COMPARING CHANGE IN NEURAL INDICES OF ATTENTIONAL BIAS AND COGNITIVE CONTROL BETWEEN COGNITIVE BEHAVIORAL THERAPY AND ATTENTION BIAS MODIFICATION FOR SOCIAL ANXIETY DISORDER

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ABSTRACT

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Cognitive models of Social Anxiety Disorder (SAD) state that an imbalance between attention bias (AB) and cognitive control (CC) maintain anxious symptoms. Event-related potentials (ERPs) serve as a precise index to assess these processes. The current study aimed to examine change in the P1 and N2 as indices of AB and CC within Cognitive Behavioral Therapy (CBT) and Attention Bias Modification (ABM) in individuals diagnosed with SAD. There were two primary aims – (1) to examine change in the P1 and N2 to the faces and probe in the dot probe task, and (2) to examine whether changes in ERPs predict symptom change. The sample consisted of 50 adult patients diagnosed with SAD, who were randomly assigned to CBT (n = 33) or ABM (n = 17). For the first aim, multi-level models (MLM) were used to estimate growth curves for each ERP. The results revealed an increase in the N2 to the faces over time for both groups (b = -.38, p = .02) at post-treatment. No other ERP changes reached significance (all p's > .09). For the second aim, individual growth slopes of ERPs and symptoms were correlated with each other, however there were no relationships between ERPs and symptom change (all p's > .40). Lagged MLMs with ERPs as predictors of symptoms demonstrated that a smaller N2 at one assessment related to more avoidance symptoms at the next assessment for the ABM group only (b = -1.23, p = .02). These results signify the need for reliable methods to assess AB, as well as increased specificity of CC processes that are targeted and exercised within treatment contexts

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INTRODUCTION

Social Anxiety Disorder (SAD), characterized by an intense fear of being negatively evaluated in social situations, affects approximately 12% of the general population (Association, 2013; Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Cognitive models of anxiety have implicated attentional bias toward threat and impaired cognitive control as key factors that maintain symptoms of SAD (Clark, 2005; Bar-Haim et al., 2007; Eysenck, Derakshan, Santos, & Calvo, 2007; Mogg & Bradley, 2016). As such, treatment interventions have aimed to target these factors to reduce distress and impairment for individuals with SAD. However, very little research has probed the effects of these treatments on neural mechanisms of attentional bias towards threat and impaired cognitive control. Even less has directly compared such effects across two active treatments. The current study aims to address these gaps in the literature by examining changes in neurophysiological markers of attention toward threat and cognitive control following Cognitive Behavioral Therapy (CBT) and Attention Bias Modification (ABM) for SAD. Secondarily, the current study aims to assess if neurophysiological change within each treatment relates to symptom change.

Attentional bias and cognitive control in SAD

An imbalance between the salience-driven (i.e., attention bias) and goal-directed attention (i.e., cognitive control) systems has been proposed to contribute to the maintenance of anxiety disorders (Eysenck et al., 2007; Mogg & Braddley, 2016). Specifically, anxiety is characterized by early hypervigilance toward threatening stimuli that impairs cognitive control to efficiently complete tasks. As noted by Eysenck et al. (2007), the goal-directed attention system is defined as a variety of functions that support goal-directed behavior. These functions include the ability to inhibit distractors, to integrate information across a range of modalities (both internal and

external), and to recruit more control in the presence of conflict (Eysenck et al., 2007; Miller & Cohen, 2001; Mogg & Braddley, 2017). Anxious individuals tend to experience biased attention to salient stimuli that reduces the ability to use cognitive control. As such, analyzing the disruption of these two cognitive systems and their treatment is vital for an enhanced understanding of the functional mechanisms that maintain anxiety, and their ability to be targeted throughout the course of treatment.

One of the most widely utilized tasks to assess attentional bias within anxiety is the dot probe paradigm (MacLeod, Mathews, & Tata, 1986). In this task, a fixation cross is presented at the center of the screen, followed by the simultaneous presentation of two faces (i.e., threatening and non-threatening) for 500 milliseconds (ms). Next, a probe (e.g., a "dot" or arrow) replaces one of the images, and participants are required to quickly and accurately identify the probe. Attention bias scores are derived by comparing reaction times (RTs) on congruent trials (when the probe replaces the threatening face) with RTs on incongruent trials (when the probe replaces the neutral face). A difference score is calculated by subtracting RTs on congruent trials from incongruent trials, with positive values interpreted as an attentional bias toward threat. It is assumed that individuals have faster RTs on congruent trials because their attention was already allocated toward the threatening image.

Past research has extensively studied attentional biases within anxiety, providing evidence for increased attentional bias to threat (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoor, 2007). Individuals with SAD specifically experience a heightened attentional bias to stimuli that could lead to negative evaluation, such as disgust and contempt faces (Moser, Huppert, Duval, & Simmons, 2008; Mogg, Philippot, & Bradley, 2004; Pyshar et al., 2004) and negative evaluative words (Amir et al., 1996; Amir, Elias, Klumpp, & Przeworski,

2003). Examining the processing of threatening faces can be particularly informative because they represent precisely the set of stimuli that make individuals with SAD anxious. Not surprisingly, in a review of 74 studies examining face processing in social anxiety, Staugaard (2010) found that individuals with SAD showed an early attentional bias toward threatening stimuli, although results become inconsistent for longer stimulus presentations (> 500 ms). For the dot probe task specifically, Bantin, Stevens, Grlach, & Hermann (2016) reviewed 10 studies and found that those with SAD evidenced early attentional biases toward threatening faces. Therefore, it is generally accepted that individuals with SAD show early attentional bias toward threatening faces.

Research also suggests that anxiety is characterized by an inability to disengage from threatening cues (i.e., delayed disengagement) (Fox, Russo, Bowles, & Dutton, 2001; Yiend & Mathews, 2001). That is, once threatening stimuli are attended to, it is difficult for anxious individuals to disengage, and execute goal-directed behavior. This is, indeed, the case for socially anxious individuals specifically. For instance, Amir, Elias, Klumpp, & Przeworski (2003) found that individuals with clinical levels of social anxiety experience delayed disengagement from socially-evaluative words on a spatial cueing task. Those with SAD were slower at responding to probes when presented at a different spatial location than threatening words. Additionally, Moriya and Tanno (2011) found that those with high levels of social anxiety (in comparison to those with low levels) were slower at responding to go trials in a modified version of the Go/No-Go task following centrally presented threatening images. The same effect was not observed for neutral faces, implying that socially anxious individuals experienced difficulty disengaging from threatening faces specifically. They also found that RTs on go trials following threatening images were positively associated with self-reported social

anxiety. Similarly, in an eye-tracking study, Buckner, Maner, and Schmidt (2010) found that those with high social anxiety took more time to disengage fixations from disgust faces than those with low social anxiety. Together, these findings suggest that socially anxious individuals experience impaired cognitive control following the processing of threatening faces. Specifically, once attention is drawn to threatening faces, it is difficult for socially anxious individuals to disengage attention from these task-irrelevant stimuli and employ goal-directed attention to the task-relevant stimuli. However, in one study that implemented the dot probe task to investigate this relationship by Klump and Amir (2009), there was no evidence of delayed disengagement in social anxiety. A potential reason for this finding is that RTs in the dot probe task do not lend themselves to disentangling rapid cognitive processes (i.e., isolating early attention from delayed disengagement). As such, more temporally precise measures are needed to further separate such rapidly unfolding processes.

Ample additional evidence suggests anxious individuals have impaired cognitive control (Eyesenk et al., 2007; Moran et al., 2015; Krugg & Carter 2010). However, findings have been limited for social anxiety, in particular. Amir and Bomyea (2011) found that working memory performance on an operational span task (with threatening and neutral words) was impaired in those with SAD. Specifically, those with SAD were able to easily remember threat-related words (i.e., exhibiting an attentional bias for threatening information), but they were not able to also remember neutral words (i.e., reduced cognitive control). The researchers interpreted this finding as an indication that those with SAD experience an inability to efficiently use cognitive control to inhibit the salience of threatening words and also remember neutral words. Moriya and Tanno (2008) found that higher levels of self-reported fear of negative evaluation (a core feature of SAD), was associated with lower levels of self-reported attentional control on the Effortful

Control Scale (EC; Rothbart, Ahadi, & Evans, 2000). The EC is comprised of three subscales including attentional control, activated control and inhibitory control. Interestingly, attentional control, defined as the ability to inhibit distraction, was the only subscale negatively correlated with social anxiety, while the other subscales were correlated with depression. This relationship was consistent even when controlling for state anxiety and depression, indicating that SAD specifically contributes to attentional control impairments. Consonant with this finding, Weiser, Pauli, and Muhlberger (2009) found that individuals high in social anxiety had more antisaccade errors on trials including facial expressions, regardless of valence, suggesting a broad cognitive control impairment. In addition, in a non-emotional anti-saccade task, Liang (2018) found that those with social anxiety had longer latencies than those with low social anxiety. Liang (2018) concluded that this indicated a general inhibition impairment in individuals with social anxiety, that are not specific to emotional stimuli. Taken together, these findings imply that socially anxious individuals may experience broad cognitive control impairments across emotional and non-emotional stimuli.

Event-related potentials (ERPs) as markers of attention bias and cognitive control

Because attentional bias and cognitive control processes are rapidly occurring functions, using reaction time measures do not allow for a precise measurement of their manifestation. Similarly, many studies have noted the poor reliability of RTs in the dot probe task for assessing bias (Schmukle, 2005; Brown et al., 2014; Macleod et al., 2019). For this reason, attempts have been made to analyze RTs in ways that can examine trial by trial changes throughout the task (Zvielli, Bernstein, and Koster, 2014), however this remains a distal proxy that can only measure downstream processing. Neuroimaging studies have aimed to address this by examining neural activity during the dot probe task (Monk et al., 2006; Telzer et al., 2008; Price et al., 2014),

however they also suffer from an inability to measure the precise time course of such dynamic processes. Event-related potentials (ERPs), on the other hand, serve as an excellent measure of online cognitive processing with millisecond precision. Importantly, because ERPs are temporally precise, it is possible to assess early attentional biases and cognitive control to the presentation of *both* emotional faces and the probe. Doing so will enable me to assess the time course of early attentional mechanisms (bias towards and delayed disengagement from threat) and later cognitive control during emotional face and the task-relevant probe processing. The P1 and N2 are two such ERPs that index attentional and cognitive control mechanisms, respectively, and will be the focus of the present investigation.

The P1 is an early positive deflecting ERP component that peaks between 70 and 150 ms post-stimulus onset. The P1 is a rapidly occurring signal generated from the parieto-occipital visual system (Luck & Kapperman, 2011) and its enlarged amplitude to threatening stimuli in social anxiety can be thought to index early negative attention bias. It has been shown to be sensitive to emotional faces (Batty & Taylor, 2003) and modulated by amygdala damage (Rothstein et al., 2010) such that damage to this region resulted in a reduced P1. Therefore, the P1 is not only a marker for visual salience but is also influenced by the emotional salience of information. Within social anxiety, the P1 is enhanced for emotional faces, (Kolassa & Miltner, 2006; Rossignol et al., 2012), threat-neutral face pairs in the dot probe task (Santesso et al., 2008; Mueller et al., 2009), and positively correlated with social anxiety symptoms (Kolassa & Miltner, 2006). Findings for the P1 to the probe in the dot probe task are limited, however, Mueller et al. (2009) observed that socially anxious individuals demonstrate a smaller P1 to probes replacing threatening faces. This finding is difficult to reconcile with extant studies showing speeded RTs on congruent trials. However, the P1 occurs earlier in time than the

execution of a response. As such, it could be that P1 activity is reduced because attentional resources continue to be devoted to processing the previously presented threatening faces. This supports the notion that socially anxious individuals experience delayed disengagement from threatening stimuli. Therefore, it is also vital to assess changes in the P1 to the probe across treatment.

The N2 is a negative-going signal that peaks approximately 200-350 ms post stimulus onset. It is thought to index recruitment of cognitive control and has been closely linked to inhibition processes in the presence of conflict (Folstein et al., 2008). There is evidence that the N2 is generated from frontal cortical regions implicated in cognitive control, such as the DLPFC and ACC (Grossheinrich et al., 2013; Lavric, Pizzagalli, & Forstmeier, 2004). Although most studies have examined the N2 in Go/No-go and the Flanker paradigms, past studies have also measured the N2 in the dot probe paradigm (Eldar & Bar-Haim, 2012; Thai et al., 2016). For individuals with social anxiety specifically, Thai et al. (2016) found that an attentional bias (measured via RTs) toward threat in socially anxious children was associated with reduced N2 amplitudes to faces in the dot probe task. This is consistent with the notion that biased attention is often coupled with a reduced implementation of cognitive control within anxiety. Therefore, the N2 is a prime candidate for examining changes in cognitive control across treatment for SAD. Additionally, due to the aforementioned research that cognitive control impairments are not specific to emotional faces, it will also be useful to assess cognitive control mechanisms to emotional and non-emotional stimuli in the dot probe task – i.e., faces and probes, respectively.

Treatments for SAD: effects on attention bias and cognitive control

Treatments aimed at relieving SAD symptoms include Cognitive Behavioral Therapy (CBT) and Attention Bias Modification Training (ABM). CBT targets cognitive biases

surrounding social situations (i.e., “They will think I’m dumb”). It also promotes re-entry into social situations that individuals with SAD typically avoid (Huppert et al., 2003; Carpenter, Curtiss & Hoffmann, 2017). Individuals with SAD are asked to complete tasks, such as in vivo exposures (e.g., engaging in a conversation with a confederate) and imaginal exposures (e.g., focusing on the worst possible outcome of social situations) that help with initiating, maintaining and ending social interactions (Huppert, Roth & Foa, 2003; Carpenter, Curtiss, & Hofmann, 2017). Throughout treatment, individuals are instructed to focus on the conversations they are having, and widen their attentional window, instead of focusing on themselves (Huppert et al., 2003) or emotional faces that will confirm their fears.

Generally, these techniques produce significant reductions in both self-reported and clinician assessed symptoms (Heinrichs & Hoffman, 2005; Heimberg, 2002; Stangier, 2016; Huppert et al., in press). There is also evidence that CBT aids in the reduction of attentional bias. In a review, Tobon et al. (2010) found that attentional biases in anxious individuals were reduced across a range of tasks (such as the dot probe and emotional Stoop) after CBT. However, for SAD specifically, findings are limited and mixed. Mattia, Heimberg, and Hope (1993) found that CBT resulted in a reduction of early attentional biases to social threat words using the Stroop paradigm, whereas Lundh and Öst (2001) did not find this effect using the same task. Additionally, neuroimaging studies indicate that amygdala activity is significantly reduced following CBT (Klump et al., 2013; Månsson et al., 2013). To my knowledge, no study has examined P1 changes in the dot probe task throughout the course of CBT for those with SAD.

There is also evidence to suggest that CBT increases engagement of top-down cortical brain regions implicated in cognitive control across a range of anxiety disorders (Bruhle et al., 2014; Porto et al. 2009). Increases in frontal control regions following CBT are attributed to the

practice of reappraisal of negative situations that draw on and exercise frontal control regions. For instance, Goldin et al. (2013) found increased activity in the dorsolateral prefrontal cortex (DLPFC) following CBT when those with SAD were asked to reappraise negative self-beliefs. Similar findings were reported by Goldin et al. (2014), such that CBT increased recruitment of frontal cortical brain regions when SAD patients were asked to reappraise socially evaluative video scenes. No studies have examined changes in the N2, as a marker of cognitive control, in patients with SAD following a course of CBT.

As compared to CBT, ABM is an intervention that specifically targets attentional biases by training attention away from threat (MacLeod, Rutherford, Campbell, Ebsworthy & Holker, 2002). ABM follows the parameters of the dot probe paradigm, with the exception that probes replace neutral faces in most or all of the trials – that is, attention is trained to focus on neutral faces instead of negative faces because that face predicts the location of the target most of the time. One advantage of ABM is that it is easily scalable and requires little effort from the patient to engage in it. ABM has shown positive effects on anxiety symptoms (Amir, Weber, Beard, Bomyea, & Taylor, 2009; Mogoase et al., 2014). However, a recent meta-analysis by Mogg, Waters, Bradley (2017) suggests the effects of ABM are more modest than previously documented. Similarly, results for ABM's effect on attentional biases have been mixed (Heeren, Mogoaşe, McNally, Schmitz, and Philippot, 2015). There is some evidence that ABM reduces attentional biases (Heeren, Lievens, & Philippot, 2011; Schmidt, Richey, Buckner, & Timpano, 2009), but there are also studies that have found no improvements (Bunnell, Beidel, & Mesa, 2013; Julian, Beard, Schmidt, Powers, & Smits, 2012). These mixed findings highlight the need for additional measures of bias change in the dot probe during ABM, such as ERPs. Evidence from past ERP research of ABM for anxiety is limited, however, O'Toole and Dennis (2012)

found a reduced P1 to face pairs in the dot probe task after ABM for trait anxious individuals trained away from threat. To my knowledge, no study has investigated changes in the P1 in the dot probe task after ABM for individuals with SAD.

There is also evidence that ABM improves cognitive control. Support comes from a study conducted by Eldar & Bar-Haim (2010) who found that individuals high in trait anxiety showed enhanced N2 amplitudes to face pairs in the dot probe task after ABM training. Dennis et al., (2017) found increased N2 amplitudes to threatening faces in the dot probe after a mobile delivered version of ABM training. Therefore, there is precedence for enhanced N2 to face pairs following ABM, which suggests that ABM improves the recruitment of cognitive control during emotional face processing. Notably, these studies did not assess changes in the N2 elicited by the probe. Examining the N2 to the probe will allow for an assessment of cognitive control to task-relevant stimuli, which I plan to do in the current study.

Importantly, although prior research points to the effects of CBT and ABM on attention bias and cognitive control, no study has assessed changes in both mechanisms in a comparative context of CBT and ABM for SAD. A recent report using behavioral and symptom data from the same sample to be used for the current study found that attentional biases measured via RTs did not change across CBT and ABM treatments, although there were significant improvement in symptoms (Huppert et al., in press). This null effect on RTs highlights the need for other measures to assess change in attentional biases and cognitive control across treatment, which serves as the central motivation of the present study.

The Present Study

In sum, little research has focused on neurophysiological change across two active treatments for SAD. Thus, the present study has a primary aim of comparing the modulation of

ERPs indexing early attention bias and cognitive control by ABM and CBT treatments. The P1 will be used to assess changes in early attention bias, and the N2 will be used to assess changes in cognitive control. These waveforms will be examined to both the faces and the probe in the dot probe paradigm to disentangle cognitive processing dynamics over the course of a trial. Using ERPs in this way will allow for an investigation of both changes in early attentional allocation to and cognitive control during emotional face processing as well as changes in the processing of task-relevant stimuli (i.e., the probe). Furthermore, this study has a secondary goal of examining whether neurophysiological change, as measured by the P1 and N2, relate to symptom change. To my knowledge, no extant studies have attempted to link ERP changes in attentional bias and cognitive control with symptom improvement in two active treatment for SAD.

There are five hypotheses to address the first aim. During face processing, I predict that (1) the P1 amplitude will decrease over treatment, indexing less attentional bias to the faces, and (2) the N2 amplitude will increase over treatment, indexing more cognitive control during face processing. For the probe, I predict that (3) the P1 amplitude will increase on congruent trials over treatment indicating enhanced attention to the probe (i.e., disengagement from threatening faces), and that (4) the N2 amplitude will increase over treatment, indicating more goal-directed cognitive control to the probes. Lastly, I predict that (5) these ERP changes will be strongest in the ABM group. I predict that ABM will show enhanced effects for two reasons. Conceptually, ABM specifically targets cognitive mechanisms in the dot probe task whereas CBT does not, thus effects on neurophysiological indices of cognition elicited in the dot probe task should be larger for ABM. Second, there is more empirical data to support that these specific neurophysiological changes will be evident in the dot probe following ABM, and no such

supportive data for CBT. With regard to the second aim, there are two hypotheses. I predict that (1) decreased P1 and increased N2 activity to faces will relate to symptom reduction. I further predict that (2) increased P1 and N2 activity to the probes will relate to symptom improvement.

METHOD

Participants

Descriptive statistics for self-reported age, gender, and handedness are reported in Table 1. Participants were recruited from advertisements or referrals from doctors and clinics in Israel. Criteria included participants who were 18 years or older and met eligibility after screening for social anxiety. Social anxiety was assessed using the Mini International Neuropsychiatric Interview (MINI; Sheehan, 2006) and the Lebowitz Social Anxiety Scale (i.e., scores >50) (LSAS; Liebowitz, 1987). Exclusionary criteria included any history of psychosis, bipolar disorder, suicidality, or current substance abuse. The final sample included a total of 50 people assigned to either CBT (n = 33) or ABM (n = 17) (see Figure 1).

Measures

All measures were translated from English to Hebrew and back-translated by another individual for validity.

Leibowitz Social Anxiety Scale (LSAS; Leibowitz, 1987). The LSAS is a semi-structured interview to screen participants for social anxiety symptom severity. It assesses for anxiety and avoidance in social and performance situations. It consists of 24 items that participants rate for level of fear from 0 (*Never*) to 3 (*Severe*), and avoidance from 0 (*Never*) to 3 (*Usually*). A global sum score (across both fear and avoidance) was calculated to assess symptom severity for those with social anxiety. Higher global scores indicate more severe social anxiety. The interviewers were doctoral students in clinical psychology who administered the LSAS in four-week intervals as part of patient's monthly assessment. They also completed the LSAS at the end of treatment, and at a three-month follow up. They were blind to experimental conditions and had no other interactions with the participants. Inter-rater reliability was

conducted by videotaping 15 evaluations and a second person rated the LSAS while watching. Inter-rater reliability was high (r = .94, p <.01). Another reliability test was conducted by randomly selecting 15 evaluations to be administered a second time within the first week of intake assessment, which also produced high reliability (r = .80, p <.01).

Social Phobia Inventory (SPIN; Conner et al., 2000). The SPIN is a 17-item questionnaire that asks participants to rate their level of fear, avoidance, and arousal in the past week. The SPIN was shown to have reliable psychometric properties in clinical populations (Connor et al., 2000). Participants are asked to respond to items using a 5-point Likert scale ranging from 0 (*Not at all*) to 4 (*Extremely*). All scores are summed to create a total score for the questionnaire, with higher scores indicating more social anxiety. Participants completed the SPIN in their initial assessment, and also completed it as part of their weekly assessments to track for self-reported symptom change throughout treatment. The SPIN was also given at the end of treatment, as well as the three-month follow-up.

Dot Probe Task. In this task, trials begin with the presentation of a fixation point at the center of the screen for 500 ms. Then, two images of facial expressions were presented vertically one above and one below the fixation cross. All trials included one neutral and one threatening (i.e., angry, disgust) facial expression. Both images terminated after 500 ms and one of the images was replaced by an arrow (probe). Participants were required to respond to the direction of the arrow with their dominant hand on a response box.

A total of 64 facial expression images were selected from NIMSTIM (Tottenham, Borscheid, Ellertsen, Marcus, & Nelson, 2002) and TAU (Frenkel & Bar-Haim, 2006) databases, with threatening images including anger, disgust or contempt. The task consisted of 256 trials in which all threat-neutral pairs were presented four times, with probes replacing the threatening

face 50% of the time. Therefore, half of the trials were congruent (probe replaced a threatening face), and half were incongruent (probe replaced a neutral face). This task was administered at the first assessment and every four sessions during treatment. Participants also completed this task at the end of treatment and three-month follow up.

Electroencephalography (EEG). Continuous EEG activity was recorded from 64 Ag-AgCl electrodes fitting into a BioSemi (BioSemi, Amsterdam, The Netherlands) stretch-Lyrca cap while participants completed the dot probe task. The cap consists of electrodes placed across the scalp to cover locations across the cerebral cortex, including central, frontal, parietal, temporal, and occipital lobes. The cap size was chosen by measuring the circumference of the head. Once the appropriately fitted cap was placed on the head, measurements were taken to ensure that electrode channels were placed over the appropriate region of the brain. These measurements were taken from the nasion (a prominent bump on the front of the head, usually between the eyebrows) to the inion (a prominent bump in the back of the head indicating the end of the skull). Additional measurements were taken from the top of the left ear to the right ear. The cap was fitted with a Velcro chinstrap to ensure its stability during data acquisition.

Two additional electrodes were placed on the right and left mastoid bones, a relatively prominent bone behind the ear, to serve as a reference. Electro-oculogram (EOG) activity generated by eye movements and blinks was recorded at FP1 and by electrodes placed above and below the left eye, and on the right and left of the outer canthi (the outer corner of the eye). During data acquisition, scalp recordings were referenced online to two electrodes called the common mode sense (CMS) active electrode and the driven right leg (DRL) passive electrode. CMS serves as a ground electrode and DRL is part of a feedback loop that drives the participant's potential current close to zero. Further analyses were conducted offline.

EEG data was processed offline using Brain Vision Analyzer 2 (Brain Products, Gilching, Germany). The recordings were re-referenced to the numeric mean of the right and left mastoid bones, such that the activity recorded at the mastoid bones was subtracted from the activity recorded from scalp electrodes to isolate activity on the scalp. Next, activity on the scalp was band-pass filtered to include activity in the frequency band between 0.1 – 30 Hz (12 dB/oct rolloff). Blinks and eye movements were corrected using the Gratton, Coles, & Donchin (1983) method. This method corrects for the confounding effects of eye movements and blinks using a regression-based method. This is achieved by averaging across EOG and scalp recorded activity and estimating propagation factors by regressing average EOG activity onto scalp activity and correcting EEG data based on the estimated propagation factors.

Trials where errors were committed were removed, and all correct trials were segmented into epochs starting 100 ms pre-stimulus onset and continued for 600 ms post-stimulus onset. This was completed twice: for the onset of the faces and the onset of the probes. Trials were rejected based on the following criteria: voltage step between two contiguous data points exceeding of 50 microvolts (uV), a voltage difference of more than 200 uV within a trial, or a maximum voltage difference less than .5 millivolts within a trial. All of these occurrences do not reflect natural occurring neurophysiological activity but rather poor connection to the scalp, sudden head movements and, and other related artifacts.

Upon visual inspection, the P1 was extracted in the 80-150 ms time window. The sites were pooled at parietal occipital sites on the left (O1, P3, P5, P7, PO3, PO7) and right (O2, P4, P6,P8, PO4, PO8) of the head. Because the faces are vertically and centrally presented, I did not hypothesize that the average activity in the P1 would differ as a function of threatening face location. Therefore, the activity across the specified time window was averaged across face

location (top and bottom) and congruency (probe replacing threatening and neutral faces). Visual inspection revealed a clear difference in activity on the left and right of the head, however. Because of this, an initial model was tested to examine if lateralized location (left vs. right of the head) interacted with time or group. The model revealed a main effect of lateralized location, such that the right side of the head was more positive than the left (b = .24, t(550) = 2.199, p = .03) (see Figure 2 and Results below)[1]. However, this did not interact with time, or group nor was there a three-way interaction (all p's >.11). Therefore, the final model averaged across the left and right of the head. Next, the N2 was identified as the average activity in the 200-350 ms time window at site Fz.

Additionally, models for site specification to the probe were first tested with a fixed effect for time, congruency, and group. This was done to examine whether congruency interacted with any additional terms. There was no effect of congruency nor did congruency interact with time or group (all p's > .75). Therefore, the average activity time-locked to probe presentation were averaged across location and congruency. Visual inspection for the probe-locked P1 revealed there was no early positive activity to the probe, contrary to my hypothesis. However, there was bilateral negative activity at parietal-occipital sites, indicating there was an N1. Therefore, the average activity between 70 and 140 ms was pooled across lateralized sites on the left (P7, PO7, O1, PO3, P5, P3, P1) and right of the head (P2, P4, PO4, P6, PO8, O2, P8) [2]. Upon visual inspection, the N2 was specified as the average activity between the 215-350 time window at site Fz.

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[1] A model was tested with an additional interaction terms for lateralized effect, including location X Group, location X time, and location X follow-up. These did not reach significance (all p's = .32), indicating that it also did not interact with follow-up.

[2] To remain consistent with the face-locked analyses, a lateralized effect was tested. The model revealed small estimates for time (b = -2.449 e-17) and time X lateralized location (b = 7.6883 e-18), causing a large p-value (p =1). Therefore, this effect was excluded from the final model.

Treatments

Cognitive Behavioral Therapy. Patients underwent up to 20 sessions of CBT on a weekly basis. Sessions lasted approximately 60-90 minutes per week. CBT was delivered by graduate students under the supervision of Dr. Jonathan Huppert, a doctoral-level, licensed expert clinician. Therapy followed the protocols of Huppert, Roth Ledley, and Foa (2006) and of Clark (2005). Its main components included the manipulation of self-focused attention and safety behaviors, strategies that those with SAD perform when confronted with anxious situations (e.g., avoiding eye contact). For manipulation of self-focused attention, patients practiced shifting attentional focus between internal stimuli (self-focused thoughts, feelings, images) and external stimuli (individuals with whom one interacts, facial expression of others, colors and shapes of buildings, etc.). Patients are instructed to resist safety behaviors.

A main component of therapy was exposure exercises (i.e., behavioral experiments) in which participants were instructed to engage in fearful social situations – this was done in session and out of session for homework. The treatment also incorporated video and audio feedback of the patients during their exposures exercises, enabling them to see themselves and an observer, and the interaction as a whole. Building on treatment techniques by Huppert et al. (2003), treatment also incorporated developing an idiosyncratic model linking negative beliefs about social interactions to internal attentional focus, safety behaviors, physiological symptoms and overt avoidance of social situations. Imaginal exposures to social situations and social skills training were also included. There was no formal coding of the adherence to therapy.

Attention Bias Modification (ABM). Up to eight sessions of ABM was delivered on a weekly basis. Sessions lasted approximately 10-15 minutes per week. The ABM procedure followed a modified dot probe protocol. A fixation cross was presented on the screen for 60 ms

followed by the presentation of threat-neutral face pairs at the center of the screen for 500 ms. To train attention away from threat, the probe replaced the neutral face on 80% of the trials.

Procedure

Participants were screened in person for clinical disorders and SAD using the MINI, LSAS, and a reaction time task[3]. Following this, all participants completed an assessment consisting of the dot probe task, the SPIN and the LSAS. Then, participants were randomly assigned to either the CBT or ABM condition and completed their respective treatment. Four-week assessments were administered in which participants completed the dot probe task, SPIN, and LSAS. Participants also completed an assessment at the end of treatment (up to 8 weeks for AMB and up to 20 weeks for CBT), and three-month follow-up (see Figure 2).

Data Analytic Approach

The analyses were conducted using the "lme4" (Bates, Maechler, Bolker, & Walker, 2015) and "nlme" (Pinheiro, Bates, DebRoy, Sarkar, & R Core Team, 2016) packages in R Version 3.5.1**.** The first aim was analyzed using multi-level models (MLMs). The analyses took an intent-to-treat approach, with all subjects having at least one observation after the pre-treatment assessment, as MLM can estimate effects with missing observations. For the first aim, I conducted a series of growth models for each ERP. Before conducting each model, I computed an interclass correlation (ICC), which computes the amount of variability contained between people in comparison to the total variability. Higher ICC's indicate greater variability between people, which provides support for a random intercept model. All models were fit using

[3] [1]Participants also had to meet a threshold of 75% accuracy on a Grammatical Decision Task (GDT) to be eligible to participate.

restricted maximum likelihood estimation. Model fit was assessed by computing the proportion of variance explained at level 1 (i.e., time) and level 2 (group) between the unconditional and random slope models. For all models, group (effect coded) was entered as a cross-level interaction with time and follow-up to examine if change in ERPs differed between treatment groups. Time was centered with the pre-treatment assessment as baseline, with a one unit increase in time being equivalent to the passing of approximately one month. Two models were estimated to examine rates of change – (1) at the 8-week mark (i.e., post-treatment for the ABM group) and (2) at post-treatment for both groups (i.e., up to 20 weeks for the CBT group). For the first model, only observations up until the 8-week mark were included. This resulted in the following model -

Level 1 Model:

$$ERP\ Amplitude\ _{ij} = \beta_{0j} + \beta_{1j}(Time_{ij}^{8-week}) + r_{ij}$$

Level 2 Model:

$$\beta_{0j} = \gamma_{00} + \gamma_{01}(Group_j) + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}(Group_j) + u_{1j}$$

Combined Model:

$$ERP\ Amplitude\ _{ij}$$

$$= \gamma_{00} + \gamma_{01}(Group_j) + \gamma_{10}(Time_{ij}^{8-week}) + \gamma_{11}(Group_j * Time_{ij}^{8-week}) + u_{0j}$$

$$+ u_{1j}(Time_{ij}^{8-week}) + r_{ij}$$

For the second model (estimating the rates of change at the post-treatment point for both groups), all observations were used with a random intercept and slope. Included in these analyses (i.e.,

those examining post-treatment change) was a test of follow-up (dummy coded), which was done by fitting a piecewise model. This allowed for the examination of change from post-treatment to follow-up. This resulted in the following model –

Level 1 Model:

$$ERP\ Amplitude_{ij} = \beta_{0j} + \beta_{1j}(Time_{ij}^{Post-Treatment}) + \beta_{2j}(Follow-up) + r_{ij}$$

Level 2 Model:

$$\beta_{0j} = \gamma_{00} + \gamma_{01}(Group_j) + u_{0j}$$

$$\beta_{1j} = \gamma_{10} + \gamma_{11}(Group_j) + u_{1j}$$

$$\beta_{2j} = \gamma_{20} + \gamma_{21}(Group_j)$$

Combined Model:

$$ERP\ Amplitude_{ij}$$

$$= \gamma_{00} + \gamma_{01}(Group_j) + \gamma_{10}(Time_{ij}^{Post-Treatment)} + \gamma_{11}(Group_j * Time_{ij}^{Post-Treatment}) + \gamma_{20}(Follow-up) + \gamma_{21}(Group_j * Follow-up) + u_{0j} + u_{1j}(Time_{ij}^{Post-Treatment}) + r_{ij}$$

In sum, all models had three primary effects of interest – a time effect, which would indicate a monthly ERP change rate over time; a group effect which would indicate different levels of average activity between groups averaged across time, and a time X group cross-level interaction, which would indicate different rates or direction of change between groups. Lastly, in models analyzing post-treatment change, it was of interest to examine if there were differences in ERPs at the follow-up assessment, and whether these effects differed by treatment group.

Next, two approaches were taken to evaluate the second aim – whether changes in ERPs relate to symptom change. The first approach involved conducting separate growth models (until

post-treatment) predicting ERPs (P1/N2 to the faces and P1/N2 to the faces) and symptoms (LSAS and SPIN scores) with a random intercept and slope for time using restricted maximum likelihood estimation. Then, each participant's slopes were extracted from these models, and bivariate correlations were conducted to examine if changes in ERPs related to changes in symptoms. This set of analyses allowed for the investigation of the relationship between the rates of change in ERPs and symptoms throughout the course of treatment. The second approach involved computing multilevel models with lagged pre-treatment centered ERP values as the predictor of symptoms. In these models, I included an autoregressive covariance structure for time, simply meaning that the covariance structure controlled for the fact that ERP values closer together in time are more correlated than values further apart. These models allowed for a more fine-gained analyses of ERP and symptom relationships, in that they examined whether the ERP amplitude measured at one time-point predicted symptom levels at the following time-point. These models also included a group effect (effect coded) to examine if changes in symptoms differed by group, as well as a group by ERP interaction to assess if the lagged relationship between ERPs and symptoms differed by group. For these models, model fit was assessed by comparing the amount of variance contained in the final model and the amount of variance in the dependent variable (obtained in the unconditional model with no predictors).

RESULTS

Face-locked Growth Models

I first examined the rate of change at the 8-week mark of treatment for the P1 to the faces. The unconditional model revealed an ICC of 65%. The final model included a random intercept and slope for time and revealed that there was no effect of time (b = .02, t(179) = .205, p = .84), group (b = -.46, t(179) = -1.52, p = .14), or group X time interaction (b = -.07, t(179) = -.75, p = .46).

The second model estimated the rate of change of the P1 to the faces until the post-treatment assessment for both groups. The unconditional model revealed an ICC of 60%. The final model revealed no main effect of time (b = -.05, t(275) = -.533, p = .60), group (b = -.07, t(275) = -.162, p = .87), or group X time interaction (b = -.13, t(275) = -1.318, p = .19). The results also showed there was no follow-up effect (b = -.17, t(275) = -1.32, p = .19), or group X follow-up interaction (b = -.34, t(275) = 1.398, p = .16). The results imply that treatment did not affect early attentional processing to threatening faces, nor did this differ by groups. They also imply that the early attentional processing to threatening faces, as measured by the task, remained unchanged even when treatment ceased.

Next, the N2 to the presentation of faces was examined at the 8-week mark. The unconditional model revealed an ICC of 64%. The final model included a random intercept and slope for time. The model revealed a main effect of time, such that the N2 increased across both treatments, (b=-.41, t(179)= -2.605, p=.01). There was no group effect (b = -.22, t(179)= -.508, p=.61), nor was there a group X time interaction (b=-.10, t(179)= -.63, p=.53) (see Table 2). This model revealed that proportion of variance explained at level 1 (i.e., time) for this model was 11% more than the unconditional model.

The unconditional model for the N2 to the faces with all observations revealed an ICC of 52%. The effect of time remained, such that the N2 increased over time for both groups (b = -.38, t(275)= -2.3, p=.02) (see Figure 3). This was not qualified by a group X time interaction (b=-.11, t(275)= -.67, p = .50) nor a group effect (b=-.22, t(275)= -.475, p = .63). There was no change at follow-up (b= .70 t(275)= .81, p = .42) nor a group X follow-up interaction (b=.32, t(275)= -.365, p = .72), indicating that changes in the N2 were sustained at follow-up for both groups (see Figure 4 and Table 3). This model revealed that 12.4% of the variance was explained with time (in comparison to the unconditional model). Together, the ERPs time-locked to face presentation evidenced no change in the P1. However, there was a significant increase in the N2 over time, seen both at the 8-week mark and at the end of treatment for both groups.

Probe-locked Growth Models

First, the effect of time on the N1 was examined at the 8-week mark. The unconditional model revealed there was 23% of variability between groups. The results of the final model revealed that there was no effect of time (b=-.16, t(164) = -1.26, p = .21), group (b =.41, t(164)= 1.58, p = .11), nor an interaction (b=-.11, t(164)= -.84, p = .41). For the model with all observations used, the ICC was 28%. This model showed there was no effect of time (b=.09, t(247)= .86, p = .39). There was a marginal effect of group (b=-.46, t(247)= 1.85, p = .07), such that the ABM group had a larger N1 than the CBT group. This implies that overall, the ABM group evidenced more attentional capture to the probe. This effect was not qualified by a group X time interaction (b = -.17, t(247)= -1.55, p = .12). There was also no effect of follow-up, nor a group by follow-up interaction (all p's > .12).

At the 8-week mark for the N2, the ICC for the model revealed was 28%. The results showed there was no effect of time (b = -.27, t(175) = -1.12, p = .26), group (b = -.14, t(175) = -

.32, p = .75), or group X time interaction (b = -.01, t(175) = -.09, p = .93). The final model including all observations revealed a marginal effect of time, such that the N2 increased over time (b = -.33, t(271) = -1.709, p = .09). There was no effect of group (b = -.14, t(271) = -.32, p = .75), or time X group interaction (b = -.05, t(271) = -.23, p = .82). There was also no effect of follow-up (b = 1.40, t(271) = 1.41, p = .15), nor was there a follow-up X group interaction (b = -.1, t(271) = -.10, p = .92).

In sum, there was no change in the N1 to the probe over time, however the ABM group evidenced more attentional capture (i.e., a larger N1) to the probes overall. Lastly, there was a marginal change in the N2 over time for both treatment groups, such that the N2 increased to probe presentation.

Relationship between ERP change and symptom change

First, individual growth models were computed for each ERP and for symptoms. These models only included the post-treatment mark, with a random intercept and slope for time (see Tables 4-11). This allowed for the extraction of the slopes for each individual, which were used for bivariate correlations. There were no relationships between ERPs and symptoms (all p's > .40) (see Table 12).

However, there were relationships among ERPs and among symptoms[4]. The P1 evidenced a positive relationship with the N2 to the faces (r(47) = .32, p < .02) and the N1 to the probe (r(45) = .30, p = .04) (see Figure 6-7). These results imply that the higher the rate of change in early attention processing to the presentation of faces, the smaller the rate of change in cognitive control to the faces and attentional capture to the probe, respectively. The N2 to the faces evidenced a marginal positive relationship to the N1 to the probe (r(45) = .26, p < .08).

[4] An outlier was identified for these sets of analyses. Upon removal, previously significant relationships were no longer reliable. Therefore, the results reported here exclude one participant.

This implies that the smaller the rate of change of cognitive control to the faces, the smaller the attentional capture to the probe. There were also high positive relationships between symptom changes (all *p*'s < .001) suggesting that the decrease in symptoms over time related to one another across all measures (see Table 12).

Multilevel Models with Lagged Pre-Treatment Centered ERPs to Face Presentation as Predictors

Next, the lagged models were conducted to obtain a more fine-grained analyses of the relationships between the ERPs and symptoms. For the P1 to the face, the models showed there was no effect of the P1 on LSAS total scores (*b* = -.20, *t*(182) = -.26, *p* = .79), nor was there a group effect (*b* = -2.86, *t*(182) = -.88, *p* = .38), or group X P1 interaction (*b* = -.87, *t*(182) = -1.13, *p* = .26). Similar effects were found with LSAS Anxiety (all *p*'s > .22) and avoidance subscale scores (all *p*'s > .33). For the SPIN, there was also no effect of the P1 to face presentation, (*b* = .31, *t*(182) = .63, *p* = .53); however, there was a marginal group effect (*b* = -3.28, *t*(182) = -1.9, *p* = .06), such that the CBT group had lower estimates (*b* = 32.66) than the ABM group (*b* = 39.16). There was no group X P1 interaction (*b* = -.24, *t*(182) = -.48, *p* = .64). Therefore, there was no relationship between early attention to face presentation and symptom measures.

For the N2 to face presentation, the N2 did not predict LSAS total scores (*b* = -53, *t*(182) = -.95, *p* = .35) nor was there a group effect (*b* = -2.76, *t*(182) = -.84, *p* = .40). There was, however, a group X N2 interaction (*b* = 1.17, *t*(182) = 2.07, *p* = .04). Simple slopes analyses showed that there was a difference in the direction of the slopes, but neither of the slopes reached significance for the ABM (*b* = -1.71, *t*(182) = -1.64, *p* = .10) or CBT group (*b* = .64, *t*(182) = 1.46, *p* = .15). Investigation of the anxiety and avoidance subscale scores on the LSAS

illuminated these findings, however. The results revealed that the above effect is mostly influenced by avoidance scores. Specifically, the final model showed there was no effect of N2 (b = -.44, t(182) = -1.45, p = .15), nor was there a group effect (b = -1.36, t(182) = -.75, p = .46), but there was a significant N2 X group interaction (b = .78, t(182) = 2.56, p = .01). This model explained 2.1% additional variance over the unconditional model. Simple slope analyses revealed that the ABM group evidenced a negative relationship between N2 and LSAS Avoidance (b = -1.23, t(182) = -2.17, p = .02), such that a smaller N2 (i.e., less negative) predicted less symptoms at the next time-point. There was no relationship for the CBT group (b = .34, t(182) = 1.45, p = .15) (see Figure 8). There was no effect of the N2 on LSAS Anxiety scores, nor was there a group effect or group X N2 interaction (all p's > .18).

Additionally, relationships between ERPs and SPIN scores also showed effects that marginally differed by treatment condition. There was no effect of the N2 (b = .15, t(182) = .44, p = .66). There was a marginal group effect (b = -3.11, t(182) = -1.90, p = .06), such that the CBT group had lower estimated scores (b = 32.75) than the ABM group (b = 40.06). However, there was also a marginal group X time interaction (b = .58, t(182) = 1.71, p = .08). Simple slope analyses showed that there was a positive relationship between the N2 and SPIN scores for the CBT group (b = .72, t(182) = 2.61, p = .01), but no relationship for the ABM group (b = -.43, t(182) =-.69, p = .49) (see Figure 9). That is, a larger N2 at one time-point predicted lower symptoms at the next time-point. This model explained 17.24% additional variance over the unconditional model. Overall, these results imply that there was no relationship between the P1 and symptoms and that this did not differ by treatment group. However there was the relationship between the N2 and LSAS Avoidance for the ABM group only, such that a smaller (i.e., less negative N2) predicted higher symptoms throughout the course of treatment. On the other hand

there was a marginal difference between the N2 and SPIN, such that a larger N2 predicted less symptoms for the CBT group only.

Taken together, the results of the lagged models revealed no relationship between the P1 time-locked to face presentation and symptoms, suggesting early attentional bias to threatening faces were not related to symptoms. However, there was a significant relationship between the N2 time-locked to the faces that differed by treatment group. Specifically, a smaller N2 at one time-point predicted lower LSAS Avoidance symptoms at the next time-point for the ABM group only. On the other hand, the relationship between the N2 and symptoms in SPIN scores marginally differed between groups, such that a larger N2 at one time-point predicted less symptoms at the following time-point for the CBT group.

Multilevel Models with Lagged Pre-Treatment Centered ERPs to Probe Presentation as Predictors

The N1 to the probe showed no effect on LSAS total scores (b = -1.17, t(171) = -1.18, p = .24), no group effect (b = -2.13, t(171) =-.65, p = .52), and no group X N1 interaction (b = 1.31, t(171) =1.32, p = .19). Similar effects were found for LSAS Anxiety scores (all p's > .42). LSAS Avoidance scores showed a slightly different pattern, however. Although there was no effect of the N1 (b = -.81, t(171) =-1.52, p = .13) or group (b = -.87, t(171) =-.49, p = .62), there was a marginal group X N1 interaction (b = .94, t(171) =1.77, p = .08). The follow-up with simple slopes revealed that none of these results reached significance, but both groups showed opposite relationships with LSAS Avoidance scores. Specifically, the CBT group showed no relationship (b = -.13, t(171) =-49, p = .63), but a marginal negative relationship for the ABM group (b = -1.75, t(171) =-1.7, p = .09), suggesting that a smaller N1 (i.e., less attentional capture to the probe) predicted lower symptoms. There was no relationship between the N1 to the probe and

SPIN scores (b = -.20, t(171) =.33, p = .74), nor was there a group X N1 interaction (b = -.009, t(171) =-.01, p = .99). Consistent with the above models, there was a group effect (b = -3.46, t(171) =-2.02, p = .04), with lower estimates for the CBT group (b = 32.54) than the ABM group (b = 39.45).

For the N2 to the probe, there was no effect of the N2 on LSAS total (b = -.57, t(179) = -1.01, p = .32), no group effect (b = -2.68, t(179) = -.83, p = .41) or group X N2 interaction (b = -.28, t(179) = -.51, p = .61). Similar effects were found with LSAS Anxiety (all p's > .30) and Avoidance (all p's > .39) scores. There was also no effect of the N2 to the probe on SPIN scores (b = .10, t(179) = .27, p = .78), a marginal group effect (b = -3.28, t(179) = -1.96, p = .05), and no (b = .25, t(179) = .69, p = .49). The group effect remained consistent, in that there were lower estimates for CBT (b = 32.56) than ABM (b = 39.47).

All in all, there was a marginal effect for the N1 for the ABM group, suggesting that less attentional capture to the probes was related to lower symptoms. Lastly, there was no relationship between the N2 to the probe and symptom measures, suggesting that cognitive control to the probe was not related to symptom change across both treatment groups.

DISCUSSION

Cognitive models of anxiety have indicated that individuals with social anxiety experience enhanced attention bias toward threatening stimuli, such as faces, which dampens the ability to exercise top-down cognitive control. There is evidence that CBT and ABM can aid in reducing attentional bias and increasing cognitive control, however, it remains unclear if there are differences between each treatment to effectively do so. In addition, it remains unclear if indices of these cognitive processes within each treatment differentially relate to symptom reduction. I found that the N2 to the faces significantly increased across both CBT and ABM, while there were no changes in the P1 to the faces or the N1 and N2 to the probe. Additionally, the rate of change of these ERPs did not relate to the rate of change in symptoms reported in the LSAS or SPIN. Lastly, we found that N2 scores to the faces were negatively related to LSAS Avoidance symptoms for the ABM group only, such that the smaller the N2 at one time-point, the higher the symptoms at the next time-point. On the other hand, there was a marginal group difference for the N2 and SPIN scores, such that a larger N2 predicted less symptoms for the CBT group.

Overall, results from the current study provided mixed support for hypotheses. Inconsistent with my predictions, results showed that there was no change in the P1 amplitude to face presentation, indicating no change in attentional capture to faces across both treatments. The literature for the modulation of attentional bias for both CBT and ABM are limited and mixed (Mattia, Heimberg, & Hope, 1993; Lundh & Öst, 2001). In addition, in a report using the data from this study, there was no change in behavioral metrics of attentional bias – both the traditional calculation and trial level variability – in either group (Huppert et al., in press). There seem to be two possible explanations for these findings. First, because attention biases are fast

and automatic, they may just be difficult to change. Second, issues of reliability of early attention biases have been raised (Schmukle, 2005; Brown et al., 2014; Macleod et al., 2019). That is, it may be difficult to detect change in an unreliable metric. To counter this, the argument has been made that attentional bias may be better conceptualized as a probabilistic phenomenon that pre-disposes anxious individuals to be more likely to have an attentional bias toward threat. However, the stability and strength of the bias depends heavily on the contextual factors that exacerbate it (MacLeod et al., 2019). This conceptualization would continue to mean that its measurement is unreliable (unless contexts are stable at every assessment). Therefore, perhaps the processes exercised in CBT and ABM are not modulating these dynamic and context-dependent changes in early attentional processes, or at the very least, the change in early attention elicited by these treatments is quite difficult to capture.

On the other hand, the N2 time-locked to face presentation increased as a function of both treatments. This signifies that both treatments exercised the engagement of frontal brain processes associated with cognitive control. In addition, the N2 increased at the 8-week mark for both groups (which is approximately half way through treatment for the CBT group), suggesting that the engagement of cognitive control processes occurs relatively early in treatment. These results are aligned with other studies that have found that the N2 increases as a function of ABM (Eldar & Bar-Haim, 2010), and CBT increases the engagement of frontal brain regions (i.e., the DLPFC) associated with cognitive control function (Goldin et al., 2014). As such, it can be concluded that the engagement of these top-down processes in the presence of emotional stimuli may be a useful mechanism of change to target and measure throughout the course of treatment.

Additionally, contrary to my hypothesis, these data did not evidence a P1 to the probe but did evidence an N1. The N1 time-locked to targets in the dot probe task has been interpreted to

index facilitated attention (Torrence & Troup, 2016; Zhang et al., 2016), and its interpretation is similar to that of the P1 waveform. There was no change in the N1 to the probe over time, suggesting that neither group produced change in delayed disengagement. Similarly, Klump and Amir (2009) also did not find evidence for delayed disengagement using this task. These results further speak to the difficulty to measure delayed disengagement in the dot probe task. However, the ABM group evidenced a larger N1 overall. This could be due the repeated exposure that the ABM group had to the task parameters, both for assessment and treatment, that resulted in more facilitated attention to the probe.

Partially supportive of my hypothesis was a marginally significant increase in the N2 over time to probe presentation. This implies that there was increased cognitive control to mitigate the interference of the salient face distractors. With the exception of a few studies (Mueller et al., 2009, Zhang et al., 2016), the majority of the literature on the dot probe task has not examined change in ERPs to the probe, and no studies have examined change in the N2. The results also speak to the need for increased measurement of the N2 to probe presentation. Doing this will allow for an examination of the ability to not only recruit cognitive control during emotionally salient information, but to also do so for a task-relevant goal (allowing for the ability to observe if treatment also helps facilitate increased ability to complete task demands). This may provide a useful way for to examine how both treatments improve sustained goal maintenance, which is often impaired in individuals with anxiety.

In sum, the change in ERPs produced by CBT and ABM broadly imply that both treatments serve to primarily exercise rapid processes influenced by frontal-cortical regions of the brain in the presence of both emotional and non-emotional stimuli. The fact that the ABM group – the treatment aimed to specifically target attentional bias – did not produce change in

attentional bias, could further imply that such early attentional processes are difficult to target, and therefore difficult to change. However, it may be more apt to identify CBT and ABM as treatments that increase the recruitment of frontal-cortical regions of the brain during the dot probe task for those diagnosed with SAD.

Furthermore, the second aim sought to examine if changes in these ERPs were related to symptom change. Contrary to my hypothesis, although neither of the ERP change slopes were related to symptom change slopes, ERP changes were related to one another, and symptom changes were related to one another. This implies that treatment produced change at a similar rate among ERPs and symptoms, but the rate of change in the neurophysiological index did not relate to the rate of change in reported symptoms of anxiety. These results speak to a broader discourse of the differential change seen in the brain and reported symptoms in individuals diagnosed with SAD. More research is needed to further elucidate these findings, such as examining if changes in ERPs are specific to the dot probe.

Results of lagged models, however, showed that changes in the N2 had differential effects on symptoms across groups. Specifically, for the CBT group, changes in the N2 were unrelated to LSAS symptoms. On the other hand, for the ABM group, a larger N2 predicted higher LSAS Avoidance symptoms at the next time-point, but no relation to LSAS Anxiety or SPIN symptoms. These findings are rather surprising because both groups evidenced increases in cognitive control to the faces, however, this increase seemed to be harmful for the ABM group in the avoidance domain. Primarily, it is important to note that cognitive control is a global term, that could be used to delineate a range of processes (e.g., top-down orienting, inhibitory control) during stimuli conflict such as what is presented in the dot probe task (Mogg & Braddley, 2017). All of these processes require the recruitment of frontal cortical regions. Because the faces in the

task or centrally and vertically presented and there are no neutral-neutral face pairs, it is unclear whether the increase in the N2 is threat-specific. However, demystification of this finding may come by considering the distinct components of CBT and ABM that may foster different aspects of cognitive control. Specifically, CBT fosters increased activity in frontal-cortical regions during reappraisal (Goldin et al., 2013;2014), a strategy that is meant to be implemented in arousing contexts for anxious individuals. On the other hand, ABM does not explicitly create a context for learning specific coping strategies in the presence of threat – rather, it aims to implicitly train a new learned association. In other words, the aim of ABM is to manipulate attention to focus on neutral faces in the presence of a threatening face (thereby reducing the threatening salience of the emotional face). Speculatively, it could be the case that those in the ABM group learned to engage cognitive control to foster avoidance of emotional stimuli to do just that, and in the cases in which this happened, the learned mechanism transferred beyond the treatment program. However, more research is needed to examine if this is the case.

Although this study provides insight on change in neurophysiological indices of cognitive processes and their relations to symptom change, the findings should be interpreted considering a few limitations. Primarily, this study included a small sample, and as such, it would be of great utility for future studies to implement similar designs in larger samples to examine if the results remain consistent and achieve greater power for non-significant or marginal effects. In addition, the study did not include a control group assessed throughout the course of treatment. Although the assessment of both treatment groups allows for the comparison of change across the interventions, it would also be of value to examine if changes are specific to the interventions themselves, over and beyond the effect of repeatedly completing the task and the passage of time. Lastly, the dot probe task utilized in this study did not have a neutral-neutral comparison.

Future studies should include such a comparison to specify changes in these processes for threat-neutral face conflict in comparison to neutral faces.

In sum, the results suggest that both treatment groups produced increases in cognitive control during the dot probe task – to both the presentation of faces and the probe. Therefore, it can be concluded that both treatment groups fostered more engagement of frontal brain processes throughout the task related to a global cognitive control mechanism. There were no changes in early attentional processing, which could signify that the malleability of these processes and their measurement is difficult to achieve. Additionally, there was no relationship between the rates of change of ERPs and symptoms throughout the course of treatment. However, there was a relationship between the rate of change among ERPs and among symptoms. Lastly, the results showed that changes in cognitive control at one time-point predicted different effects on symptoms depending on the treatment context. Specifically, a higher N2 predicted more avoidance symptoms in the ABM group, but marginally predicted less anxious and avoidance symptoms in the CBT group. More research is needed to delineate a more conclusive interpretation of these findings, however, it is speculated that these differential effects are due to the specific aspects of cognitive control that are fostered within each treatment and thereby implemented in the dot probe task.

APPENDICES

APPENDIX A: Tables

Table 1: Descriptive Statistics on the full sample

	CBT (n = 33)	ABM (n = 17)
Age	28.67 (7.1)	27.76 (9.1)
% Female	39.4	52.9
% Right-handed	84.8	100

Note. All p's > .23

Table 2: Final model for N2 change to face presentation in the Dot Probe Task (Pre-Treatment – 8 week mark)

Fixed Effect	Estimate	Standard Error	t	p
Intercept	-2.10	0.44	-4.75	.000*
Time	-0.41	0.16	-2.6	.013*
Group	-0.22	0.44	-0.51	.61
Group X Time	-0.10	0.16	-0.63	.53
Variance Components	Variance	Standard Deviation	Covariance	--
Intercept	6.06	2.46		
Slope (Time)	0.139	0.37	0.67	
Residual	3.77	1.94		

Note. * p < .05, ** p < .001

Table 3: Final model for N2 change to face presentation in the Dot Probe Task (Pre-Treatment – Post Treatment)

Fixed Effect	Estimate	Standard Error	*t*	*p*
Intercept	-2.14	0.47	-4.5	.000**
Time	-0.38	0.17	-2.3	.022*
Group	-0.22	0.47	-0.48	.64
Group X Time	-0.11	0.17	-0.67	.50
Follow-up	0.70	0.87	0.80	.42
Follow-up X Group	0.32	0.87	0.37	.72
Variance Components	Variance	Standard Deviation	Covariance	--
Intercept	5.95	2.44		
Slope (Time)	0.08	0.28	0.43	
Residual	6.18	2.49		

Note. * $p < .05$, ** $p < .001$

Table 4: Simple Model for P1 Change Over Time to Face Presentation for Slope Extraction

Fixed Effect	Estimate	Standard Error	*t*	*p*
Intercept	2.71	0.28	9.597	.000***
Time	-0.15	0.09	-1.68	.10
Variance Components	Variance	Standard Deviation	Covariance	--
Intercept	2.84	1.69		
Slope (Time)	0.18	.42	0	
Residual	1.95	1.40		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 5: Simple Model for N2 Change Over Time to Face Presentation for Slope Extraction

Fixed Effect	Estimate	Standard Error	*t*	*p*
Intercept	-2.15	0.43	-4.99	.000**
Time	-.48	0.13	-3.77	.001**
Variance Components	Variance	Standard Deviation	Covariance	--
Intercept	5.07	2.38		
Slope (Time)	0.20	.44	0.27	
Residual	6.36	2.52		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 6: Simple Model for N1 Change Over Time to Probe Presentation for Slope Extraction

Fixed Effect	Estimate	Standard Error	*t*	*p*
Intercept	-1.35	0.23	-5.98	.000**
Time	-.02	0.08	-.33	.74
Variance Components	Variance	Standard Deviation	Covariance	--
Intercept	.65	.81		
Slope (Time)	0.007	.08	1	
Residual	3.03	1.74		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 7: Simple Model for N2 Change Over Time to Probe Presentation for Slope Extraction

Fixed Effect	Estimate	Standard Error	*t*	*p*
Intercept	3.00	0.36	8.37	.000**
Time	-.38	0.16	-2.36	.02*
Variance Components	Variance	Standard Deviation	Covariance	--
Intercept	1.55	1.25		
Slope (Time)	0.43	.66	1	
Residual	8.41	2.90		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 8: Simple Model for LSAS Total Scores Change Time for the Slope Extraction

Fixed Effect	Estimate	Standard Error	*t*	*p*
Intercept	87.70	2.30	38.19	.000***
Time	-6.98	1.04	-6.70	.000***
Variance Components	Variance	Standard Deviation	Covariance	--
Intercept	212.38	14.57		
Slope (Time)	39.14	6.26	-.14	
Residual	83.67	9.15		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 9: Simple Model for LSAS Anxiety Scores Change Time for the Slope Extraction

Fixed Effect	Estimate	Standard Error	*t*	*p*
Intercept	43.45	1.09	39.96	.000***
Time	-3.20	.49	-6.51	.000***
Variance Components	Variance	Standard Deviation	Covariance	--
Intercept	44.76	6.69		
Slope (Time)	8.15	2.85	-.12	
Residual	23.65	4.86		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 10: Simple Model for LSAS Avoidance Scores Change Time for the Slope Extraction

Fixed Effect	Estimate	Standard Error	*t*	*p*
Intercept	44.24	1.41	31.32	.000***
Time	-3.78	.55	-6.90	.000***
Variance Components	Variance	Standard Deviation	Covariance	--
Intercept	83.69	9.15		
Slope (Time)	10.48	3.24	-.23	
Residual	26.22	5.12		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 11: Simple Model for SPIN Scores Change Time for the Slope Extraction

Fixed Effect	Estimate	Standard Error	*t*	*p*
Intercept	48.16	1.27	38.03	.000***
Time	-5.36	.61	-8.81	.000***
Variance Components	Variance	Standard Deviation	Covariance	--
Intercept	70.39	8.39		
Slope (Time)	16.05	4.01	-.35	
Residual	25.31	5.03		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 12: Bivariate Correlations between ERP and Symptom Slopes

	1	2	3	4	5	6	7	8
1. P1 Face Slope	--							
2. N2 Face Slope	.32*	--						
3. N1 Probe Slope	.30*	.26†	--					
4. N2 Probe Slope	.23	0	.12	--				
5. LSAS Total Slope	-.01	.05	-.09	.09	--			
6. LSAS Anxiety Slope	.06	.12	-.02	.12	.97**	--		
7. LSAS Avoidance Slope	-.07	-.01	-.12	.06	.97**	.89**	--	
8. SPIN Slope	.18	.20	.06	.12	.76**	.81**	.67**	--

Note. † $p < .1$, * $p < .05$, ** $p < .001$

APPENDIX B: Figures

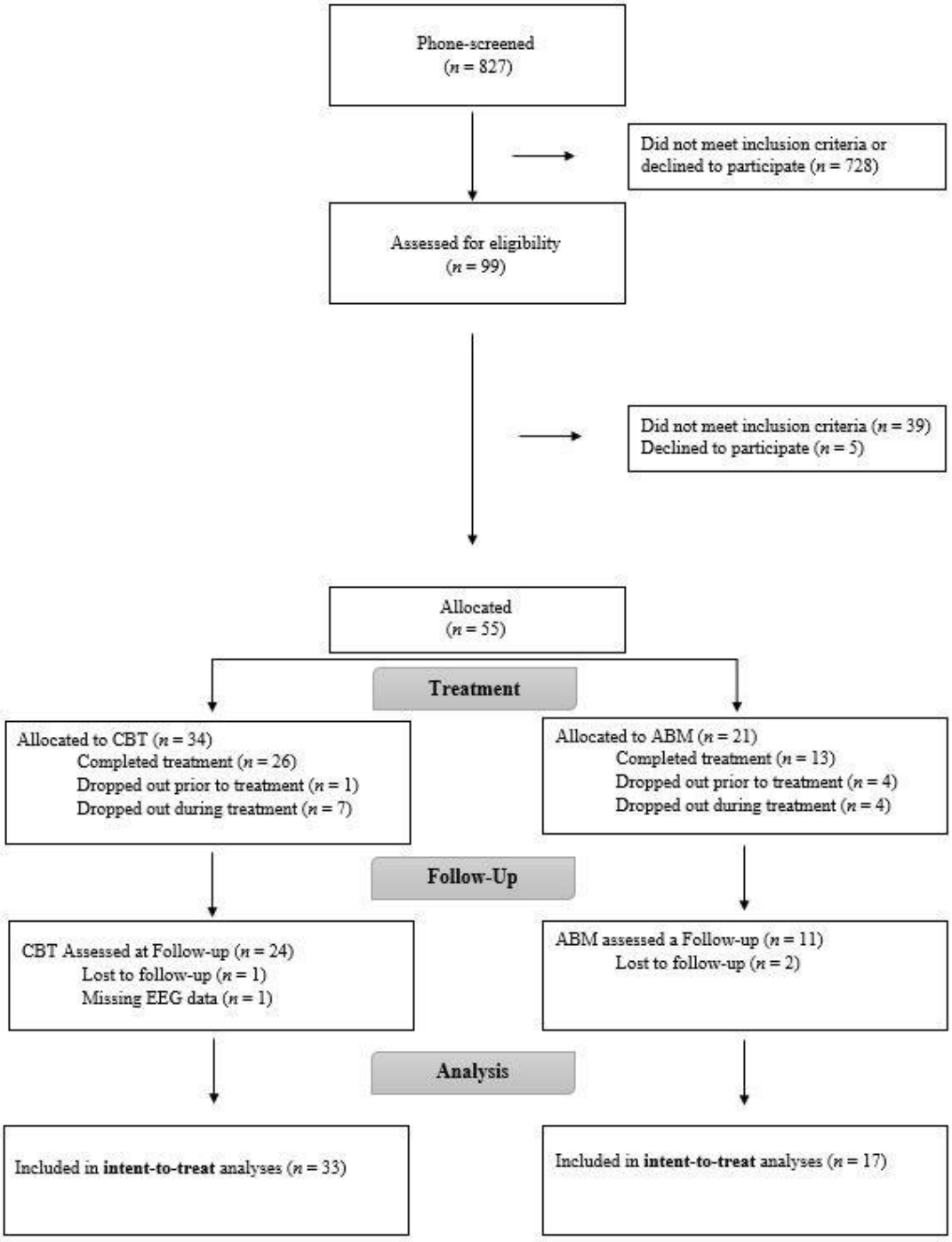

Figure 1: Flow Chart of Study Design

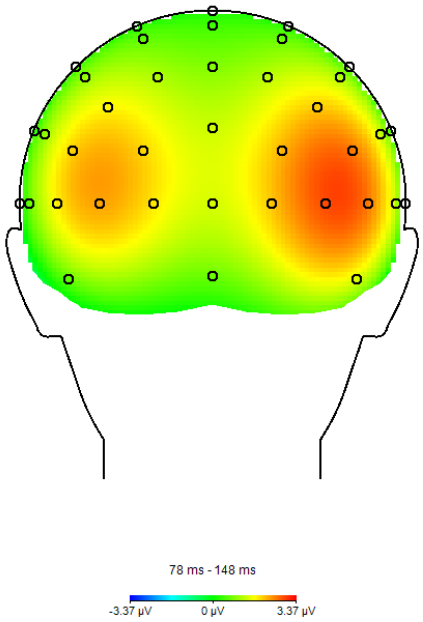

Figure 2: Topographic representation of the P1 time-locked to the presentation of the faces including all observations for the final sample in the 80-150 ms time window.

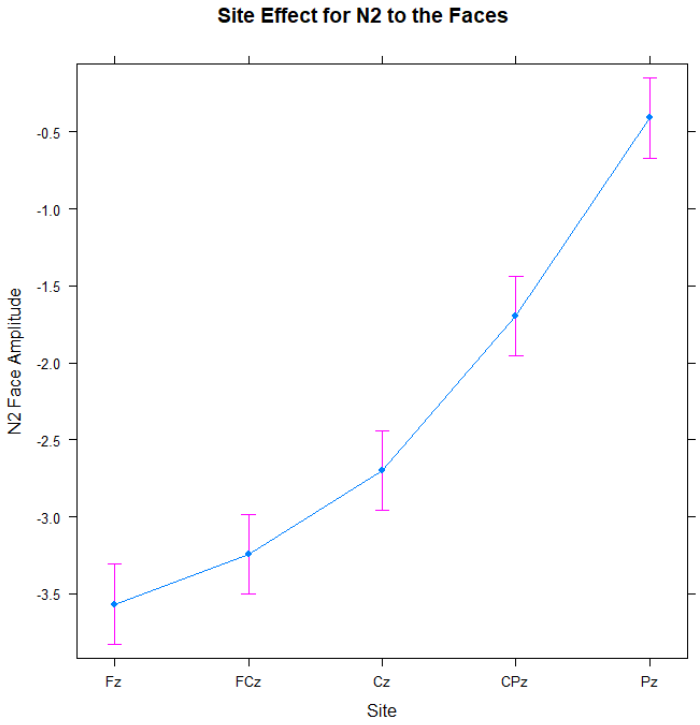

Figure 3: The effect of Site across time for the N2 to face presentation in the dot probe task. The N2 was largest at site Fz.

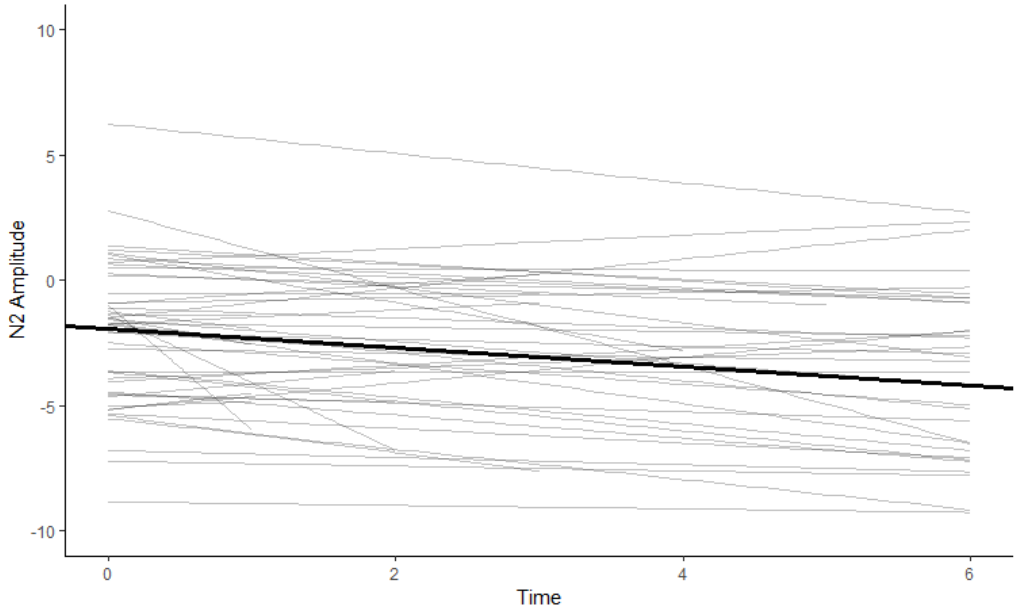

Figure 4: The N2 to the faces over time collapsed across both groups. Increased N2 was observed across treatment.

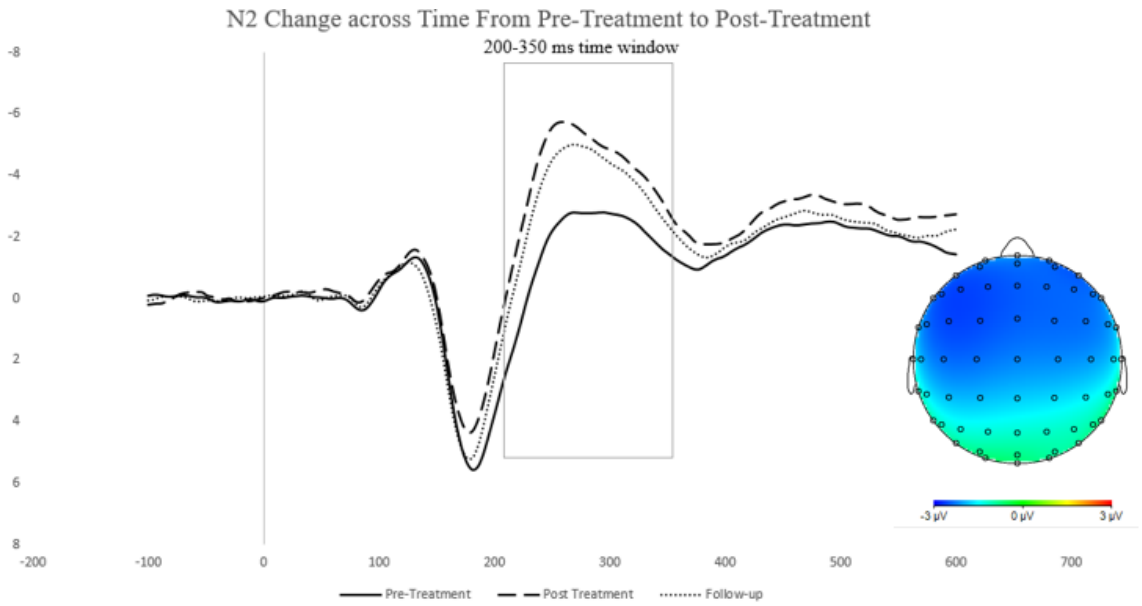

Figure 5: Line Graph of N2 to the faces. The topographic representation depicts the N2 change difference (Pre-Treatment-Post-Treatment).

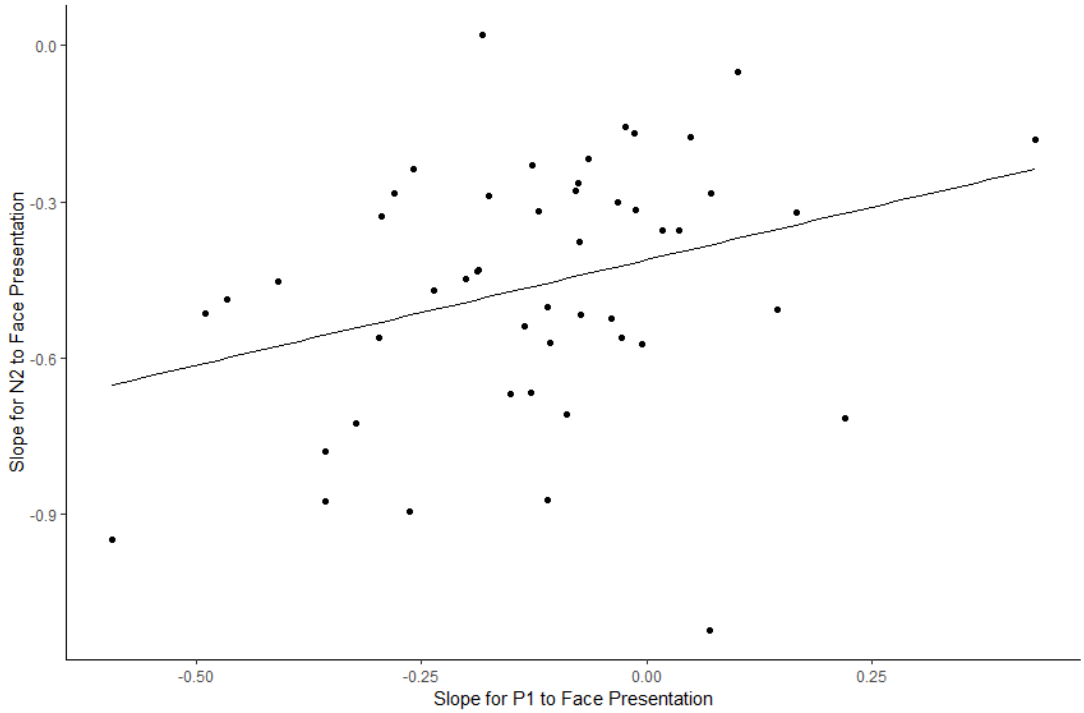

Figure 6: The relationship between the slopes (pre-post treatment) for the P1 and the N2 to face presentation.

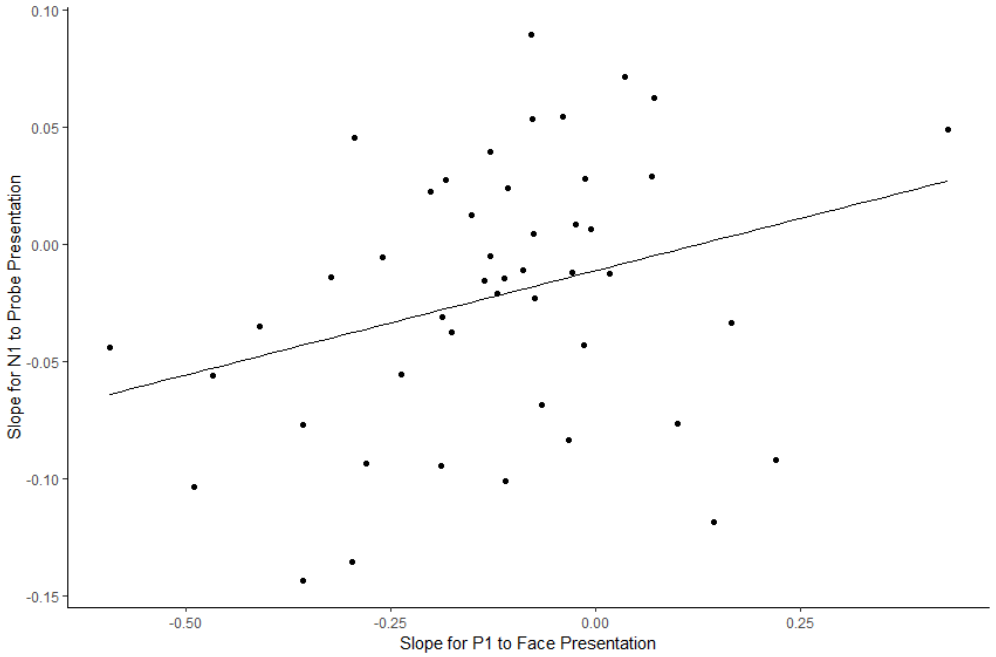

Figure 7: The relationship between the slopes (pre-post treatment) for the P1 to face presentation and the N1 to probe presentation.

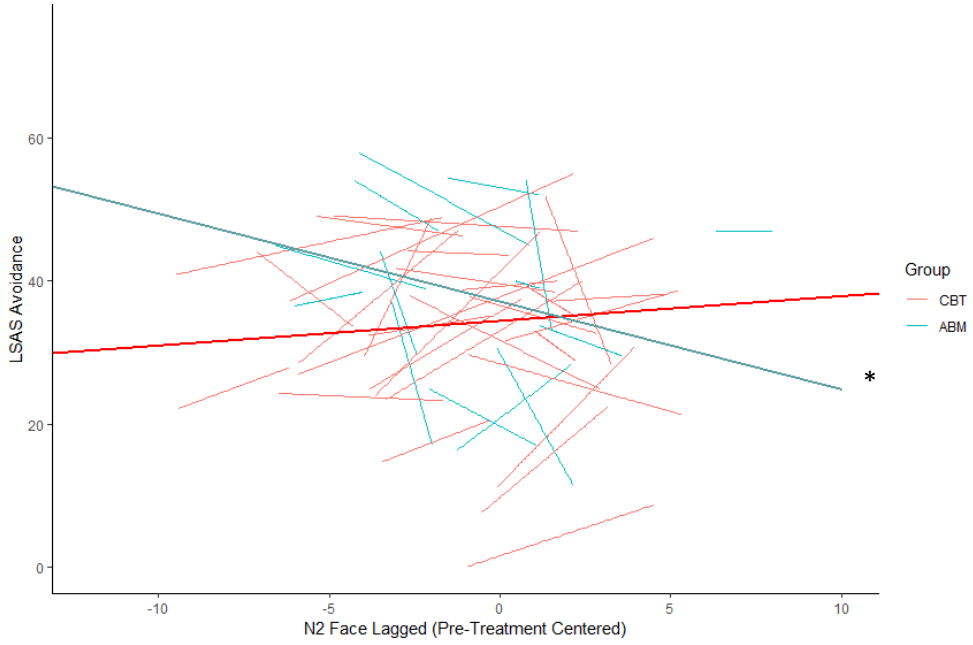

Figure 8: The relationship the N2 to face presentation and LSAS Avoidance scores, showing that a smaller N2 predicted less symptoms for the ABM group while there was no relationship in the CBT group.

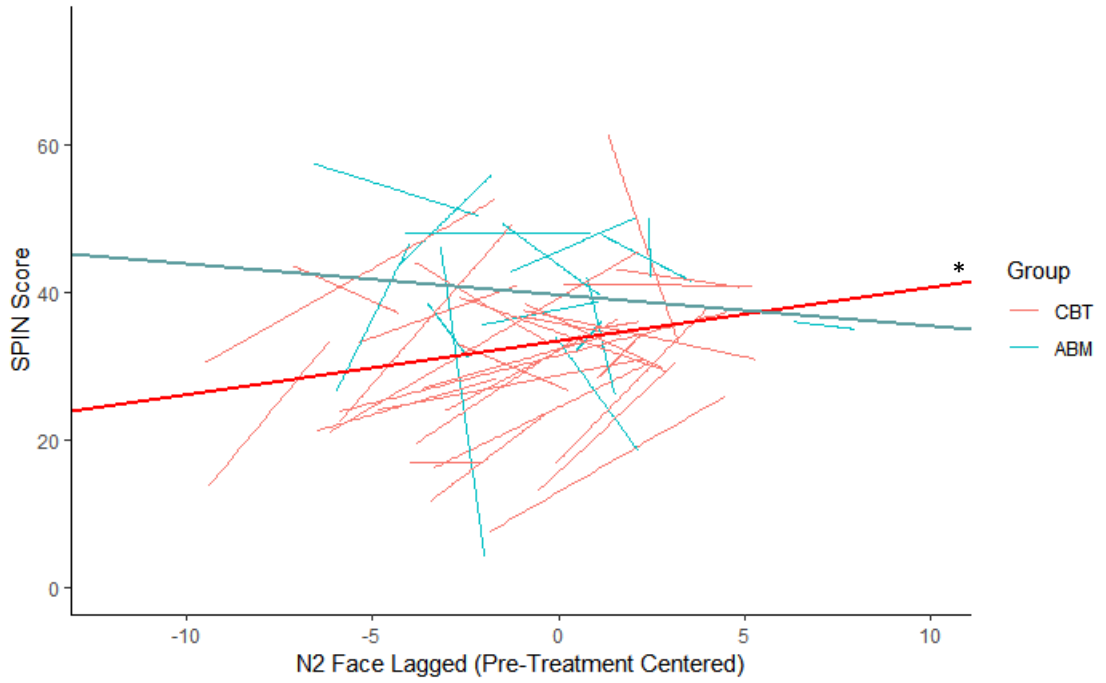

Figure 9: The relationship the N2 to face presentation and SPIN scores, showing that a smaller N2 predicted higher symptoms for the CBT group while there was no relationship in the ABM group.

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