBT for social anxiety disorder (Guastella et al., 2008; Hofmann et al, 2006; Hofmann, Otto, Pollack, & Smits, 2015). The first session consists of psychoeducation about social anxiety disorder and rationale for exposure therapy, while sessions 2-5 consist of public speaking exposure exercises.

**Assessment**

**Liebowitz Social Anxiety Scale – Self-Report (LSAS-SR).** The LSAS-SR (Fresco et al., 2001) is a self-report measure of social anxiety disorder severity commonly used in treatment studies. The self-report version of the LSAS has sound psychometric properties, which are comparable to those of the clinician-rated version (Fresco et al., 2001).

**Attention Bias.** We employed a modified version of the Posner spatial cueing paradigm (Posner, Snyder, & Davidson, 1980), which has been previously used to assess attention bias in social anxiety disorder (Amir et al., 2003; Amir, Weber, Beard, Bomyea, & Taylor, 2008; Julian, Beard, Schmidt, Powers, & Smits, 2012). In this task, a word that is either socially positive (e.g. "delighted"), socially negative/threatening (e.g. "embarrassed"), or neutral (e.g. "dishwasher") is presented on the computer screen. After 600 milliseconds, the word disappears and an asterisk appears in either the same location or the location opposite the word. There are 288 trials, of which 192 are valid (i.e., the word validly predicts the location of the target), 48 are invalid (i.e., the word does not predict the location of the target), and 48 are uncued (i.e., no word is presented at all). The participant must select either the left button on the keyboard if the asterisk appears on the left side of the screen, or the right button on the keyboard if the asterisk appears on the right side of the screen.

*Conventional Approach: Aggregated Mean Bias Score.* The conventional approach to attention bias calculation involves comparing average invalid and valid trial reaction times within threat and neutral trials separately, then comparing performance depending on valence (for more information on this calculation, see Koster, De Raedt, Goeleven,

Franck, & Crombez, 2005). Accordingly, we first calculated a threat index (i.e., subtracted average valid threat RTs from average invalid threat RTs) and a neutral index (i.e., subtracted average valid neutral RTs from average invalid neutral RTs), and then subtracted the neutral index from the threat index to compute an attention bias score.

*Dynamic Process Approach: Trial-Level Bias Score*. As noted, this study is the first to utilize TLBS in the spatial cueing task; accordingly, this computation required some adaptations. The overall number of invalid trials in the task (16%; Amir et al., 2003) precludes the computation of TLBS by matching valid and invalid trials as is done when congruency is fully counterbalanced (Zvielli et al., 2015a). Therefore, we performed the TLBS computation by matching each threat trial with the most proximate neutral trial (e.g., Zvielli, Amir, Goldstein, & Bernstein, 2015b; valid threat trial RT was subtracted from valid neutral trial RT and invalid neutral trial RT was subtracted from invalid threat trial RT). This was done in order to (1) optimize TLBS ability to capture any temporal dynamic in attention bias, regardless if it is linked to hypervigilance or disengagement (i.e., valid/invalid); and (2) to maximize the number of potential matches in time, thus providing maximal temporal resolution of the attention bias estimation (Zvielli et al., 2015a). We then used this TLBS signal-like sequence of RT differences to compute three subject-level variables (i.e., TLBS parameters) that indicate the overall direction and magnitude of bias toward and away, separately, as well as the variability in attention bias toward and away over time. First, $TLBS_{toward}$ is the mean of a participant's positive TLBS scores (i.e., faster RTs in response to valid threat than valid neutral trials or slower RTs in response to invalid threat than invalid neutral trials), indexing level of attention bias toward threat (wherein

higher means reflect greater attention bias toward threat). $TLBS_{away}$ is the mean of negative TLBS scores (i.e., slower RTs in response to valid threat than valid neutral trials or faster RTs in response to invalid threat than invalid neutral trials), indexing level of attention bias away from threat (wherein lower means reflect greater attention bias away from threat). $TLBS_{variability}$ is the temporal stability/variability in the expression of attention bias toward and/or away from threat over time (i.e., sum of the distances between sequential TLBS scores divided by the number of TLBS scores), wherein higher means reflect greater variability in attention bias.

**Data Analysis**

**Data Cleaning.** Following the data cleaning procedures utilized in previous trials (Amir et al., 2008; Julian et al., 2012), response latencies for inaccurate trials (i.e., when the participant pressed the button corresponding to the incorrect location of a probe) were deleted and not used in the analyses. Accordingly, 2.97% of trial data were eliminated due to incorrect responses. Additionally, sessions with an overall accuracy of less than 90% (5.06% of trial data) were removed from the analysis due to potential inattention or random guessing. Also in line with these procedures, response latencies of less than 200 ms or greater than 1,500 ms were removed and not used in the analyses (2.08% of trial data). Outliers were then removed on an individual, session level basis, with outliers defined as reaction times more than three standard deviations outside of the individual's mean reaction time for a particular session (1.29% of trial data).

**Psychometric Properties.** We utilized data from the baseline and week 1 assessment (both before treatment and drug administration) to estimate retest reliability for

the conventional attention bias index and TLBS attention bias indices in the sample. Following procedures delineated by Price et al. (2014), we calculated the Intraclass Correlation Coefficient (ICC) using a 2-way random effects model and the 'absolute' agreement definition. We calculated both single measure ICC scores (i.e., the reliability for a single assessment point per individual) and average measures ICC scores (i.e., the reliability for assessments averaged across individuals; analogous to the internal consistency index, α).

**Hypothesis Testing.** Data were analyzed using multilevel modeling (MLM). We used maximum likelihood estimation and robust standard errors for the variances of the regression coefficients. The repeated assessments of the outcomes, attention bias, and social anxiety symptom severity were nested within subjects.

To investigate the changes in attention bias over time, we modeled time from assessment one (prior to the first CBT session) to the follow-up assessment. In addition, we added a term to allow attention bias at follow-up to be freely estimated in order to reflect potential differences between the treatment (session 1 through post-treatment) and follow-up (post-treatment to one month follow-up) sessions. Initial symptom severity (at baseline, one week pre-treatment) and the interaction of initial symptom severity with time were included as covariates in all analyses. Though not a primary focus of the current study (but rather a consequence of the secondary nature of this analysis), treatment condition was added as a level-two moderator of all the predictors in the models to explore (i.e., no *a priori* hypothesis) whether any of the relationships were different for yohimbine (YOH) vs. placebo (PBO).

In order to test whether changes in symptom severity caused subsequent changes in attentional dysregulation (or vice versa), we employed within-subjects cross-lag panel analyses (see Table 3; for other examples of this type of quasi-causal analysis, see Meuret, Rosenfield, Seidel, Bhaskara, & Hofmann, 2010; Smits, Rosenfield, McDonald, & Telch, 2006; Tschacher & Ramseyer, 2009). In these analyses, LSAS at a time point (t) was entered as a predictor of attentional dysregulation at the next time point (t+1), controlling for attentional dysregulation at the previous time point (and vice versa for attentional dysregulation changes predicting symptom changes).

Given that recent research (Hamaker, Kuiper, & Grasman, 2015) shows that one must disaggregate the effects of time varying predictors (TVPs) to obtain accurate, unbiased estimates of their effects on outcome, we disaggregated each (TVP) into the person's mean across all assessments ($TVP_{mean}$; the between-person component) and their deviation from their mean at each session ($TVP_{dev}$; the within-person component): $TVP_{dev} = TVP_{raw} - TVP_{mean}$. Accordingly, in these analyses, significant $TVP_{mean}$ effects can be interpreted merely as between-subjects covariation between the predictor and the outcome (i.e., people with higher average levels of $TLBS_{variability}$ might have greater symptoms) whereas significant lagged $TVP_{dev}$ effects can be interpreted as reflecting quasi-casual effects of the predictor on the outcome (Hamaker et al., 2015). Given the study aims to identify quasi-causal relations, we limit our reporting to $TVP_{dev}$ effects, although $TVP_{mean}$ effects were included in all analyses (as was necessary to accurately assess the TVP effects; Hamaker et al., 2015). To further strengthen causal inference, we also controlled for the growth curve for each variable in these analyses, as this helps rule out the possibility that

the predictor and outcome are related merely because they are both changing over time (Wang & Maxwell, 2015).

Finally, we repeated the analysis with Time as a moderator to test the stationarity assumption that the relations do not vary over the course of treatment (Maxwell & Cole, 2007; Smits et al., 2012).

Power analyses (PinT 2.12; Snijders & Bosker, 1993) indicated that we had sufficient power (> .80) to detect a medium effect size ($d$=.50) for our least powerful test (those involving treatment condition differences or the mean levels of TVPs). Furthermore, the tests involving the repeated measures over time (those involving the change in the attention bias indices over time, and those involving the cross lag effects between deviations in attentional dysregulation and LSAS) were more powerful and able to detect effect sizes as small as $d$=.36 with power>.80. Effect sizes were calculated using the $t$ to $d$ conversion.

## Results

### Psychometric Properties of Attention Bias Indices

See Table 2 for correlations between attention bias measures and social anxiety symptom severity at week 1. Using scores from a baseline assessment (not shown in Table 2) and week 1 (both pre-treatment), internal consistency and retest reliability indices were significant for each of the three TLBS measures: $TLBS_{toward}$ (ICC-single measure=.44, ICC-average measures=.61, $p$=.005), $TLBS_{away}$ (ICC-single measure=.46, ICC-average measures=.63, $p$=.003), and $TLBS_{variability}$ (ICC-single measure=.53, ICC-average measures=.69, $p$=.001). Internal consistency and retest indices were not significant for the

attention bias index computed using the conventional aggregated mean bias score approach (ICC-single measure=.05, ICC-average measures=.10, $p$=.379). We thus only retained the TLBS indices of attention bias in all subsequent analyses.

Correlations between the attention bias indices and social anxiety symptoms (LSAS) at baseline are displayed in Table 2. None of the attention bias scores were significantly related to LSAS. However, the three TLBS indices were very highly correlated with one another. Higher $TLBS_{toward}$ (i.e., increased bias toward threat) was strongly related to lower $TLBS_{away}$ (i.e., increased bias away from threat; $r$=-.75), and higher $TLBS_{variability}$ (i.e., increased attentional dysregulation) was strongly related to both higher $TLBS_{toward}$ ($r$=.87) and lower $TLBS_{away}$ ($r$=-.96). This is in line with strong symmetry of within-subject variability toward *and* away from threat, such that those exhibiting more bias towards threat also subsequently exhibit more bias away from threat, repeatedly in time.

| | $AB_{conventional}$ | $TLBS_{toward}$ | $TLBS_{away}$ | $TLBS_{variability}$ |
|---|---|---|---|---|
| LSAS | .146 | .042 | -.012 | .019 |

| | | | |
|---|---|---|---|
| $AB_{conventional}$ | -.056 | .624** | -.443** |
| $TLBS_{toward}$ | | -.753** | .873** |
| $TLBS_{away}$ | | | -.960** |

*Note.* **= $p \leq .001$.

**TABLE 1.2** CORRELATIONS AT WEEK 1.

**Change in Attention Bias and Social Anxiety Symptoms Over the Course of CBT**

**Bias Toward Threat: $TLBS_{toward}$.** Consistent with our prediction, there was a significant decrease in $TLBS_{toward}$ over the course of treatment (session 1 to post-treatment), $b$=-1.84, $p$=.011, $d$=.99, and no significant change in $TLBS_{toward}$ over the follow-up period, $b$=4.04, $p$=.309, suggesting that changes were maintained during follow-up period. No treatment condition (yohimbine vs. placebo) effects were observed, suggesting that these changes were seen in participants irrespective of treatment assignment.

**Bias Away from Threat: $TLBS_{away}$.** Consistent with our prediction, $TLBS_{away}$ tended to decrease over the course of treatment, $b$=1.33, $p$=.082, although that decrease failed to reach conventional levels of significance. $TLBS_{away}$ did not change significantly over the follow-up period, $b$=-3.57, $p$=.154. No treatment condition effects were observed.

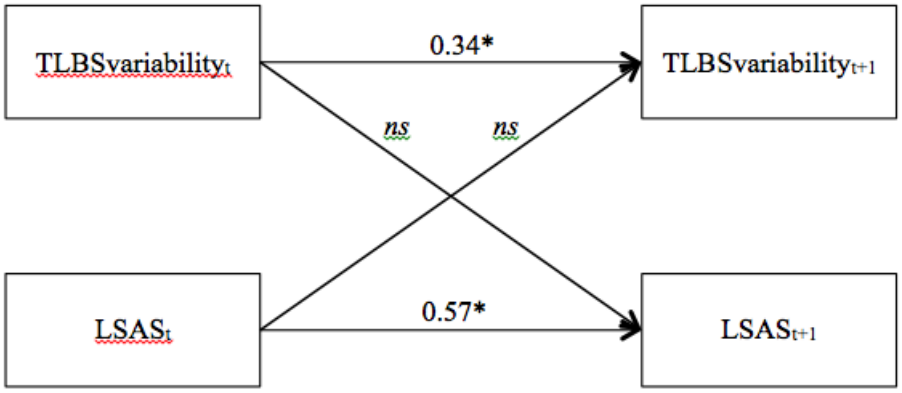
**Temporal Variability in Bias: $TLBS_{variability}$.** Consistent with our prediction, there was a significant decrease in $TLBS_{variability}$ over the course of treatment, $b$=-2.76, $p$=.001, $d$ =1.62, which tended to revert toward baseline levels during follow-up, $b$=7.01, $p$=.061. No treatment condition effects were observed.

As previously reported (Smits et al., 2014), the slope of change in social anxiety symptoms was significant for participants in both treatment conditions, but those assigned to yohimbine-augmented CBT (YOH) demonstrated a significantly faster rate of improvement than those assigned to placebo-augmented CBT (PBO).

**Relation between Attentional dysregulation and Social Anxiety Symptom Severity over Time**

Given 1) the extremely high correlations among the three TLBS parameters or high *within-subject* symmetry between the magnitude of attentional bias towards and away from threat, 2) the fact that the changes in the TLBS measures over the course of the study mimicked those correlations (reductions in for $TLBS_{toward}$ and $TLBS_{variability}$, and a trend toward reductions in $TLBS_{away}$), and 3) the conceptual and mathematical inter-relations between these parameters, we opted to perform cross lag analyses on only the $TLBS_{variability}$ parameter.

As can be seen in Figure 1, autocorrelations for $TLBS_{variability}$, $b$=0.34, $p$<.001, and LSAS, $b$=0.57, $p$<.001, were significant. However, contrary to our predictions, neither the $TLBS_{variability}$→LSAS nor the LSAS→ $TLBS_{variability}$ relations were significant.



Note: *=p≤.001; ns=not significant. LSAS = Liebowitz Social Anxiety Scale; TLBS = trial-level bias score; TVPdev = time-varying predictor, deviation component.

**STUDY 1, FIGURE 1.** CROSS-LAG SLOPES OF TLBSVARIABILITY AND LSAS RELATIONS USING TVPDEV.

**Time as a Moderator of the Relation between Attention Bias and Symptom Severity over Time**

Our previous cross lag analyses examined the relation between attentional dysregulation and social anxiety symptom severity across the full length of the study. Such analyses assume that these relations are stable across the length of the study (stationarity). It is possible that some of these relations may have been diminished because they varied over time (e.g., perhaps the relations were strong at the beginning of the study but weaker at the end of the study, or vice versa). To test for stationarity, we added Time as a moderator of all the $TLBS_{variability}$/LSAS relations in the cross lag panel analysis, including each of the cross lag relations and each of the auto-correlation relations. None of the interactions between time and these attentional dysregulation/LSAS relations were significant, indicating that the relations did not change significantly over time.

## Discussion

The present study is the first to report on the temporal and causal relations between threat-related attention bias and symptom severity during and following CBT for social anxiety disorder. Using an intense repeated assessment schedule over the course of CBT and a novel approach to quantify attention bias as a dynamic process in time (i.e., trial-

level bias score; TLBS), the study yielded a number of findings. First, we found that TLBS parameters reflecting attention dysregulation as measured by the modified spatial cueing task demonstrate greatly improved psychometric properties as compared to aggregated mean bias scores. These results are consistent with earlier work using the modified dot probe task (Zvielli et al., 2015a), suggesting that quantifying features of the temporal dynamics of attention bias provides a psychometrically stronger representation of the underlying phenomenon of attention dysregulation than aggregated mean scores. We did not find significant correlations between any of the attention bias indices and social anxiety symptom severity at baseline. These results comport well with previous research showing that, while attention bias distinguishes people with (social) anxiety disorder(s) from non-anxious individuals, attention bias does not vary significantly among anxious individuals with varying degrees of symptom severity (Bar-Haim et al., 2007).

Second, our results showed that attention bias toward threat, and variability in attending to threat, decreased significantly over the course of treatment, while attention bias away from threat tended to decrease, although this latter tendency did not reach conventional levels of significance. These results are consistent with and extend those reported by Pishyar and colleagues (2008), who found that participants who received CBT, compared to those who did not, demonstrated reduced levels of attention bias toward social threat. Interestingly, Pishyar et al. also did not find that attention bias away from threat changed significantly with CBT for social anxiety. Though our findings similarly failed to support the hypothesis that CBT effectively modifies attention bias away from threat, we did observe evidence of attention bias away from threat prior to CBT in our sample, which

was highly correlated with attention bias toward threat. Accordingly, it appears premature to rule out attention bias away from threat as an important attentional mechanism in social anxiety disorder.

Third, the findings from our cross lag analyses found no support for the hypothesis that improvement (i.e., reductions) in attentional dysregulation leads to symptom severity reduction, or vice versa, suggesting that these two change processes observed in response to CBT may occur independently. In some studies, null associations are best explained by low statistical power. However, in part because of the design (i.e., 8 repeated assessments), power to detect a longitudinal relation between attentional dysregulation and LSAS in our study was actually relatively good, with over .80 power to detect effect sizes as small as $d$=.36. This effect size is equivalent to $\eta^2$=.031. In other words, $d$=.36 is equivalent to accounting for about 3.1% of the variance, an effect size that Cohen (1988) considers within the "small effect size" range. Hence, despite the small sample size, we had sufficient power to detect relationships that were relatively small.

Fourth, by adding time as a moderator in our models, we were able to test the, often false, assumption inherent to most regression models of causation that the causal structure is constant over time (Maxwell & Cole, 2003; Smits et al., 2012). Time did not emerge as a moderator, thus suggesting that relations between symptom reduction and the reduction between attentional dysregulation did not change during the course of CBT or follow-up.

These findings must be considered in the context of a number of limitations. First, although we had sufficient power to detect meaningful effects ($\eta^2$>.031) as per Cohen (1992), we were underpowered to detect smaller effects if they existed. Second, our use of

the spatial cueing task with verbal stimuli (though utilized as an assessment tool in previous work) may preclude direct comparison to literature utilizing the more common dot-probe task, or tasks using facial stimuli or stimuli that may most fundamentally capture feared stimuli in social anxiety (i.e., signs of negative evaluation). Third, we employed a standardized 5-session exposure-based CBT protocol. It is possible that changes in attention bias and their relations with social anxiety disorder severity observed in the current study would not generalize to protocols that employ more sessions or emphasize cognitive or a combination of cognitive and behavioral interventions. Fourth, because our design did not include a control condition for CBT, we cannot infer that observed findings are specific to CBT (as prescribed in the present protocol) or whether they simply speak to how attention bias and symptom severity change and relate to each other over time. Finally, we should note that we observed high correlations among the three TLBS parameters at baseline, which is not unexpected because the calculations are conceptually and mathematically inter-related components of the same process of emotional attention. Due to these very high intercorrelations, we chose to examine only one of the TLBS measures as it related to LSAS to avoid duplicate analyses. Given our small sample size, it was inadvisable to perform any traditional methods of combining data, such as latent variable SEM analysis or factor analysis, both of which require hundreds of subjects to establish reliable factors.

Despite these limitations, our findings provide new insight into the relation between a putative maintaining factor of social anxiety disorder and symptom severity over time. Notably, we observed no evidence suggesting that reduction in attentional dysregulation

serves a key mediator of CBT efficacy. Though our analyses do not allow us to conclude that attention bias is not an important factor in either the maintenance and/or treatment of social anxiety disorder, our null findings leave open this possibility. The question remains whether, if specifically targeted/manipulated during CBT (e.g., via modification methods targeting attentional bias), attention bias might then serve as a mediator. Indeed, multiple candidate mediating processes may drive social anxiety, and change in some but not necessarily all such mediating processes may equifinally lead to reduced social anxiety symptoms.

Future work may also examine whether attentional dysregulation may mediate CBT outcomes in other types of anxiety disorders. Perhaps most importantly, our study adds to the growing body of evidence suggesting a dynamic process perspective, and quantification of key features of attention bias temporal dynamics via the TLBS approach may improve measurement of attention bias broadly, the capacity to model inter-relations of bias with respect to psychopathology, and to study (therapeutic) change in attention bias over repeated measurements in a more psychometrically sound manner. This approach, especially when complemented with research aiming to shed light on the mechanisms underlying temporal dynamics of attention bias, may be relevant in efforts to disambiguate the role of attention bias in various forms of psychopathology, such as social anxiety, as well as efforts to therapeutically target attention bias and putatively related psychopathology vulnerability.

## STUDY 2: LANGUAGE USE IN A PUBLIC SPEAKING EXPOSURE AS A PREDICTOR OF SOCIAL ANXIETY SYMPTOM CHANGE[2]

We use language to communicate with others and to understand the world around us. Not only does the content (i.e., nouns, verbs, adjectives, and adverbs) of our words provide insight into our thoughts, focus of attention, and emotional state, but our language style – the way we clarify meaning in written or verbal speech by relating content words to one another with function words – can also serve as an implicit reflection of these otherwise difficult to measure constructs (Chung & Pennebaker, 2007). There are only approximately 500 distinct function words (i.e., pronouns, articles, conjunctions, etc.), but these account for almost 60% of spoken or written language (Rochon, Saffran, Berndt, & Schwartz, 2000) and are vital for effective communication. For example, if the function words were removed from the first sentence of this manuscript, it would be difficult to understand its meaning: "use language communicate understand world".

### Pronouns

Of the function words, pronouns are the most commonly used, accounting for 14% of all word usage (Pennebaker, Chung, Ireland, Gonzales, & Booth, 2007). Pronouns are social words by nature, directing the focus of attention toward others (second person pronouns; e.g., "he", "she", "they"), toward the self as separate from others (first-person singular pronouns; e.g., "I", "me", "my"), or toward the self as a part of a social relationship (first-person plural pronouns; e.g., "we", "us", "our").

2. Davis, Baird, Witcraft, Pennebaker, & Smits (in preparation)

First-person pronoun use has been identified as a stable, independent difference variable (Mehl & Pennebaker, 2003; Pennebaker & King, 1999). First-person *singular* pronoun use (e.g., I, me, my) has been demonstrated to reflect an internal focus of attention and to predict negative mood and relationship outcomes (Tausczik & Pennebaker, 2010). For example, manipulating self-focus by having speakers look into a mirror while completing a questionnaire increases first-person singular pronoun use (Davis & Brock, 1975). Further, depression, which is associated with increased self-focus (Ingram, 1990), has been linked to increased use of first-person (particularly singular) pronoun use (Fast & Funder, 2010; Jarrold et al., 2011; Rude, Gortner, & Pennebaker, 2004; Weintraub, 1989). Stirman and Pennebaker (2001) sought to examine language use in 18 suicidal and nonsuicidal poets across the span of their careers and, in accordance with a social disengagement model of suicide, they found that suicidal poets used first-person singular pronouns at a higher rate and first-person plural pronouns at a lower rate. First-person singular pronoun use has also been related to interpersonal problems and interpersonal distress (Zimmermann, Wolf, Bock, Pheam, & Benecke, 2013); neuroticism (Raskin & Shaw, 1988), social anxiety (Anderson, Goldin, Kurita, & Gross, 2008), and marital dissatisfaction (Seider, Hirschberger, Nelson, & Levenson, 2009). This high degree of first-person pronoun use across the mood and anxiety disorders is potentially indicative of an elevated degree of self-preoccupation (Pennebaker, Mehl, & Niederhoffer, 2003), which is associated with negative affect (Pyszczynski & Greenberg, 1992).

In addition to the directional element of focus toward others, there is also work to suggest that first-person *plural* pronoun, or relational pronoun use (e.g., we, us, our) may

be helpful in assessing perceived belongingness to a group. Cegala (1989) examined linguistic parameters of conversational engagement and detachment. Specifically, low involvement in social interaction was characterized by fewer relational pronoun references. More recent work has shown that use of "we" can be indicative of a stronger sense of shared identity (Simmons et al., 2005). Contrary to first-person singular pronoun use, first-person plural pronoun use has been associated with adaptive outcomes, including positive relationship qualities (Agnew et al., 1998; Rohrbaugh, et al., 2012) and coping after a stressor (Chung & Pennebaker, 2007). In sum, first-person pronoun use appears to capture the way that people orient and relate to others, potentially providing insight into an individual's level of social integration (Pennebaker et al., 2003).

Given that we tend to have little to no control over (or memory of) their usage (Chung & Pennebaker, 2007), first-person pronoun use may serve as an implicit marker of (mal)adaptive psychological processes. An obvious area of application is the examination of first-person pronoun use in psychotherapy, where language and communication are fundamental components. Psychotherapy research lacks tangible measures of constructs such as attentional focus, cognition, and processes of therapeutic change, which can create difficulty in understanding why certain therapies are beneficial for some individuals, but not others. Exposure-based therapies have demonstrated their efficacy for anxiety disorders (Hofmann & Smits, 2008); however, we still do not fully understand how and for whom exposure therapy works best. Theory suggests that individuals must attend to and engage with a perceived threatening stimulus in order to learn that it is safe (Foa & Kozak, 1987). Research supports this, with evidence suggesting that avoidance or distraction during

exposure reduces its efficacy (for review, see Hermans et al., 2006). Researchers and clinicians typically rely on fear ratings (i.e., self-reported subjective fear levels) as a proxy for successful engagement in exposure exercises, assuming that individuals who are attending to (and not avoiding) their feared stimulus will have higher levels of fear. However, research does not fully support this, as fear levels during a single exposure session are not reliable predictors of anxiety symptom reduction (Craske et al., 2008).

Accordingly, novel measures of attention and avoidance of threat within an exposure session are necessary to learn more about exposure therapy processes A commonly used research protocol for group exposure therapy for social anxiety disorder (Guastella et al., 2009; Hofmann, 2004; Hofmann et al., 2006; Smits et al., 2014) involves having all participants deliver the same speech (a five-minute speech on the topic "social anxiety and the model for exposure therapy"), offering a naturalistic, standardized platform for examining language differences between individuals to provide initial insight into word choice during exposures and SAD symptom change.

**Aims**

We examined the language used within a single public speaking exposure during group treatment for SAD to examine whether first-person pronoun use (singular and/or plural) would predict SAD anxiety severity one week post-exposure. We hypothesized that (1) increased first-person singular pronoun use (e.g., "I", "me", "my") would predict higher social anxiety symptoms post-exposure, given its associations with depression, relationship difficulties, and increased self-focus. Conversely, we hypothesized that (2) increased first-person plural pronoun use (e.g., "we", "us", "our") would predict lower social anxiety

symptoms post-exposure, given its associations with attention toward others, positive group dynamics, and healthy coping.

## Method

### Participants

Data from this study was derived from a larger trial investigating the efficacy of yohimbine-augmented cognitive behavioral therapy for social anxiety disorder. Participants were adults (*n*=34) in the Dallas area between the ages of 18 and 65 who met DSM-IV criteria for a principle diagnosis of social phobia and who reported a significant fear of public speaking. Exclusion criteria included comorbid medical and psychiatric diagnoses contraindicated with the use of yohimbine. Full information on inclusion and exclusion criteria, as well as participant characteristics, is provided in the outcomes from the original study (Smits et al., 2014).

### Procedure

The original study consisted of a 5-week group CBT intervention in which participants received either yohimbine or pill placebo one hour prior to each exposure session. The first session consisted of psychoeducation regarding exposure therapy and social anxiety disorder. At their first exposure session (i.e., session 2), participants completed an exposure exercise wherein each participant was asked to deliver a 5-minute speech on the same topic ("social anxiety disorder and the model for exposure therapy"). Exposure exercises in subsequent sessions were personalized. Participants completed measures of social anxiety symptom severity prior to each exposure session. The current manuscript reports data obtained pre-session 2 (the first public speaking exposure) and one

week later (i.e., pre-session 3). We opted to examine the linguistics in this exposure as all participants completed the same public speaking exercise, and would thus have comparable data.

**Measures**

Linguistic Inquiry and Word Count (LIWC; Pennebaker, Boyd, Jordan, & Blackburn, 2015). Speeches from session 2 were transcribed and then coded using a program called Linguistic Inquiry and Word Count (LIWC; Pennebaker, Booth, & Francis, 2007), which computes the percentage of words that fall into one of over eighty categories (e.g., personal pronouns, affective processes). LIWC has two main features: processing text files (e.g., essays, blogs) by systematically analyzing each word individually and comparing it to a dictionary file (the second feature), which houses the definition, connotation, and part of speech of the individual word (Tausczik & Pennebaker, 2010). For instance, the word "it" would be categorized into three categories: 1) a function word; 2) more specifically, a pronoun; and 3) even more specifically, an impersonal pronoun. Once LIWC has coded all of the words in a document, it calculates the percentage of each category throughout the document, and lists the rate of usage across all LIWC categories by comparing the category's usage to the total number of words in the text. As such, LIWC reveals what is being communicated (i.e., content words) and how it is being communicated (i.e., style words). The internal consistency of LIWC is measured independently for each category (i.e., the internal consistency of each category's constituent words), rather than providing a Cronbach's $\alpha$ for the output as a whole.

Corrected internal consistency differs for each category but is typically good, with a category-wide average Cronbach's $\alpha = 0.68$ (Pennebaker et al., 2015).

Variables derived from LIWC and used in the current analysis were the rate of first-person singular pronoun use (i.e., rate of "I" usage) and first-person plural pronoun use (i.e., rate of "we" usage), calculated as a percentage of total word count during the speech.

Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987). The LSAS is a two-scale, 24-item self-report measure of social anxiety symptoms. Participants are asked to rate both their anxiety and avoidance of commonly feared social situations is rated on a Likert scale from 0 to 3, for a total score from 0 to 144. The self-report version of the LSAS was used in the current study, which has been shown to have good psychometric properties that are comparable to the clinician-rated version (Cronbach's $\alpha = 0.95$; Fresco et al., 2001). LSAS ratings were obtained prior to sessions 2 and 3.

**Data Analysis**

The study hypotheses were tested using a multiple regression analysis with LSAS scores (collected one week after the exposure session) as our dependent variable. In order to control for the influence of potential third variable explanations, several different predictor variables were included in the model alongside initial LSAS scores (collected prior to the exposure session) and original study condition (Yohimbine versus placebo). Given the differences in language based on gender, race, age, and education level (for review, see Tausczik & Pennebaker, 2010), these variables were added to initial models. Additionally, given the correlations between language and depression symptoms (Rude et al., 2004) and language and extraversion (Mehl, Gosling, & Pennebaker, 2006), measures

of initial depression symptom severity (using the Beck Depression Inventory-II or BDI-II; Beck, Steer, & Brown, 1996) and extraversion (using the extraversion subscale of the NEO Five-Factor Inventory or NEO-FFI; Costa & MacCrae, 1992) were also added to the model as predictors. In order to assess for the contribution of each set of components to the final model, we added components to the regression analysis in blocks, with initial LSAS scores entered in step 1, plausible third variables added in step 2, and linguistic variables added in step 3.

## Results

### Data Cleaning

In checking for normality of the dependent variable (i.e., LSAS scores one week after the exposure session), one data point was found to be 3 standard deviations outside of the mean, and was considered an outlier. The below analyses were conducted both with and without this data point, and the overall findings did not change, regardless of its inclusion. Accordingly, we opted to retain this data point in the final reported analyses.

Also, in *post hoc* analyses, the study authors returned to the dataset to check the use of "we" as utilized in participants' speeches and found that in all instances, participants used "we" to address the audience (rather than using the "Royal We", or addressing an out-group other than the audience).

### Baseline Characteristics

Of those with complete data to conduct our analyses ($N$=34), participants were on average 37.26 years old (SD=12.96), and were predominantly Caucasian (61.8%), male (67.6%), and college educated (44.1%). The average baseline LSAS score was 66.47

(*SD*=23.94). See Table 1 for all participant characteristics and word counts at baseline. Baseline LSAS and BDI-II scores were not correlated with either of the word use rates, and "I" and "we" use were not correlated (all $p$ values > 0.05). Examining changes in language use throughout the speech exposure, there were no differences from the first to second half of the speech in either "I" use rates, $t(66) = -.81$, $p=.421$, nor "we" use rates, $t(66) = 1.09$, $p=.280$.

A *post-hoc* power analysis (power=0.8, α=.05) indicated that the final model, with our sample size of 34 participants, allowed us the sensitivity to detect a medium to large effect size ($f^2=0.32$).

| Variable | Mean | *SD* |
|---|---|---|
| Age | 37.26 | 12.96 |
| Pre-exposure LSAS | 66.47 | 23.94 |
| BDI-II | 10.47 | 10.21 |

| | | |
|---|---|---|
| NEO-FFI Extraversion | 18.33 | 6.29 |
| Word count: Total (length of speech) | 550.47 | 201.79 |
| Rate of "I" use | 7.26 | 3.98 |
| Rate of "we" use | 0.72 | 1.20 |
| | N | % |
| Yohimbine condition | 17 | 50.0 |
| Female | 11 | 32.4 |
| White | 21 | 61.8 |
| College graduate | 15 | 44.1 |

*Note*: Each of the indices reporting language use report word use as a percentage of total word count.

**TABLE 2.1** PARTICIPANT CHARACTERISTICS (*N*=34).

**Language Use as a Predictor Symptom Severity One Week Post-Exposure**

In step 1, baseline LSAS scores were added to the model and significantly predicted next-week LSAS scores [$B$=.88, $t(32)$=10.42, $p$<.001]. In step 2, all proposed third variables were added to the model, none of which were significantly predictive of the dependent variable (all $p$-values > .05). In step 3, "I" use, "we" use, and negative emotion word use rates were added to the model. The contributions of each block of variables to the final model are provided in Table 2.

In the final model, only baseline LSAS scores [$B$=.79, $t(23)$=8.04, $p$<.001] and "we" use [$B$=-.24, $t(23)$=-2.57, $p$=.017] were significantly predictive of LSAS scores one week later, such that, consistent with our hypothesis, higher use of "we" within the public

speaking exposure predicted lower SAD symptom severity one week later. Inconsistent with our hypothesis, "I" use [$B$=.11, $t$(23)=1.12, $p$=.273] was not significantly predictive of SAD symptom severity one week later.

The multiple correlation coefficient ($R$) of the final model was .93, suggesting that 87% of the variation in LSAS scores one week following the exposure could be accounted for by our final model. The addition of the linguistic variables (added in step 3) uniquely accounted for approximately 6% of the unique variance in outcomes.

| Step | R | R square | Adjusted R Square | Std. Error of the Estimate | R Square Change | Sig. F Change |
|---|---|---|---|---|---|---|
| 1 | .88 | .77 | .77 | 11.54 | .77 | .000 |
| 2 | .90 | .81 | .74 | 12.04 | .03 | .728 |
| 3 | .93 | .87 | .81 | 10.39 | .06 | .013 |

*Note:* Step 1 predictors: Baseline LSAS; Step 2 predictors: Baseline LSAS, condition, gender, age, race, education level, BDI-II score, and NEO-FFI extraversion score; Step 3 predictors: Baseline LSAS, condition, gender, age, race, education level, BDI-II score, NEO-FFI extraversion score, "I" word use rates, and "we" word use rates.

**TABLE 2.2** MODEL SUMMARY.

## Discussion

The findings of our regression analysis were partially consistent with our hypotheses. Inconsistent with our first hypothesis, "I" use during a public speaking exposure was not predictive of SAD symptom severity one week following an exposure session. Consistent with our second hypothesis, "we" use was significantly predictive of

next-week symptoms, such that higher use of "we" during the public speaking exposure predicted lower next-week SAD symptom severity.

**Pronoun Use**

Our finding that "I" use did not predict social anxiety symptom changes may be attributable to the fact that "I" use, given its well-researched relationship with negative mood and relationship outcomes (for review, see Tausczik & Pennebaker, 2010), better reflects depression and general poor coping, rather than being sensitive to social anxiety severity changes. We attempted to control for the effects of mood by adding pre-exposure symptoms of depression to the model; however, this measure was also not correlated with "I" use. It is possible that, though we were powered to detect medium to large effect sizes, we were underpowered to detect small effects if they existed.

Our findings regarding first-person plural pronoun use raise a number of possible explanations that warrant consideration, including facilitated attentional focus toward perceived social threat (i.e., the other group members in the audience), increased group cohesion, and reduction in stigma. As previously reported (Tausczik & Pennebaker, 2010), pronoun use may reflect attentional focus; accordingly "we" users may have attended more to audience. Given the theorized importance of attending to the perceived threatening stimulus (in this case, the audience) during exposure therapy (Foa & Kozak, 1986), greater "we" users may have derived more benefit from the intervention by focusing on the audience, which was reflected in their word selection. Alternatively, greater use of "we" may reflect a greater sense of group cohesion. Greater perceived closeness between the self and group is related to lower social anxiety severity. Previous work suggests that an

individual's perception may change in two ways over the course of successful exposure therapy: 1) greater identification with the in-group (those in treatment) and 2) greater identification with the out-group (those in the community without SAD; Meuret et al., 2016). Finally, it may also be that greater use of "we" is associated with a decrease in the stigma associated with social anxiety disorder. Mental illness has been associated with stigma (Corrigan & Watson, 2002; Link, Struening, Rahav, Phelan, & Nuttbrock, 1997; Rüsch, Angermeyer, & Corrigan, 2005); thus, there may be a desire to conceal one's social identity (Goffman, 1963). Individuals who use "we" may feel more connected and accepted within the group, which promotes a subsequent reduction in bias and need to hide identity (Meuret et al., 2016).

**Limitations and Future Directions**

Our addition of language variables to the model accounted for roughly 5% of the variance in our outcome measure ($R^2$ change = .05). Using the formula $f^2 = R^2/(1-R^2)$, this corresponds to an effect size of $f^2$=.05. According to Cohen's (1992) conventions, an $f^2$ of .02 equates to a small effect size, while .15 reflects a medium effect size. Though our effect sizes are small to medium, it is important to note that we focused on one session of exposure therapy, and it is possible that the observed effects would accumulate over longer protocols. Future research should replicate our study, examining pronoun use as an implicit predictor of SAD symptom change over the course of exposure therapy. Further, future researchers may consider combining this approach with more direct measures of group cohesion, mental health stigma, immersion, or attentional focus in order to better understand why

first-person plural pronoun use might be beneficial within an exposure session. Our study is limited by our lack of these measures, as well as a relatively small sample size.

Overall, use of implicit measures such as linguistic selections may offer us useful insight into how participants are processing the exposure experience, which may help identify potential mechanisms of change. Learning more about *how* exposure works (e.g., perhaps via increased attention toward the feared social stimuli) may aid in the development of procedures to enhance exposure therapy among those who currently do not benefit from this treatment. Linguistic analysis offers an avenue for learning more about these processes in an unobtrusive manner, though future research is necessary to glean further insight beyond this preliminary analysis. For example, in order to assess whether first-person plural pronoun use reflects attention toward threat, future research could compare pronoun use in an exposure where attention is directly manipulated (e.g., by using distraction), or where approach is directly manipulated (e.g., by using strategies demonstrated to increase social threat-approach, such as testosterone administration; van Honk et al., 1999), as attentional control is one component on approach and/or avoidance in exposure therapy.

Finally, in future studies, researchers may consider exploring the effects of instructing participants to address the group as a whole (i.e., by using "we") in public speaking exposures. It is unclear whether these utterances need to be spontaneous in order to be beneficial. If found to be useful in enhancing exposure outcomes, instruction on language use offers a practical, efficient clinical strategy.

## STUDY 3: A RANDOMIZED CONTROLLED STUDY OF POWER POSING BEFORE PUBLIC SPEAKING EXPOSURE FOR SOCIAL ANXIETY DISORDER: NO EVIDENCE FOR AUGMENTATIVE EFFECTS[3]

Testosterone, a steroid androgen hormone, has been shown to be an important regulator of social motivational behavior, and particularly approach behavior. Several studies have now demonstrated that testosterone administration increases social approach motivation (Bos, van Honk, Ramsey, Stein, & Hermans, 2013; Enter, Spinhoven, & Roelofs, 2014; Hermans, Putman, Baas, Koppeschaar, & van Honk, 2006; Hermans, Ramsey, & van Honk, 2008; Radke et al., 2015; Terburg, Aarts, & van Honk, 2012; van Honk et al., 2001; van Honk, Peper, & Schutter, 2005; van Honk & Schutter, 2007). Given these observations, testosterone levels emerge as a potentially important target for clinical interventions that rely on approach behavior. Exposure therapy, an established treatment for social anxiety disorder (SAD; Hofmann & Smits, 2008), involves systematically and repeatedly approaching feared social cues (i.e., stimuli perceived as threatening) in order to re-establish a sense of safety around these cues (i.e., fear extinction; Hofmann, et al., 2004; Powers, et al., 2007). Though efficacious for SAD (Hofmann & Smits, 2008), there is much room for improvement, and thus targeting testosterone levels may hold clinical value. Indeed, recent basic research shows that testosterone administration can facilitate approach toward angry (i.e., perceived socially threatening) faces (Enter, Spinhoven, & Roelofs, 2016), and reduce gaze avoidance (Enter, Terburg, Harrewijn, Spinhoven, &

[3] Davis, Rosenfield, Roelofs, Kolb, Powers, & Smits (under review).

Roelofs, 2016) among persons with SAD. Accordingly, increasing testosterone levels prior to exposure therapy may lead to enhanced fear extinction and thus better outcomes. In addition to testing the effects of direct testosterone administration, it is also important to develop and test non-pharmacological augmentation strategies that are preferable to patients and easily implemented into an exposure session, and thus easier to disseminate (McHugh et al., 2013).

Results from a recent study indicate that it may be possible to manipulate testosterone via changes in posture (Carney et al., 2010). In this study, men and women were asked to hold either poses associated with dominance and high power (e.g., expansive, open postures; or power poses) or poses associated with submission and low power (e.g., contractive, closed postures; or submissive poses) for two minutes. Participants in the power posing condition evidenced increases in testosterone levels, decreases in cortisol levels, and increases in subconscious feelings of power and risk taking. Though a recent study (Ranehill, Dreber, Johannesson, Leiberg, Sul, & Weber, 2015) - published after the current study was initiated - successfully replicated the findings regarding power posing leading to increased subjective feelings of power, they found no impact of postural manipulation on hormone levels. However, it is important to note that the Ranehill study protocol deviated from the Carney study in important ways (e.g., participants were given the rationale for the postures rather than using deception; see review by Carney and colleagues, 2015). Additionally, a recent review notes a history of the embodied effects of expansive postures on feelings of dominance and power (Carney et al., 2015). In sum, power posing has demonstrated the capacity to increase: confidence and positive attitude

towards the self (Briñol, Petty, & Wagner, 2009), feelings of power and risk tolerance (Carney et al., 2010; Cesario & McDonald, 2013; Huang, Galinsky, Gruenfeld, & Guillory, 2010; Michalak, Mischnat, & Teismann, 2014), risk taking in a social context (Fischer, Fischer, Englich, Aydin, & Frey, 2011), action orientation (Cesario & McDonald, 2013; Michalak et al., 2014), self esteem (Riskind & Gotay, 1982), persistence (Riskind, 1984; Stepper & Strack, 1993), and pride feelings (Welker, Oberleitner, Cain, & Carré, 2013). Additionally, two studies have demonstrated potential anxiolytic effects of power posing, noting reduced fear (Riskind & Gotay, 1982) and decreased threat perception (Welker et al., 2013). Accordingly, there is overlap between the psychological and anxiolytic effects of power posing and the effects of testosterone administration, and the Carney (2010) study lends preliminary evidence that power posing may cause increases in endogenous testosterone levels.

**Aims**

The current manuscript details a proof-of principle study examining power posing as an augmentative strategy for exposure therapy for SAD. We tested whether power posing (compared to submissive posing or rest) would (1) increase testosterone; (2) result in superior exposure therapy outcomes (i.e., decreased symptom severity and fear responding during a public speech); and (3) whether increased testosterone would mediate facilitated symptom reduction among individuals engaging in power posing. Due to the aforementioned research indicating potential decrements in cortisol (Carney et al., 2010) and/or anxiolytic effects of power posing (Riskind & Gotay, 1982; Welker et al., 2013),

we also tested whether power posing (compared to submissive posing or rest) would (4) decrease cortisol and (5) result in increased self-reported fear within the exposure session.

## Method

### Participants

Participants (aged 18 to 70) were recruited from advertisements at the University of Texas and in the Austin community (see Table 1). Participants (N=73) were diagnosed with SAD as their primary psychiatric diagnosis (i.e., the most important source of current distress) and endorsed fear of public speaking as a primary concern. Exclusion criteria included current use of testosterone enhancing products or corticosteroid medications, a lifetime history of bipolar or psychotic disorders, a history of substance or alcohol use disorders in the past six months, significant suicidal ideation, current utilization of psychotherapy for SAD, and prior non-response to exposure therapy. Participants using psychotropic medication were allowed to participate in the study if they had been on a stable dose of medication for three weeks prior to the treatment session. Participants were not paid for their participation, though students were offered course credit. All participants completed in the informed consent process prior to beginning the study procedures. This study was registered on clinicaltrials.gov (NCT02482805).

### Procedures

**Eligibility Screening.** Participants first completed an online prescreen which was examined for clear exclusion criteria (e.g., no social anxiety symptoms, current exposure therapy treatment, etc.). Participants who appeared eligible were invited to participate in a phone interview for diagnostic screening, using the M.I.N.I. International Neuropsychiatric

Interview (M.I.N.I.; Sheehan et al., 1998) to evaluate the presence of psychiatric inclusion and exclusion criteria. Eligible participants were invited to participate in the treatment session. See Figure 1 for consort diagram.

**Randomization.** Participants were randomized to participate in power posing, submissive posing, or rest (no posture manipulation) using a randomization sheet developed by an independent investigator. Randomization was blocked by subject pool (i.e., community participants versus University students, and low SAD severity versus high SAD severity) to control for potential differences in compensation and baseline severity levels. The cut-off for high SAD severity was a score of 70 or higher on the pre-treatment Liebowitz Social Anxiety Scale (LSAS) total score. Randomization information was placed in envelopes that were not opened by the therapist until immediately prior to the posture manipulation (i.e., the therapist was blind to treatment condition throughout the rationale and exposure planning components of the treatment session).

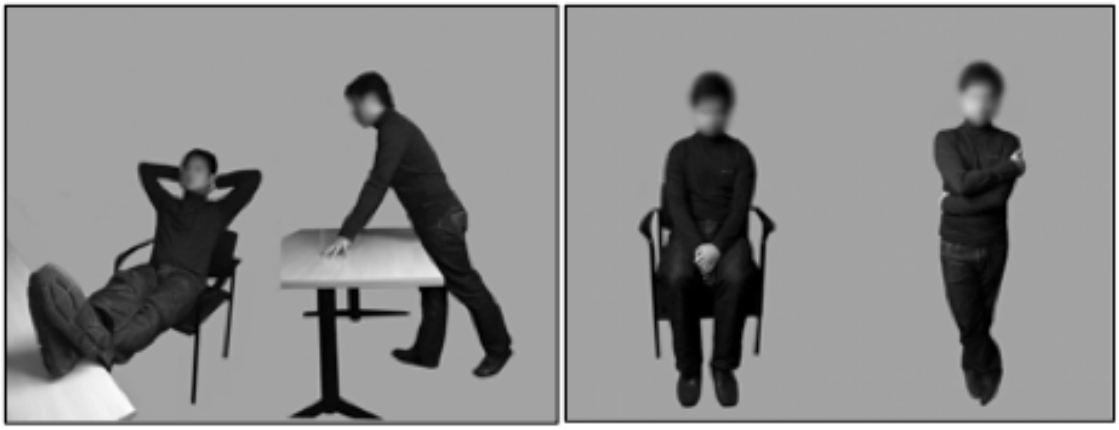
**Treatment Session.** Rodebaugh and colleagues (2013) recently described a standardized test (i.e., clinical assay) for examining augmentative strategies (e.g., pharmacotherapy) for exposure therapy for SAD in an efficient, feasible manner. This protocol involves a standardized exposure therapy session in which participants plan a public speaking exposure expected to elicit a peak fear rating (using the Subjective Units of Distress Scale or SUDS, described below) of 75, varying the following flexible elements: topic of speech, utilization of confederate audience members, availability of notes, time for preparation, and reaction of experimenter. The intent of this approach is to standardize the experience of anxiety (using predicted SUDS), rather than standardizing

elements of the procedures. Therapists were three graduate-student level therapists trained and supervised by the senior author.

Prior to the session, participants completed questionnaires assessing demographic and SAD severity measures. At the onset of the session, participants first watched a video describing the cognitive-behavioral model of SAD and the rationale for exposure therapy. Therapists then familiarized the participant with the SUDS scale and guided participants in designing a 5-minute speech exposure with specific behavioral goals designed to decrease avoidance. The participants then participated in the posturing manipulation protocol (see below). Following the posturing manipulation, they began their speeches after a brief wait period. Participants provided fear ratings at the start and end of each speech (recalling the highest level of fear they experienced over the course of the speech), and delivered the same 5-minute speech (with the same behavioral goals) three times. After the exposures, participants processed the exercise with the therapist.

**Posturing Manipulation Protocol.** Individuals were randomized to one of three posturing conditions completed prior to the speech exposures. In order to prevent attribution effects, participants were not informed of the true purpose of these poses. Instead, we followed the procedures of the Carney (2010) study, providing the participants with the rationale that they were being put into certain bodily positions to test physiological responses (via the heart rate and skin conductance monitors) as a function of sensor placement relative to their heart. After planning their speech exposure, participants put on heart rate and skin conductance monitors (i.e., watches and finger bands). Participants in the power posing condition were asked to hold two 1-minute, open, expansive postures

associated with high power. Participants in the submissive posing condition were asked to hold two 1-minute, closed, contractive postures associated with low power. These postures were identical to those used in the Carney (2010) study. Participants in the rest (no posture) condition were asked to sit and wait for 2 minutes, with the rationale that their physiological responses were being tested (but without the sensor placement rationale). Hormone samples were assessed immediately prior to the posturing manipulation protocol, then 17 minutes later (see full description of hormone sampling below).



*Note:* Two high-power (dominance) postures (left). Two low-power (submissive) postures (right).

**FIGURE 3.1** DOMINANT AND SUBMISSIVE POSTURES.

**Post-Treatment Session.** At one week post-treatment, individuals returned to the laboratory to complete questionnaires assessing for changes in SAD severity, as well as to participate in a behavioral assessment task (BAT). They delivered the same 5-minute speech they planned and delivered during the treatment session (to the same audience and with the same speech element manipulations and behavioral goals) as the same indices

were measured as in the exposure session (e.g., SUDS). After the BAT, the therapist discussed with the participant how they could continue to apply home-practice strategies. Participants were then debriefed about the posturing deception and those who wished to receive additional exposure-based treatment were provided with CBT treatment referrals in the Austin community.

**Measures**

**Salivary Hormone Samples.** Participants provided a passive drool saliva sample (1) immediately prior to the behavioral posturing manipulation, then (2) 15 minutes later (e.g., approximately 17 minutes after the first sample), immediately prior to the speech exposures. This is in line with the Carney (2010) study, wherein a change in hormone levels was evident 17 minutes after power posing. Saliva samples were collected using the passive drool protocol (in 2ml vials). The samples were stored at -20° C until radio immune assay was performed by Dr. Clemens Kirschbaum's laboratory in Dresden, Germany, which yielded both testosterone and cortisol values.

**Endocrine Questionnaire.** Participants were asked to provide information that could affect their hormone levels on the day of the session, including meals consumed, physical activity, menstrual cycle stage, use of oral contraceptives, and sleep/wake times. This questionnaire was used in the Carney study (2010) upon which the dominance posturing protocol has been based. Responses to this questionnaire were used as control variables in the hormone analyses.

**Liebowitz Social Anxiety Scale (LSAS).** The LSAS is a 24-item scale which yields separate scores for fear and avoidance in both social and performance situations. It

is a commonly used outcome measure in SAD treatment studies and has very good psychometric properties (Heimberg et al., 1999; Safren et al., 1999). As participants in this study endorsed fear of public speaking as their primary concern (and because the exposure consisted of public speaking exposure), we utilized the "social performance" subscale of the LSAS as a primary outcome measure. This subscale utilizes fear and avoidance ratings for the 13 items of the LSAS assessing performance situations (e.g., "giving a report to a group", "acting, performing, or giving a talk in front of an audience"). The range of the LSAS performance total subscale is 0 to 78. The LSAS was completed prior to the treatment session and one week later.

**Subjective Units of Distress Scale (SUDs).** Participants provided fear ratings at the beginning and end of each speech (i.e., initial, end, and retrospective peak SUDS) using the SUDS scale (Wolpe, 1958), which ranges from 0 to 100. We used the peak rating within the exposure session (i.e., the highest rating of the three speeches), as well as the difference between this peak rating and the end (final) rating after the third speech, as process measures to examine for between-group differences in within-session fear activation and fear extinction. We also looked for between-group differences in peak fear during the BAT as a primary outcome measure.

**Analyses**

Three separate AN(C)OVAs were performed with treatment group (0, 1, 2) as a predictor variable to determine whether individuals assigned to power posing (relative to submissive posing or rest) evidenced the following changes in dependent variables: (Aim 1) increased post-manipulation testosterone levels (covarying for pre-manipulation

testosterone levels), (Aim 2) decreased peak SUDS at BAT (covarying for pre-treatment SAD severity using the LSAS-performance score), and (Aim 3) decreased post-treatment LSAS-performance (covarying for pre-treatment LSAS-performance). In the event that these predictions were met, a mediation analysis was planned to determine whether (Aim 4) testosterone increases mediated the relation between power posing and decreased peak SUDS at BAT and/or decreased post-treatment LSAS-performance. Finally, to test whether power posing elicited anxiolytic effects, two additional AN(C)OVAs were performed with treatment group as a predictor variable to determine whether individuals who participated in power posing evidenced (Aim 5) decreased post-manipulation cortisol levels (covarying for pre-manipulation cortisol levels), and (Aim 6) lower peak SUDS within the exposure session. In all analyses, demographic (sex, age, race, cohabitation status, education) and pre-treatment severity (LSAS-performance) variables were added to the models as potential predictors, as well as therapist (dummy coded for the 3 therapists) and number of confederates during exposures. In the hormone analyses, all variables from the endocrine questionnaire were additionally added to the model as potential predictors. Given that no study outcomes changed as a function of their inclusion/exclusion, all non-significant control variables were dropped from the models prior to reporting.

An *a priori* power analysis (power=0.8, $\alpha$=.05) was conducted to determine the sample size necessary to detect hormonal effects given the effect sizes in the Carney et al. (2010) study (i.e., $f$ = .36 for testosterone and $f$ =.47 for cortisol findings). This analysis indicated that we needed a total sample size of at least 63 to detect the smallest of these

effects (f = .36). Accordingly, our sample was large enough to detect similarly sized effects to those seen in the Carney study.

## Results

Of the 73 individuals included in the analyses, participants were on average 26 years old (*SD*=7.48), female (71.2%), white (53.4%), and college-educated (49.3%). The average baseline LSAS (performance subscale) score was 37.27 (*SD*=11.96). There were no significant differences in these baseline characteristics between groups (see Table 1). In hormone analyses, six testosterone and seven cortisol data points were identified as outliers (i.e., standardized residuals were >3 standard deviations outside the mean for the condition). Analyses were performed with and without these data points and primary outcomes were unaffected by their exclusion. Accordingly, the testosterone and cortisol analyses presented below exclude these data points for a total of 67 and 66 participants in each, respectively. Of the 73 participants enrolled in the study, only 69 completed the post-treatment LSAS, and only 66 participants completed the post-treatment BAT. The consort diagram is presented in Figure 1.

| | Power Posing (*n*=26) | | Submissive Posing (*n*=27) | | Rest (*n*=20) | |
|---|---|---|---|---|---|---|
| Variable | Mean | *SD* | Mean | *SD* | Mean | *SD* |
| Age | 26.88 | 6.99 | 25.26 | 8.99 | 24.60 | 5.83 |
| LSAS-performance | 37.96 | 14.39 | 36.19 | 10.92 | 37.85 | 10.16 |
| | *n* | % | *n* | % | *n* | % |

| | | | | | | |
|---|---|---|---|---|---|---|
| White | 14 | 53.8 | 14 | 51.9 | 11 | 55.0 |
| Female | 18 | 69.2 | 18 | 66.7 | 16 | 80.0 |
| College graduate | 11 | 42.3 | 16 | 59.3 | 9 | 45.0 |

**TABLE 3.1** PARTICIPANT CHARACTERISTICS BY CONDITION (*N*=73).

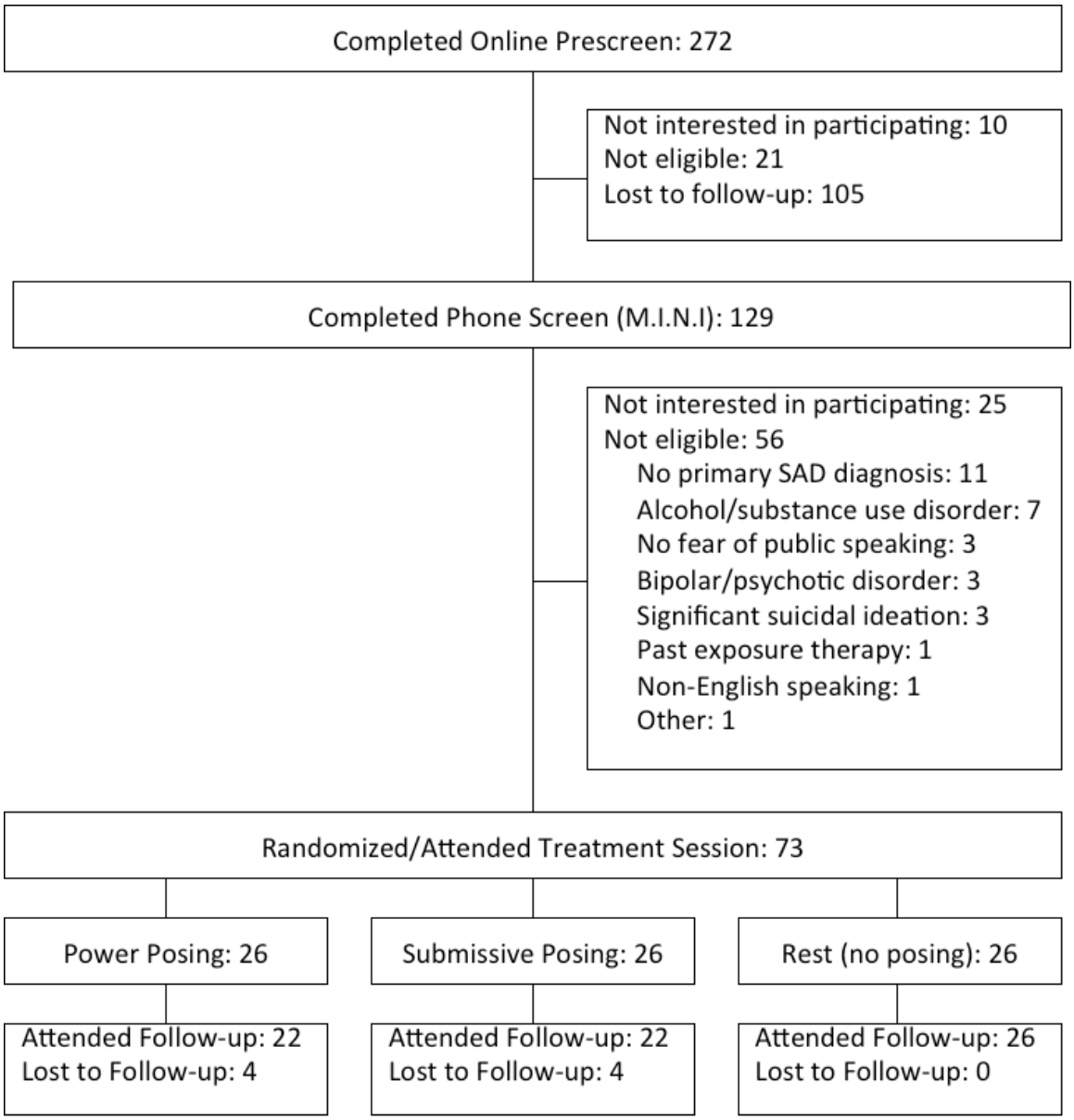



**FIGURE 3.2** CONSORT DIAGRAM.

**Aims 1-4: Does power posing result in testosterone increases that facilitate SAD symptom reduction?**

**Does power posing increase testosterone levels?** Covarying for pre-manipulation testosterone levels, $F(1,63) = 2041.86$, $p<.001$, partial $\eta 2 = .970$, there were no significant

differences in post-manipulation testosterone levels by condition, $F(2,63) = 0.62$, $p>.250$, partial $\eta 2 = .019$. Table 2 provides the post-manipulation testosterone and cortisol means by condition, adjusted for pre-manipulation hormone levels.

| | Testosterone[1] | | | Cortisol[2] | | |
|---|---|---|---|---|---|---|
| Condition | N | Mean | SE | N | Mean | SE |
| Power pose | 24 | 43.48 | 2.10 | 21 | 3.24 | 0.17 |
| Submissive pose | 23 | 44.03 | 2.16 | 25 | 3.14 | 0.16 |
| Rest (no pose) | 20 | 46.78 | 2.31 | 20 | 3.53 | 0.18 |

*Note.* [1]Pre-manipulation testosterone covariate evaluated at 47.29. [2]Pre-manipulation cortisol covariate evaluated at 3.80.

**TABLE 3.2** POST-MANIPULATION HORMONE MEANS BY CONDITION.

**Does power posing result in better exposure outcomes?** Covarying for pre-treatment LSAS-performance scores, $F(1,65) = 100.18$, $p<.001$, partial $\eta 2 = .61$, there were no significant differences in (1 week) post-treatment LSAS-performance scores by condition, $F(2,65) = 0.39$, $p>.250$, partial $\eta 2 = .01$. Similarly, controlling for sex, $F(1,61) = 4.93$, $p=.030$, partial $\eta 2 = .08$, neither pre-treatment LSAS-performance scores, $F(1,61) = 0.94$, $p>.250$, partial $\eta 2 = .02$, nor study condition, $F(2,61) = 2.74$, $p=.072$, partial $\eta 2 = .08$, were significant predictors of peak SUDS at BAT. Estimated marginal means of outcome measures are presented in Table 3.

| | LSAS-performance[1] | | | Peak SUDS (exposure)[2] | | | Peak SUDS (BAT)[3] | | |
|---|---|---|---|---|---|---|---|---|---|
| Condition | N | Mean | SE | N | Mean | SE | N | Mean | SE |
| Power pose | 23 | 33.54 | 1.65 | 26 | 73.10 | 2.80 | 23 | 56.75 | 4.06 |
| Submissive pose | 26 | 33.33 | 1.55 | 27 | 76.53 | 2.69 | 23 | 56.82 | 4.07 |
| Rest (no pose) | 20 | 31.60 | 1.76 | 20 | 74.69 | 3.68 | 20 | 44.95 | 4.52 |

*Note.* [1]Adjusted for pre-treatment LSAS-performance score covariate evaluated at 37.30. [2]Controlling for sex. [3]Controlling for sex and adjusted for pre-treatment LSAS-performance score covariate evaluated at 37.64.

**TABLE 3.3** ESTIMATED MARGINAL MEANS OF OUTCOME AND PROCESS MEASURES.

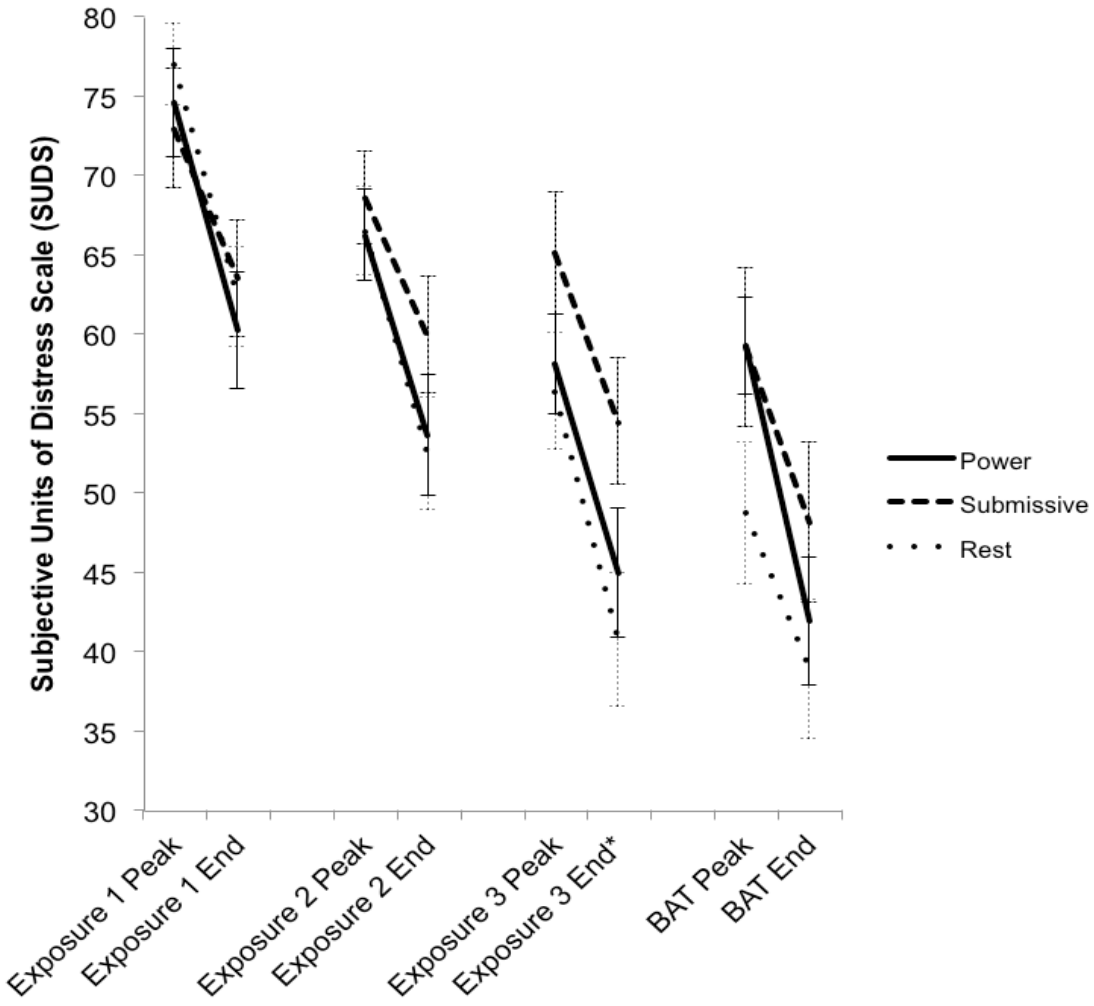
Due to the lack of significant effects of condition on testosterone levels or treatment outcome, mediation analyses (Aim 4) were not performed. In an exploratory post-hoc analysis, we tested whether testosterone/cortisol changes from pre- to post-manipulation were predictive of primary outcome variables. Both hormone variables (i.e., changes in both testosterone and cortisol from pre- to post-manipulation) were added to the models as predictors simultaneously, as they could have opposite effects. Controlling for pre-treatment LSAS, $F(1,58) = 115.39$, $p<.001$, partial $\eta 2 = .67$, neither testosterone change, $F(1,58) = 0.97$, $p>.250$, partial $\eta 2 = .00$, nor cortisol change, $F(1,58) = 0.04$, $p>.250$, partial $\eta 2 = .00$, were predictive of post-treatment LSAS scores. Controlling for pre-treatment LSAS, $F(1,55) = 4.92$, $p=.031$, partial $\eta 2 = .08$, neither testosterone change, $F(1,55) = 0.19$, $p>.250$, partial $\eta 2 = .00$, nor cortisol change, $F(1,55) = 0.01$, $p>.250$, partial $\eta 2 = .00$, were predictive of peak SUDS at BAT.

**Aims 5 & 6: Does power posing result in anxiolysis?**

**Does power posing decrease in cortisol levels?** Covarying for pre-manipulation cortisol levels, $F(1,62) = 246.15$, $p<.001$, partial $\eta 2 = .80$, there were no significant between-condition differences in post-manipulation cortisol levels, $F(2,62) = 1.39$, $p>.250$, partial $\eta 2 = .04$.

**Does power posing decrease subjective levels of fear?** Controlling for sex, $F(1,69) = 10.85$, $p=.002$, partial $\eta 2 = .14$, there were no significant differences between conditions in within-session fear activation (peak SUDS), $F(2,69) = 0.15$, $p>.250$, partial $\eta 2 = .00$. Estimated marginal means are presented in Table 3.

The average peak SUDS across the three treatment session exposure sessions was 77.16 (*SD*=13.85) and the average peak SUDS at BAT was 56.05 (*SD*=20.03). This change was significant, $t(65) = 9.39$, $p<.001$, and represents an average decrease of approximately 27% in peak SUDS from pre- to post-treatment. These values did not differ between conditions (see Figure 2 for SUDS by condition).




*Note,* Error bars are standard errors of the means. * = Significant difference in exposure 3 end SUDS between the submissive and rest conditions, $t(41) = 10.97$, $p<.001$. No other significant differences in means at $p\leq.05$.

**FIGURE 3.3** SUDS WITHIN SPEECH EXPOSURES ACROSS TIME POINTS BY CONDITION.

## Discussion

We conducted a randomized controlled study to compare the effects of power posing, submissive posing, and rest (no posing) prior to a public speaking exposure for

individuals with SAD (*N*=73). This study was based on theory and preliminary evidence that power posing may increase testosterone levels, which may enhance the efficacy of exposure therapy for SAD by increasing approach toward perceived-threatening social stimuli. However, we found no evidence to suggest an effect of power posing on testosterone levels, or on exposure therapy outcomes. There were also no between-group differences in either cortisol levels or in fear activation within the public speaking exposure, suggesting that power posing did not result in anxiolytic effects.

Possible explanations for our lack of findings are related to the sample size, population, and study design. Though our study was powered to detect medium to large effect sizes (as found in the Carney study), it may have been underpowered to detect small effect sizes. Additionally, to our knowledge, our study is the first to apply power posing toward a clinical population (i.e., individuals diagnosed with SAD). It is possible that any hormonal and/or psychological effects of power posing may not have been evident due to the stressful nature of the public speaking exposure for this group of individuals, which may have led to hormonal changes which obscured any effects of our study manipulation. Though we designed the study based on the Carney et al. (2010) study, several features of the study differed from this trial. Individuals in our study were likely highly stressed during the power posing procedures, given that they were aware they would be participating in an upcoming public speaking exposure. We also did not utilize a filler task during the pose (while the Carney study had individuals in the study complete a social filler task). Additionally, the Carney study was conducted in 2008-2009, while our study was conducted after extensive media and University course coverage of power posing. Though

we attempted to control for whether participants in the study were blind to the purpose of the study manipulation by questioning them about the deception at follow-up, it is possible that they were aware of the literature on power posing and of the true purpose of the study. Finally, it is possible that the timing/dose of power posing was not appropriate to yield hormonal changes or enhance exposure outcomes in this study, but it is possible that there could be effects of power posing that would be evident with differential hormone sample assessment times (i.e., shorter or longer time between samples) and/or a larger "dose" of power posing (e.g., longer holding of poses, a shorter period of time between power posing and exposures, or power posing within the exposure session rather than prior to it).

Assuming that the above noted differences in study methodology do not prevent a comparable replication of the Carney study, our findings, along with those of the Ranehill (2015) study, offer no evidence to suggest that power posing impacts hormone levels. Replication is important, however, particularly when sample sizes are relatively small, and null findings contribute to the body of literature regarding the extent of the effects (if any) on power posing. As a testament to this, a recent analysis reviewed the 33 studies identified in the Carney (2015) review of power posing, and accounted for selective publishing (i.e., the failure to publish null findings, or the file-drawer effect; Rosenthal, 1979) using a *p*-curve analysis (Simmons & Simonsohn, 2017). They found that the distribution of *p*-values from the 33 published studies on power posing was indistinguishable from the expected *p*-value if the average effect size were zero, and/or if selective reporting was the sole reason for the significant effects. Thus, power posing does not appear to be a robust method of

facilitating change, whether psychological, behavioral, or hormonal. Our study provides further evidence toward this end.

Finally, our hypothesis that increased testosterone during exposure therapy might lead to facilitated exposure therapy outcomes among individuals with SAD remains untested, as we were unable to successfully increase testosterone levels prior to the public speaking exposure. This remains an important area for future research to address, perhaps by applying low doses of exogenous testosterone prior to exposure to ensure that hormone levels are sufficiently increased. Though our study yielded null findings, the single session assay utilized here (derived from Rodebaugh and colleagues; 2013) offers a timesaving, inexpensive way to test augmentative psychopharmacological strategies, such as testosterone administration. We elicited significant variability (i.e., no floor effects) and meaningful reductions in both pre-post LSAS-performance scores (mean change = -4.41; *SD* = 8.04) and peak SUDS elevations from treatment to BAT (mean change = -21.42; *SD* = 18.53), with only a single treatment session and 1-week follow-up. Utilizing this brief assay as a preliminary proof-of-principle test may be an important step prior to assessing effects on a full dose of exposure therapy. Indeed, assessing the null effects of power posing with this clinical assay, combined with other research regarding the effects of power posing, allows us to assume that power posing is likely not a useful strategy for enhancing exposure therapy outcomes for SAD. Finally, given that exposure is an effective treatment for many with SAD, it may be useful to selectively apply augmentation strategies (such as testosterone administration) to those who have previously not responded to exposure therapy (Mataix-Cols et al., 2017).

# GENERAL DISCUSSION

## Review of Study Findings

**Study One.** Threat-related attention bias figures prominently in contemporary accounts of the maintenance of anxiety disorders, yet longitudinal intervention research relating attention bias to anxiety symptom severity is limited in scope. Capitalizing on recent advances in the conceptualization and measurement of attention bias, we aimed to examine the relation between attention bias, indexed using trial-level bias scores to quantify temporal dynamics reflecting dysregulation of attentional processing of threat (as opposed to aggregated mean bias scores) and social anxiety symptom severity over the course of cognitive behavior therapy (CBT) and one-month follow-up.

Adults with social anxiety disorder (N=39) assigned to either yohimbine- or placebo-augmented cognitive behavioral therapy completed measures of attention bias and social anxiety symptom severity weekly throughout CBT (5 sessions) and at one-week and one-month post-treatment.

Our prediction that 1) modeling attention bias as a dynamic process, rather than using the conventional computation, would yield more reliable indices of attention bias, was met. Our prediction that 2) attention bias would decrease over time was also met. However, our prediction that 3) attention bias reduction and social anxiety symptom reduction would be reciprocally related was not met (i.e., there were no relations between attention bias and SAD symptom changes).

**Study Two.** Studying pronoun and emotion word use within language may provide insight into attentional focus during exposure therapy: first-person singular pronoun use tends to reflect self-focus (and is associated with negative mood and relationship outcomes) while first-person plural pronoun use tends to reflect other-focus (and is associated with adaptive outcomes, such as group belongingness. Analysis of the linguistic features of speeches delivered during exposure therapy for SAD offers an implicit method for detecting attentional focus during exposure therapy, which may predict whether the exposure session is successful, resulting in reduced social anxiety symptoms. Accordingly, we examined pronoun use during a public speaking exposure for SAD to assess whether these linguistic variables could predict symptom reduction post-exposure.

Thirty-four speeches were transcribed using the Linguistic Inquiry and Word Count (LIWC). Regression analyses were performed using first-person singular (i.e., "I") use and first-person plural (i.e., "we") use as predictors of social anxiety symptom changes from pre- to post- exposure.

Our prediction that 1) greater "I" use would predict higher social anxiety symptoms post-exposure was not met. However, our prediction that 2) greater "we" use would predict lower social anxiety symptoms post-exposure was met.

**Study Three.** There is evidence that testosterone, a hormone associated with threat-related social approach, may be an important target for exposure therapy for SAD, and that it may be possible to manipulate testosterone levels by power posing (i.e., briefly holding postures associated with dominance and power). We performed a randomized controlled study designed to test the efficacy of power posing (i.e., briefly holding postures associated

with dominance and power) as an augmentative strategy for exposure therapy for social anxiety disorder (SAD).

Seventy-three individuals diagnosed with SAD were assigned to one of three conditions: power posing, submissive posing, or rest (no posing) prior to participating in an exposure therapy session. Participants were assessed for between-group differences in pre- and post-manipulation salivary hormone levels, within-session subjective experiences of fear, and pre- and 1-week post-treatment SAD severity outcome measures.

Our prediction that 1) power posing (compared to submissive posing or rest) would result in increases in endogenous testosterone levels was not met, nor were our predictions that 2-3) power posing would result in superior exposure outcomes via an increase in testosterone. We also did not find evidence to support our predictions that 4) power posing would increase cortisol levels or 5) increased fear activation.

**Implications for Studying Attention in Exposure Therapy**

**Changes in attentional biases may not be an important mechanism of symptom reduction in exposure-based therapy.** Due to our study design we cannot definitively state that changes in attention bias are not a mechanism of change; however, our findings leave open this possibility. It is possible that there is a relation between attention bias and SAD symptom severity, but that the relation is non-linear (e.g., one may change more quickly than another, and/or the timing of changes may not be consistent over the course of assessments). The timing of assessments in our analysis did not allow for us to test for this possibility. Further research, ideally consisting of exposure therapy and a control

comparison group), is necessary to learn more about whether attention bias changes are important to exposure outcomes.

**Exposure-based treatments may impact attention bias toward threat, but not attention bias away from threat.** We demonstrated that attention bias toward threat (i.e., hypervigilance) appeared to decrease over the course of exposure; however, attention bias away from threat (i.e., attentional avoidance) did not change over the course of our study. This replicates a previous study wherein individuals who participated in cognitive-behavioral therapy (compared to those who were assigned to a waitlist condition) also evidenced declines in attention toward threat (but not attention away from threat). These findings are surprising given the rationale for exposure therapy, where reduced avoidance of threat in general is emphasized. If exposure therapy does not effectively alter attentional avoidance, this may be an important target for exposure therapy augmentation in SAD (i.e., using attention bias modification or other attention training paradigms alongside exposure therapy). Additionally, if directly targeted alongside exposure therapy, attention bias changes might then emerge as a predictor of SAD symptom change.

**Computerized attention bias tasks may not be sensitive to differences in SAD symptom severity.** Some previous studies (Boettcher et al., 2013; Julian, et al., 2012; McNally et al., 2013), have found no evidence for biased attention at baseline in samples of individuals with SAD. Though we did find evidence for biased attention (both away from and toward threat) in our sample at baseline, these biases were not correlated with SAD symptom severity. This replicates previous research demonstrating that attention bias task performance may not be a strong predictor of SAD symptom severity (for review, see

van Bockstaele et al., 2014), which has several potential explanations. First, it is possible that computerized attention bias measurements may not have the sensitivity to detect differences in attention biases based on SAD symptom severity. Second, as noted by van Bockstaele and colleagues (2014) the poor reliability of attention bias measures implies that the measure captures noise, prohibiting correlation with levels of SAD severity even if the two are causally related. Though the TLBS scoring appears to improve the reliability of the attention bias measure in our study, it still may not be adequate. Finally, it is possible that the relation between SAD symptom severity and attention bias is non-linear. This correlation is complicated by the fact that there are different types of biased attention (e.g., hypervigilance, avoidance) that may be operating inconsistently across individuals. For example, certain stimuli in our attention bias assessment may have led to hypervigilance for one individual, and avoidance for another. Regardless of the explanation, given that computerized attention bias tasks tend to not be predictive of SAD symptom severity, this limits their use as a measure of attention as a mechanism of change in exposure therapy.

Ways to improve these tasks include use a more reliable scoring methods, such as TLBS (see below), careful design of the study stimuli (i.e., choosing stimuli that is directly relevant to the individual being studied) and inclusion of eye tracking measurement. Adapting newer virtual reality interfaces which use eye tracking devices to be used in exposure therapy may also be effective (for review, see Lindner et al., 2017).

**A dynamic scoring procedure may be more reliable for assessing repeated measurements of attention bias in SAD.** Our study is the first to apply a novel scoring approach to quantify attention bias as a dynamic process in time (i.e., TLBS) toward

exposure therapy for SAD. Our finding that TLBS scores were more reliable replicates and extends previous research (Zvielli et al., 2015a). Not only does this have implications for repeated measures studies of attention bias (i.e., that this scoring technique may be more reliable), this finding also adds to the evidence suggesting a dynamic process perspective of attention bias, which may improve attention bias measurement more broadly, increasing our ability to study therapeutic changes in attention bias.

**Linguistic features may offer an unobtrusive way to study attentional focus during exposure therapy.** As our study is the first to study language during a public speaking exposure, more research is necessary, both to replicate our finding regarding the beneficial effects of "we" us, as well as to better understand the underlying cause of the effects (i.e., whether this is relation due to increased attention toward the audience, greater cohesiveness of the group, or a reduction in stigma, for example). In general, studying language may offer insight into exposure therapy processes. However, there are practical difficulties to studying language in exposure therapy. First, for the study detailed in this manuscript, recorded speeches had to be transcribed prior to linguistic analysis, making it a time-intensive endeavor. Additionally, though we may be able to find relations between linguistic features and exposure outcomes, it is difficult to make much of these relations (as in our study) without also relating the linguistic features to concurrent measures of the purported underlying construct. Accordingly, in order to use pronoun use as a proxy for attentional focus within exposure therapy, we would also need a valid measure of attentional focus within the session (rather than an assessment of attention prior to or after

the session, as in attention bias assessment tasks). As mentioned previously, eye-tracking devices may be a good application for assessing in-session attentional focus.

As noted previously, if our findings regarding "we" use are replicated, it may also be interesting to test whether utterances have to be spontaneous in order to be beneficial to exposure outcomes. In other words, could manipulating the prompt of the exposure topic by instructing participants to directly address the audience in their speech (compared to generic speech instructions) yield benefit by intentionally creating increased focus on the audience?

**Implications for Studying Approach in Exposure Therapy**

**Power posing is not an effective way to increase testosterone levels for studying approach.** We anticipated that power posing would be an efficient way to manipulate approach during exposure therapy without administering testosterone exogenously. However, our study, alongside another failed replication attempt (Ranehill et al., 2015) seems to suggest that there are no hormonal effects of power posing. Indeed, the recent review by Simmons & Simonsohn (2017) indicates that much of the excitement surrounding the purported effects of power posing may simply be attributable to selective reporting (i.e., the fact that statistically significant results are much more likely to be published than null findings). This points to a larger problem in (though not specific to) the field of psychology that is outside the scope of this manuscript. We also plan to examine individual patterns of hormone changes within the exposure session in an exploratory fashion to examine whether endogenous testosterone/cortisol response to exposure predicts symptom reduction.

Because our study manipulation was not effective in altering testosterone levels, there remains a possibility that increasing testosterone levels (perhaps via testosterone administration) prior to exposure therapy for SAD could have beneficial effects. The thesis behind this idea is theory-driven and remains plausible, and the research suggesting that exogenous testosterone administration increases threat-related social approach has been replicated in both cognitive and brain imaging studies. It may behoove future researchers to use the brief clinical assay described in Rodebaugh et al. (2013), as we found that a single session generated variable reductions in SAD symptoms and fear (making it possible to examine differential responses to treatment). If this step proves exogenous testosterone administration to be useful (and is replicated), administration within a full dose of exposure-based treatment will be necessary. Including measures of approach (ideally within in the session) would also help to establish increased threat-related social approach as a mechanism of testosterone enhancement of exposure efficacy.

**Directions for Future Research**

Identifying and attempting to manipulate various targets involved in fear extinction may be a fruitful avenue toward improving outcomes for exposure therapy for SAD; however, there are areas for improvement this approach. Importantly, augmentation trials generally involve studying exposure therapy (plus an augmentative strategy) in samples of individuals who have never received exposure-based treatment before. Given that exposure for SAD is largely successful for many individuals (see Hofmann & Smits, 2008), it may be important to apply augmentative strategies only to those individuals who do not respond to treatment. Additionally, it is important to better understand various reasons why

individuals may not respond to exposure therapy, in order to test potential augmentative strategies. Some potential explanations for treatment non-response are individual difference factors (e.g., motivation, emotion regulation skills, expectancies), or failed extinction learning acquisition, retention, or generalization (either due individual deficits in learning or due to suboptimal exposure techniques). Some recommendations toward these ends are as follows:

**Study individual difference variables as predictors of treatment success/failure**

In line with the approach of selectively applying augmentative strategies to treatment non-responders, it will be important to understand potential explanations for poorer treatment outcomes in order to identify appropriate augmentative strategies. Studies examining moderators/mediators of treatment outcome for SAD have yielded some predictors of exposure response, including depression (Chambless, Tran, & Glass, 1997; Craske et al., 2014), treatment expectancies (Chambless et al., 1997; Safren, Heimberg, & Juster, 1997), anger suppression (Erwin, Heimberg, Schneier, &Liebowitz, 2003), homework compliance (Edelmann & Chambless, 1995; Leung & Heimberg, 1996).

Though it is important to continue examining predictors of treatment outcome in this manner, studies are rarely replicated and further studied after initial identification of predictors. Conducting qualitative research (e.g., conducting interviews or collecting written responses) among treatment non-responders (or therapists) may offer further insight into why exposure therapy may not have been effective. Themes derived from this qualitative research could then be tested quantitatively by including valid measures of the constructs of interest in treatment outcome research.

**Study individual differences in extinction learning acquisition, retention, and generalization**

As mentioned previously, several studies have now indicated that DCS administration may only be effective when individuals reach low levels of end fear within the session (Smits et al., 2013a; Smits et al., 2013b; Smits et al., 2014). Researchers have utilized this new understanding to develop a randomized controlled trial to test if DCS is only useful when extinction acquisition has occurred by comparing tailored DCS administration (i.e., administration only after "successful" sessions, or sessions in which participants reach low levels of end fear) to indiscriminate DCS administration (for review of study protocol, see Hofmann, Carpenter, Otto, Rosenfield, Smits, & Pollack, 2015). This line of research highlights how an augmentation strategy (initially designed to target retention) has yielded important information about the importance of acquisition of extinction learning. This supports augmentation of extinction learning mechanisms as a useful strategy for understanding and improving exposure therapy. By refining DCS augmentation to understand for whom and under what circumstances augmentation may be most beneficial, not only is there an opportunity to improve exposure outcomes, but also to better understand fear extinction acquisition.

These findings have direct clinical implications – for example, because translational research has indicated that a low end fear level (i.e., SUDS of 40 or less; Hofmann et al., 2015) may serve as a marker for successful fear extinction acquisition, clinicians can now decide when to end an exposure session based on self-reported fear levels. If fear levels remain high, clinicians may decide to enact pharmacological or

behavioral strategies to enhance acquisition of extinction learning. A recent article details behavioral strategies for enhancing extinction learning in exposure therapy, including increasing expectancies regarding the likelihood or severity of negative outcomes; removing/prohibiting safety signals and behaviors; deepening extinction by combining multiple threat-related stimuli; occasionally reinforcing extinction with the aversive outcome; varying threat-related stimuli, exposure spacing, context, and anxiety levels independent of habituation; affect labeling; and combining the feared stimulus with a novel neutral outcome or a less aversive outcome (Pittig, van den Berg, & Vervliet, 2016). After extinction learning has been successfully acquired, therapists can then enact pharmacological or behavioral strategies designed to increase extinction learning retention. Behavioral strategies for boosting retention of learning are also outlined by Pittig and colleagues (2016), including use of retrieval cues, sleep after exposure therapy, induction of positive affect, physical exercise before exposure, exposure to a novel context, and activation of a consolidated fear memory prior to exposure therapy.

Finally, in addition to learning more about acquisition and retention of fear extinction, it may also be important to study generalization of this learning. In other words, how do we best understand how changes in fear that occur within exposure therapy (extinction learning) generalize to other relevant situations in order to improve the individual's overall quality of life? Some individuals may evidence a decrease in fear response to public speaking-based exposures, but this learning may not "transfer" to other social-threat related fears (e.g., asking someone out on a date, participating in a group meeting). Basic laboratory research indicates that certain factors, including induction of

stress prior to extinction learning (Drexler, Hamacher-Dang, & Wolf, 2017) and inclusion of retrieval cues during exposure to be used in other contexts afterwards (Brooks & Bouton, 1994; Dibbets & Maes, 2011; Vansteenwegen et al., 2006), may enhance generalization of extinction learning. It may also be important to maximize the "relevance" of the exposure to the individual (e.g., someone whose career depends on public speaking may benefit more from public speaking-based exposures). Further, it may also be useful to design exposure exercises using a values-based approach, similar to that of Acceptance and Commitment Therapy (Hayes, Strosahl, & Wilson, 1999), to ensure that fear reduction occurs in an area that the individual considers most important to well being.

## Conclusion

The efficacy of exposure therapy for anxiety disorders is a success story in the history of psychological research. Despite the strides made over the last 60 years of research on behavioral therapy, there is still much to learn as we try to ensure that exposure therapy is maximally effective for a broad array of individuals. This is especially important in light of the fact that, when implemented in mental health settings, empirically supported treatments such as exposure therapy are substantially less effective than in efficacy trials (for review, see Parker & Waller, 2015). These observations underscore the importance of efforts toward enhancing the understanding of exposure therapy mechanisms - how and for whom it works best.

Studying mechanisms is complicated. Because it is often difficult to directly manipulate hypothesized mechanisms (for technical and/or ethical reasons), much of the research in this area remains observational and thus conclusions, although perhaps

consistent with hypotheses, remain open to alternative explanations. Hence, this line of research has high potential to lead investigators on paths that may be ultimately unfruitful. Take for example the investigation of DCS as a augmentation strategy for exposure therapy. The first study applying this agent toward exposure therapy for SAD occurred in 2006 (Hofmann et al., 2006). Despite generating early excitement, more than ten years later we are still trying to understand for whom and under what circumstances DCS augmentation might be most useful, and yielding underwhelming effect sizes (Mataix-Cols et al., 2017). A relevant metaphor for researchers might be to think of the early discoveries regarding the efficacy of exposure therapy for anxiety disorders as the "Big Bang", where massive changes led to the presence of the universe as we know it today. Discoveries in the field of anxiety disorders today more closely resemble the slow, gradual changes of evolution, as existing treatments are refined over time.

This problem may also be exacerbated by the "publish or perish" model of academia, which discourages researchers from attempting innovative, novel perhaps unconventional ideas for fear of null findings (which are likely to go unpublished), or because they are unlikely to be funded. The third study described in this manuscript is a good example of this: the aforementioned research linking testosterone administration with not only psychological and behavioral changes, but also changes in the brain systems governing threat-related social approach, is a theoretically promising next step for augmentation research. Behaviorally manipulating testosterone levels would be a safe, innovative strategy; however, the evidence base for power posing was not strong enough to pursue this project with grant funding. Despite this, the potential benefits of the

knowledge gained from the study were deemed greater than the costs (i.e., time). As is the case with many novel ideas, our attempt to behaviorally manipulate testosterone levels was unsuccessful; however, the project itself provided valuable research skills to the first author (a graduate student); information that adds to the field (regarding the usefulness of a single-session assay and the futility of power posing); and a rich dataset, which can be examined for additional predictors of treatment outcome. Indeed, students may be best positioned to examine "riskier" ideas in the area of exposure therapy research, as they do not yet face the pressures of relying on grant funding.

In sum, the model for manipulating potential mechanisms of fear extinction is likely a useful one for better understanding exposure therapy efficacy for SAD. In next steps, it will be important to consider how to best conceptualize and measure within-session threat-related attention and approach, as well as to consider applying augmentation strategies only to those individuals who do not already benefit from exposure therapy. This line of research will likely be facilitated by collaboration with researchers from other areas (e.g., those who study basic science regarding learning and memory) to fully develop strong theoretical models and innovative ways to test them.

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