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Neural Reactivity to Reward as a Predictor of Cognitive Behavioral Therapy Response in Anxiety and Depression

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Abstract

Background—Cognitive behavioral therapy (CBT) is a well-established treatment for anxiety and depression; however, response to CBT is heterogeneous across patients and many remain symptomatic after therapy, raising the need to identify prospective predictors for treatment planning. Altered neural processing of reward has been implicated in both depression and anxiety, and improving hedonic capacity is a goal of CBT. However, little is known about how neural response to reward relates to CBT outcomes in depression and anxiety. The current study used the reward positivity (RewP) event-related potential (ERP) component to examine whether neural reactivity to reward would predict CBT response in a sample of patients with anxiety without depression ($n = 30$) and comorbid anxiety and depression (CAD, $n = 22$).

Methods—Participants completed a guessing reward ERP paradigm before completing 12 weeks of standard CBT.

Results—The majority of the sample (68%; 35 of 52 patients) responded to treatment, and those with a reduced RewP at baseline were more likely to respond to treatment. A reduced RewP was also associated with a greater pre-to-post CBT reduction in depressive symptoms among individuals with CAD, but not among individuals with pure anxiety.

Conclusions—CBT may be most beneficial in reducing depressive symptoms for individuals who demonstrate decreased reward reactivity prior to treatment. CBT may target reward brain function, leading to greater improvement in symptoms. These effects may be strongest, and therefore most meaningful, for individuals with reward processing deficits prior to treatment.

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Keywords

depression; anxiety; reward; cognitive behavioral therapy; event-related potentials

Introduction

Cognitive behavioral therapy (CBT) is an effective, first-line psychological treatment for anxiety and depressive disorders.^[1] However, response rates to CBT for anxiety and depression vary, with a range of 51%–87% for depression and 38%–77% for anxiety disorders.^[1] These findings suggest that for many patients, CBT is ineffective, with between 13% and 62% of individuals failing to show significant reductions in symptoms. Identifying predictors of treatment response to CBT have the potential to inform treatment planning in order to maximize the likelihood of response and thereby optimize limited resources.

Altered neural processing to reward has been observed in individuals with depression^[2,3] and to a lesser extent anxiety,^[4,5,6,7] relative to healthy individuals. For example, functional magnetic resonance imaging (fMRI) studies suggest that individuals with depression exhibit reduced activity in the basal ganglia during the anticipation of^[8] and in response to^[8,9,10] receiving a reward compared to healthy controls. Electroencephalogram (EEG) studies have also elucidated electrocortical measures, as indexed by event-related potentials (ERPs), of reward response in relation to depression. In particular, the reward positivity (RewP) is an ERP component that is maximal at frontocentral electrode sites approximately 200–250 ms following the receipt of a reward and reflects processing of positive feedback (e.g., monetary reward) versus breaking even or losing.^[11,12] The amplitude of the RewP is sensitive to the valence of outcomes^[13,14] and is larger for positive versus negative outcomes.^[12] Importantly, the RewP has demonstrated excellent psychometric properties, including high retest reliability over two years and high internal reliability.^[15] There is growing evidence that the RewP is a valid measure of individual differences in reward processing, as it has been correlated with self-report reward sensitivity and reward learning behavior,^[16] positive emotionality,^[17] and activation in the ventral striatum and medial prefrontal cortex.^[18,19,20,] Notably, depression is associated with a reduced RewP among adults,^[21] and studies show that a reduced RewP predicts the onset of depressive symptoms^[15] and episodes of depression among girls of depressed mothers.^[22]

Fewer studies have examined the association between reward reactivity and anxiety, and findings have been less consistent relative to studies with depression. For example, neuroimaging studies suggest that relative to healthy individuals, youth with anxiety disorders exhibit increased striatal^[7] and orbitofrontal cortex^[5] reactivity during anticipation of reward. However, these studies have yielded inconsistent findings when examining reward responsiveness, with evidence for increased activation in the striatum,^[7] and also decreased activity in the caudate^[5] among youth with anxiety. Additionally, using ERPs, studies have found that a smaller RewP is associated with greater trait anxiety^[6] and generalized anxiety symptoms in children.^[23]

Taken together, these prior studies suggest that there are inherent differences in reward reactivity among individuals with anxiety and depression, relative to controls. Importantly, a

primary goal of CBT is to improve hedonic capacity (e.g., behavioral activation to increase frequency of positive experiences, strategies to reduce maladaptive thoughts); thus, individual differences in reward systems prior to treatment may predict response. To our knowledge, only one small study has examined whether reward-related brain activity predicts response to CBT in anxiety and depression. In an fMRI study of thirteen adolescents with comorbid anxiety and depression, greater striatal reactivity in response to reward at baseline was associated with a greater reduction in anxiety symptoms during treatment.^[9] We are not aware of any studies, however, that examined the influence of reward-related brain function on CBT response in anxious and/or depressed adults.

The current study, therefore, sought to examine how individual differences in responsiveness to reward predict CBT outcome in a sample of adults with pure anxiety and comorbid anxiety and depression (CAD). Due to high rates of depression and anxiety comorbidity evident among treatment-seeking samples,^[24] we included a comorbid anxious and depressed sample to increase the generalizability of our findings. Moreover, given the inconsistent relation between reward responsivity and anxiety, we also examined whether diagnostic status (i.e., pure anxiety versus comorbid anxiety and depression) moderated the relation between CBT response and pre-CBT reward-related neural activity.

To examine reward-related brain activity, we utilized EEG data, which compared to fMRI, is more economical, easily administered, and easily transportable to clinical settings to inform treatment planning. In addition to the previously described RewP, which measures response to reward feedback, we also examined the stimulus-preceding negativity (SPN)^[25] during reward anticipation and the N2 in response to losses. The SPN is a slow, negative-going wave that has been shown to be enhanced in anticipation of reward,^[26] whereas the N2 to losses is apparent at frontocentral electrode sites approximately 200–250 ms following the receipt of a loss.^[11,12] While previous studies have yet to examine whether adults with anxiety or depression differ from healthy controls in these ERP components, we examined whether they predicted CBT response, given the previously described relation between internalizing disorders and altered anticipation of reward.^[5,7,8]

In summary, the primary aim of the current study was to examine whether responsiveness to reward, measured via the RewP, would predict CBT response (i.e., clinician-rated improvement) in adults with anxiety and depression, and whether diagnostic status (i.e., anxiety disorders only versus comorbid anxiety and depression; CAD) would moderate this association. Second, given the well-established link between depression and reward responsiveness,^[8,9,15,21,22] we examined whether the RewP would correlate with depressive symptom change among individuals with CAD. Finally, exploratory analyses were conducted to determine if other aspects of reward functioning, including anticipation to reward and response to losses would predict CBT response.

Methods

Participants

Fifty-nine patients had pre-treatment EEG data and completed the full twelve weeks of CBT after being recruited from the community and outpatient clinics at the University of Illinois

at Chicago (UIC). Participants with excessively noisy EEG data ($n = 7$) were excluded from analyses. The final sample consisted of 52 participants, was 76.9% female, and had a mean age of 23.94 ($SD = 6.54$; range: 18–47 years). With regard to race, the sample was 61.5% Caucasian, 1.9% African American, 19.3% Asian, and 17.3% Biracial. Participants were between 18–55 years of age, met criteria for a current primary anxiety diagnosis (social anxiety disorder, panic disorder, and/or generalized anxiety disorder), or a current primary major depressive disorder diagnosis, and had no major active medical or neurological problems. Patients with the following were excluded from the study: a) clinically significant and active medical or neurologic condition, b) history of bipolar disorder, schizophrenia, presence of an organic mental syndrome, intellectual disability, or pervasive developmental disorder, c) history of psychotic symptoms, d) current substance abuse/dependence, or e) ongoing active psychotherapy and/or current treatment with any psychotropic medication. This study was approved by the UIC Institutional Review Board, and informed consent was obtained from all participants.

Measures

Diagnostic Interview—Participants were interviewed by Master's-or Doctorate-level clinicians using the Structured Clinical Interview for DSM-IV (SCID-IV) [27] to assess current and lifetime diagnoses of Axis I disorders. Thirty participants met criteria for current anxiety disorders with no history of depression (i.e., pure anxiety). In the anxiety-only group, primary diagnosis was social anxiety disorder (SAD; $n = 19$) and generalized anxiety disorder (GAD; $n = 11$). Of these 30 participants, 4 had comorbid panic disorder, 3 had comorbid SAD, 3 had comorbid specific phobia, and 2 had comorbid GAD. In the CAD cohort, primary diagnosis comprised major depressive disorder (MDD; $n = 13$) and SAD ($n = 9$). Of these 22 adults, 7 had comorbid GAD, six had comorbid panic disorder, 6 had comorbid SAD, 5 had comorbid post-traumatic stress disorder, 5 had comorbid MDD, 4 had comorbid pervasive depressive disorder, 2 had comorbid specific phobia, and 1 had comorbid obsessive-compulsive disorder.

Treatment—Within a week following the EEG assessment, patients began once-weekly sessions of manualized individual CBT for 12 weeks related to the patient's primary diagnosis and predominant symptoms,^[28,29,30,31] which comprised psycho-education, strategies to reduce negative beliefs, behavioral procedures (e.g., exposure to fears, behavioral activation), and relapse prevention. A CBT-trained licensed clinical psychologist or post-doctoral clinical psychologist conducted treatment. Clinicians were supervised by a licensed clinical psychologist with expertise in CBT.

Response to Treatment—To assess response to CBT, clinicians completed the Clinical Global Impression Rating Scale (CGI),^[32] which encompasses the Improvement Scale (CGI-I) measuring global improvement with scores ranging from 1 (very much improved) through 7 (very much worse). Patients were considered to be 'Responders' if they received a 1 or 2 following treatment, and 'Non-Responders' if their CGI-I score was greater than 2. The CGI Severity Scale (CGI-S) was also used to measure clinician-rated symptom severity with scores ranging from 1 (normal, not at all ill) through 7 (among the most extremely ill patients).

Anxiety and Depression Symptoms—To assess for generalized and social anxiety symptoms, participants completed the Penn State Worry Questionnaire (PSWQ)^[33] and the Liebowitz Social Anxiety Scale (LSAS).^[34] The Beck Depression Inventory (BDI-II)^[35] was used to evaluate depression level. All measures were completed before and after CBT.

Guessing Reward Task—Participants completed an adaptation of a guessing reward task based on work by Forbes and colleagues.^[9] As shown in Figure 1, the task comprised 60 trials (15 win, 15 loss, 15 no-win and 15 no-loss), each consisting of a decision, anticipation, and outcome period, separated by an inter-trial interval ranging between 4–7 sec. During the decision period, participants were presented with a question mark (4 sec) and pressed a button in order to guess whether a computer-selected number was greater than or less than 5. For the anticipation phase, participants viewed a circle with the numbers 1–9 and a yellow arrow indicating the range of the actual number (i.e., whether the participant was correct or incorrect; presented for 6 sec). Participants were informed that a correct response indicates the possibility of winning \$1 or breaking even, while an incorrect response indicates the possibility of losing 50¢ or breaking even. During the outcome period, participants were presented with the “actual” number for 500 ms and received feedback for 500 ms in the form of a happy face for wins, sad face for losses, and neutral face for breaking even. Participants saw their total earnings every 20 trials (\$2.50, \$4.50, \$7.50). Participants were told that they would receive the total amount of their winnings, but in fact, each participant received \$10.

EEG Data Acquisition and Processing

Continuous EEG was recorded using a 34-channel cap (32 channel setup based on 10/20 system with the addition of FCz and Iz) and the BioSemi system (BioSemi, Amsterdam, Netherlands). Electrodes were placed on the left and right mastoids, and the electrooculogram was recorded from four facial electrodes. The data were digitized at 24-bit resolution with a Least Significant Bit value of 31.25 nV and a sampling rate of 1024 Hz. The voltage from each active electrode was referenced online with respect to a common mode sense active electrode.

Data were processed offline using Brain Vision Analyzer software (Brain Products, Gilching, Germany). Data were converted to a linked mastoid reference, filtered with high- and low-pass filters of .1 and 30 Hz, respectively. Continuous EEG data were segmented beginning 100 ms before stimulus onset and continuing for the 500 ms after onset. Eyeblinks were corrected using the method by Gratton and colleagues,^[36] and semi-automated artifact rejection procedures removed artifacts with the following criteria: voltage step of more than 50 μ V between sample points, a voltage difference of 300 μ V within a trial, and a maximum voltage difference of less than 0.5 μ V within 100 ms intervals. Additional artifacts were removed using visual inspection. The mean number of artifact-free trials in each condition was 14 ($SD = 1.35$). Data were baseline corrected using the 100 ms interval prior to feedback. ERPs were averaged across gain, even, and loss trials, and the RewP was scored as the mean amplitude 230–300 ms following feedback at a pooling of frontal sites (AF3, AF4, and Fz), where the win minus breaking even difference was maximal (Figure 2). Analyses focused on the win minus breaking even difference score (RewP); more positive values for the difference score indicate greater reactivity to reward. The N2 to losses was scored as the

mean amplitude 230–300 ms following feedback of a loss at a pooling of frontal sites (AF3, AF4, and Fz), where the loss minus breaking even difference was maximal compared to breaking even (more negative values indicate greater response to losses). Finally, the stimulus-preceding negativity (SPN) was scored as the mean amplitude 200 ms prior to receiving a reward at a pooling of parietal sites (Pz, P3, and P4).^[26] Analyses focused on the anticipation of reward versus loss difference score, that is, more negative values indicate greater differentiation in the ERP when anticipating the possibility of a win compared to a loss.

Data Analysis

Hierarchical logistic regression analyses were used to examine whether the RewP, SPN, or N2 to losses at baseline prospectively predicted treatment response (CGI-I). Step 1 of the regression analyses included the ERP component (RewP, SPN, or N2 to losses) and diagnostic status, and Step 2 included the diagnostic status $\times$ ERP component interaction. Hierarchical linear regression analyses were then used to examine whether the RewP, SPN, or N2 to losses predicted change in post-treatment depressive symptoms (BDI). Step 1 of the regression analyses included pre-treatment BDI, the ERP component (RewP, SPN, or N2 to losses), and diagnostic status, and Step 2 included the diagnostic status $\times$ ERP component interaction.

Results

Based on the CGI-I, 68% of the patients (35 of 52 patients) were considered to be ‘Responders’ as they were rated to be ‘very much improved’ or ‘much improved’ (score of 1 or 2) while 17 of 52 patients were considered to be ‘Non-Responders’. There was a significant decrease in depressive and anxiety symptom severity pre-to-post CBT (BDI: $t(51) = 8.96$, $p < .001$, M difference = 15.12; PSWQ: $t(51) = 7.87$, $p < .001$, M difference = 13.28; LSAS: $t(51) = 9.16$, $p < .001$, M difference = 25.63). Table 1 presents participant characteristics separated by diagnostic status (pure anxiety vs. CAD) and CBT response [CGI-I Responders (R) and Non-Responders (NR)]. Baseline symptoms of depression and anxiety were not significantly related to the RewP, N2 to losses, or SPN (lowest $p = .17$).

Does the RewP predict response to CBT?

Results revealed a significant main effect of the RewP for CBT response [$Wald = 4.63$, $p = .03$, odds ratio = 1.18 (1.02, 1.38)]. Specifically, a lower RewP at baseline predicted better CBT response among individuals with anxiety and CAD. Figure 3 shows the topographical layout of the RewP for CBT Responders and Non-Responders. This effect was not significantly moderated by diagnostic status as the diagnostic status $\times$ RewP interaction was not significant, [$Wald = 1.70$, $p = .19$, odds ratio = 1.33 (.87, 2.05)].

Is the RewP associated with depressive symptom change pre-to-post CBT?

Results revealed no significant main effect of the RewP for BDI change ($t(47) = 1.50$, $p = .14$, $\beta = 0.08$); however, there was a significant RewP $\times$ diagnostic status interaction, ($t(47) = 2.51$, $p = .02$, $\beta = 5.35$). The RewP predicted BDI symptom change among the CAD group, ($t(19) = 2.73$, $p = .01$, $\beta = 1.13$, but not the pure anxious group, ($t(27) = -.18$, $p = .86$, $\beta = -$

04. As shown in Figure 4, among the CAD group, a smaller RewP at baseline (less responsiveness to reward) was related to lower depressive symptoms following CBT. This finding remained significant after adjusting for age, $p = .04$, $\beta = 1.18$, and baseline clinician-rated symptom severity (CGI-S), $p = .01$, $\beta = 1.12$.

Do the SPN and the N2 to losses predict CBT outcome and/or depressive symptom change pre-to-post CBT?

To evaluate whether these effects were specific to responsiveness to reward, we also examined whether anticipation to reward, measured via the SPN, and response to losses, measured via the N2 to losses, predicted CBT response. Neither the main effect of the SPN ($Wald = 1.49$, $p = .22$), the N2 to losses ($Wald = .36$, $p = .55$), nor the diagnostic group interactions (lowest $p = .68$) were significant in predicting response to CBT. We also found no evidence for the SPN or the N2 to losses predicting depressive symptom change pre-to-post CBT (lowest $p = .42$).

Discussion

The current study is the first to use the reward positivity (RewP), an ERP propagated by structures involved in reward-related functions (e.g., basal ganglia)^[18,19,37] to predict CBT outcome in anxious and/or depressed patients. Moreover, given the well-established link between depression and neural impairments in reward circuitry,^[8,9,15,21,22] we examined whether the RewP corresponded with pre-to-post CBT reductions in depression and the extent to which diagnostic status (pure anxiety versus comorbid anxiety and depression [CAD]) moderated the relationship.

First, we found that a reduced RewP to monetary award predicted CBT outcome (i.e., greater clinician-rated improvement) among patients with pure anxiety and CAD, regardless of diagnostic status. Findings indicate patients with internalizing conditions with decreased pre-CBT response in the consummatory phase of positive emotion processing (e.g., experience of achieving a goal)^[38,39] are more likely to benefit from CBT. Future studies can determine whether these levels are below healthy controls (CBT remediates this deficiency), or whether the non-responders have abnormally high levels of reactivity (CBT works in those with normative reward responses). Nonetheless, whether treatment is directed at reducing anxiety or depression, CBT may be especially helpful for patients who have less positive affect to a rewarding outcome. Historically, positive emotion responsivity has been understudied in predicting CBT outcome, particularly for anxiety disorders, which emphasize fear exposures as the central therapeutic mechanism.^[40,41] Here, results suggest individual differences in positivity disturbance plays a role in CBT response in anxious as well as depressed patients.

We also observed reduced reactivity to reward at baseline was associated with a greater reduction in depressive symptoms among individuals with CAD; however, this effect was not observed among adults with pure anxiety. Therefore, CBT may be most effective in reducing depressive symptoms in individuals with CAD who experience a blunted reward-related neural response prior to treatment, though without a depression-only comparative

cohort, we cannot disambiguate the extent to which findings relate to overlapping or unique characteristics in anxiety and depression. [42,43,44]

The specific direction of these associations (i.e., decreased reward reactivity predicting better outcome) is noteworthy, given that the opposite pattern – increased reward reactivity – could be expected to predict greater symptom change, as high hedonic individuals may be more likely to be engaged in therapy and consequently get more out of treatment. However, one interpretation of the current findings is that altering reward responses may be a mechanism of action in CBT, and that this effect may be most meaningful for individuals with *less* reactivity to rewarding outcomes prior to treatment. For example, such patients may be especially responsive to explicit cognitive and behavioral techniques that increase the likelihood the patient will experience positive emotions. In support, positive affect has been shown to increase after completing CBT in depression^[45] and in other interventions, the reward system is a direct target of treatment (e.g., deep brain stimulation for depression).^[46] Alternatively, deficits in reward reactivity may be due in part to emotional dysregulation such as suppression, which decreases hedonic capacity in anxious patients^[47,48] and improves with CBT as patients learn more adaptive coping strategies.^[49] Considered together, individuals with diminished reward reactivity prior to CBT may have more room for improvement though the paths by which CBT exerts its effects may vary. Future studies are needed that include pre- and post-measures of the RewP to determine whether CBT alters reward-related responsivity and its mechanisms of change.

Contrary to our expectation, we did not find evidence that neural response during reward anticipation (SPN) predicts CBT response or depressive symptom change. This is somewhat surprising as reward anticipation likely plays a role in CBT; however, this may be due to the use of the SPN as a neural marker of anticipation. Specifically, studies have yet to examine whether individuals with depression or anxiety differ in reward anticipation using the SPN. Therefore, other measures such as fMRI might be more sensitive in identifying differences in anticipation to monetary reward at the neural level in clinical groups. Nonetheless, even in depression where anhedonia is a core characteristic, evidence of deficient activation (e.g., basal ganglia) during reward outcome is more robust than in anticipation of reward, relative to healthy individuals.^[10] Therefore, pre-CBT individual differences in the consummatory phase of positive emotion processing may be a more stable predictor of CBT response due to greater dysfunction, at least in depression.

Strengths of the current study include examining a neural reward-related predictor of CBT response in a sample of adults with comorbid anxiety and depression. Further, this study is among the first to use ERP measures, which have the potential to be applied in clinical settings, as predictors of treatment response. Despite these strengths, there are limitations, which provide important avenues for future research. First, CBT incorporates several therapeutic components, including psychoeducation, cognitive intervention, behavioral procedures (e.g., exposures, behavioral activation, relaxation strategies), therefore, it is unclear whether a specific technique results in symptom improvement for individuals with reward responding deficits. Notably, a previous study found that behavioral activation resulted in functional changes in structures involved in responses to reward, including the striatum, among individuals with major depressive disorder.^[41] Second, while previous

studies have found that the RewP reflects activation of the basal ganglia/striatum,^[18,19,37] future studies are needed to examine which specific neural areas implicated in reward predict CBT response. Third, while the inclusion of a comorbid, generalizable sample was a strength of the current study, we were unable to examine the influence of the RewP on CBT response among individuals with pure depression. Additional studies are needed to examine if a similar pattern of results emerges for patients with pure depression relative to patients with CAD. Additionally, CBT was delivered in an open-label (non-randomized) context and thus we cannot conclude that clinical improvement is specific to CBT. It will also be important for future studies to examine whether self-report measures of reward responsiveness predict CBT response and, if so, if the RewP outperforms these measures. Finally, given the relatively small sample size and absence of a control group, it is difficult to interpret whether the magnitude of the RewP is aberrant in the current sample of adults with anxiety and depression. Future studies with control groups are needed to determine whether these relative differences in the RewP that confer greater or lesser levels of CBT response are normative, dysfunctional, or compensatory.

Conclusion

In summary, the current study provides promising evidence for reduced reactivity to reward being a neural predictor of CBT response. Findings suggest that a reduced RewP predicted better clinician-rated response to CBT among patients with anxiety and depressive disorders, and a reduction in depressive symptoms among individuals with CAD pre-to-post CBT. CBT may decrease depressive symptoms by enhancing reward brain function, where the RewP may be generated. These effects may be strongest, and therefore most meaningful, for individuals with reward processing deficits prior to treatment.

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Figure 1.

Design of the guessing reward task. The task comprised 60 trials (15 win, 15 loss, 15 no-win and 15 no-loss), each consisting of a decision period, anticipation period, and outcome period, separated by an inter-trial interval ranging between 4–7 sec.

Figure 2. ERPs (negative up) across frontal electrode sites (AF3, AF4, Fz) in response to wins and breaking even, and scalp distributions depicting the win minus even difference (reward positivity, RewP) from 230–300ms.

Figure 3.

Scalp distributions depicting the win minus even difference (reward positivity, RewP) from 230–300ms among CBT Responders and Non-Responders. Patients were considered to be ‘Responders’ if they received a 1 or 2 on the Clinical Global Impression Rating Scale, and ‘Non-Responders’ if their score was greater than 2. Responders to CBT exhibited less activation in response to reward, relative to the non-responders.

Figure 4. Scatter plot depicting the association between depressive symptoms, measured via the BDI (controlling for pre-treatment BDI), following CBT and the RewP among individuals with comorbid anxiety and depression. A smaller RewP at baseline was associated with lower depressive symptoms following CBT.

Table 1

Demographic and Clinical Characteristics of Patients Separated by Diagnostic Status

	Pure Anxious (<i>n</i> = 30)		Comorbid Anxiety/Depression (<i>n</i> = 22)		χ^2	<i>p</i>
	<i>n</i> , %		<i>n</i> , %			
CGI-I Post	-		-		0.51	0.48
I Responders (R)	19, 63.3%		16, 80.0%			
I Non-Responders (NR)	11, 36.7%		6, 20.0%			
Gender (Female)	24, 80.0%		16, 72.7%		0.38	0.54
	R (16, 84.2%)	NR (8, 72.7%)	R (10, 62.5%)	NR (6, 100%)		
Race (Caucasian)	19, 63.3%		13, 59.1%		1.44	0.69
	R (12, 63.2%)	NR (7, 63.6%)	R (12, 75.0%)	NR (1, 16.7%)		
	Mean, SD		Mean, SD		<i>t</i>	<i>p</i>
Age	27.00, 7.64		22.14, 2.97		-2.83	0.01
	R (26.89, 8.34)	NR (27.18, 6.65)	R (22.62, 3.22)	NR (20.83, 1.72)		
BDI Pre	16.20, 10.09		31.00, 8.55		5.57	<0.001
	R (16.26, 11.55)	NR (16.09, 7.44)	R (30.06, 6.32)	NR (33.50, 13.29)		
BDI Post	4.60, 5.95		11.09 (9.08)		3.11	0.01
	R (2.58, 3.64)	NR (8.09, 7.60)	R (6.56, 7.60)	NR (23.17, 5.15)		
LSAS Pre	64.30, 19.49		73.91 (27.27)		1.48	0.14
	R (63.00, 22.44)	NR (66.55, 13.68)	R (77.00, 22.90)	NR (65.67, 37.92)		
LSAS Post	40.17, 22.43		48.00 (28.56)		1.11	0.27
	R (30.58, 19.78)	NR (56.73, 16.69)	R (43.88, 23.90)	NR (59.00, 38.88)		
PSWQ Pre	62.67, 10.31		64.73 (7.96)		0.78	0.44
	R (60.68, 11.06)	NR (66.09, 8.25)	R (64.25, 8.08)	NR (66.00, 8.22)		
PSWQ Post	48.63, 13.31		53.45 (13.56)		1.28	0.21
	R (42.53, 12.53)	NR (59.18, 6.18)	R (48.50, 11.27)	NR (66.67, 10.17)		
RewP	2.05, 4.76		2.14 (4.22)		0.07	0.95
	R (1.27, 5.10)	NR (3.39, 3.98)	R (0.94, 3.77)	NR (5.34, 3.91)		
SPN	0.05, 7.78		-1.43 (6.00)		-0.74	0.46
	R (-0.71, 8.27)	NR (2.34, 5.80)	R (-2.07, 6.96)	NR (0.67, 3.45)		

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Note: CGI-I = Clinical Global Impression-Change; BDI = Beck Depression Inventory; LSAS = Liebowitz Social Anxiety Scale; PSWQ = Penn State Worry Questionnaire; RewP = Reward Positivity; SPN = Stimulus Preceding Negativity.