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Neural Predictors of Improvement With Cognitive Behavioral Therapy for Adolescents With Depression: An Examination of Reward Responsiveness and Emotion Regulation

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Abstract

Earlier depression onsets are associated with more debilitating courses and poorer life quality, highlighting the importance of effective early intervention. Many youths fail to improve with evidence-based treatments for depression, likely due in part to heterogeneity within the disorder. Multi-method assessment of individual differences in positive and negative emotion processing could improve predictions of treatment outcomes. The current study examined self-report and neurophysiological measures of reward responsiveness and emotion regulation as predictors of response to cognitive-behavioral therapy (CBT). Adolescents (14–18 years) with depression ($N = 70$) completed monetary reward and emotion regulation tasks while electroencephalogram (EEG) was recorded, and self-report measures of reward responsiveness, emotion regulation, and depressive symptoms at intake. Adolescents then completed a 16-session group CBT program, with depressive symptoms and clinician-rated improvement assessed across treatment. Lower reward positivity amplitudes, reflecting reduced neural reward responsiveness, predicted lower depressive symptoms with treatment. Larger late positive potential residuals during reappraisal, potentially reflecting difficulty with emotion regulation, predicted greater clinician-rated improvement. Self-report measures were not significant predictors. Results support the clinical utility of EEG measures, with impairments in positive and negative emotion processing predicting greater change with interventions that target these processes.

Keywords Adolescence · Depression · Reward · Emotion regulation · Cognitive behavior therapy · EEG

Depression is one of the most prevalent and impairing forms of psychopathology, with lifetime prevalence estimates ranging from 16.6% to 41.4% (Kessler & Wang, 2008; Moffitt et al., 2010). There is a sharp increase in the incidence rate for depressive disorders during adolescence (Lewinsohn et al., 1994), and rates of adolescent depression have increased by 52% in the past decade (Twenge et al., 2019). Earlier onsets are associated with an increased risk for comorbidities, greater functional impairment, and overall poorer quality of life (Zisook et al., 2007). This highlights the importance of effective intervention selection for adolescent depression. Although cognitive behavioral therapy (CBT; Wright & Beck, 1983) is among the most empirically supported treatments for depression (Cuijpers et al., 2010), 40% to 60% of adolescents do not respond to CBT (March et al., 2007; Weersing et al., 2017). Given individual variability in response, methods for predicting who will improve with specific interventions are critically needed to better match individuals to the treatments most likely to be effective.

Depression is characterized by alterations in both positive and negative emotion processing. Substantial changes in the neural circuitry involved in emotion processing occur in adolescence, potentially contributing to the increased risk for depression during this period (Powers & Casey, 2015). Disruptions in reward processing are particularly central to the emergence of internalizing disorders, with

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consistent evidence of associations between reduced reward responsiveness and depression (Alloy et al., 2016; Olino, 2016). For negative emotions, depression is associated with impaired emotion regulation, including more elaborative processing of negative content and less use of adaptive regulatory skills (Disner et al., 2011; Joormann & Gotlib, 2010). Pre-treatment individual differences in positive and negative emotion processing may predict responses to interventions that directly target these impairments. Considering the core components of CBT include efforts to increase positive emotions through behavioral activation (Dimidjian et al., 2011), and the reduction of negative emotions through strategies like cognitive reappraisal (Wright & Beck, 1983), CBT may be particularly useful for adolescents with impairments in reward responsiveness and emotion regulation. Previous research has supported the possibility that behavioral activation may increase reward responsiveness (Dichter et al., 2009), suggesting that CBT may be most effective for people with deficits in reward processing prior to treatment. Similarly, given evidence that CBT increases activation in brain regions involved in emotion regulation in adults (Roiser et al., 2012; Rubin-Falcone et al., 2018), adolescents with difficulties regulating sad and dysphoric emotions may similarly benefit most from CBT. At the same time, there is also evidence that interventions can be enhanced by building on individual strengths (Cheavens et al., 2012), suggesting that adolescents who begin CBT with relatively strong reward responsiveness and emotion regulation skills may show the most improvement (e.g., Webb et al., 2021).

Validated, objective predictors of improvement with treatment are critically needed to facilitate patient engagement and retention, inform clinical decision-making, and improve treatment outcomes. Although previous research has demonstrated that clinical characteristics predict treatment response (Curry et al., 2006), few studies have examined individual differences in neural indices of emotional processing as predictors of CBT outcomes in depressed adolescents. Further, incorporating both self-report and neural measures of emotion enables direct comparisons of the predictive utility of each type of measure. Neural measures may be stronger predictors of future behavior than traditional measures (Gabrieli et al., 2015), and may improve the prediction of treatment outcomes beyond clinical indicators (Ball et al., 2014), but this has yet to be empirically tested with similar constructs assessed across methods.

Event-related potentials (ERP) derived from electroencephalogram (EEG) capture individual differences in both reward responsiveness and emotion regulation. ERP research provides a non-invasive, economical measure of neural processing that is easily assessed across development and could be implemented in clinical settings. In particular, the reward positivity (RewP) is an ERP component indexing reward responsiveness that typically emerges around 250 ms after feedback onset over frontocentral sites and is enhanced for reward compared to loss feedback (Proudfit, 2015). RewP has demonstrated good reliability across development (Bress et al., 2015; Kujawa et al., 2018), supporting its potential for informing prognosis and treatment planning. Prior research indicates that a reduced RewP predicts a better response to selective serotonin reuptake inhibitor (SSRI) treatment in adults (Burkhouse et al., 2016, 2018), and greater depressive symptom reductions following CBT in anxious youth (Kujawa et al., 2019a, b).

The late positive potential (LPP) component is a sustained positive deflection in the ERP waveform beginning approximately 300 ms post-stimulus onset that is enhanced for emotional stimuli compared to neutral (Schupp et al., 2000). Prior research has shown that the LPP is modulated by regulatory efforts such as cognitive reappraisal (Foti & Hajcak, 2008; Hajcak & Nieuwenhuis, 2006), supporting the LPP as a measure of emotion regulation (Hajcak et al., 2010). Although research on the LPP during emotion regulation in clinical depression and depression treatment is relatively limited, one study found potentiated LPP responses to negative stimuli, reflecting heightened emotional reactivity, predicted more of a decrease in depressive symptoms following CBT in anxious and/or depressed adults (Stange et al., 2017).

Based on previous evidence of alterations in both positive and negative emotional processing in depression and the importance of effective early intervention for adolescent depression, we aimed to identify predictors of improvement with CBT. Adolescents with depression completed EEG and self-report measures of reward responsiveness and emotion regulation prior to a 16-session group CBT intervention. Self-reported depressive symptoms and clinician-rated improvement were assessed across treatment. We hypothesized that reduced neural reward responsiveness measured by RewP would predict greater response to treatment, reflected in both reductions in self-reported depressive symptoms and greater clinician-rated improvement. Despite the relatively limited research on LPP modulations during emotion regulation in association with treatment response, we expected less differentiation between the LPP during the passive viewing and emotion regulation conditions would predict greater symptom reductions and clinical-rated improvement. Further, we expected that neural measures would account for unique variance in treatment outcomes beyond demographic, clinical, and self-report predictors.

Methods

Participants

Participants were adolescents 14–18 years of age ($M = 15.81$, $SD = 1.46$) with current major depressive disorder (MDD; five or more symptoms most of the day nearly every day

for at least two weeks) and/or persistent depressive disorder (PDD; milder symptoms for at least one year) based on semi-structured clinical interviews and at least moderate severity based on interviewer ratings on the Clinical Global Impression Scale at intake (CGI; Guy, 1976). Participants were recruited through community advertisements, social media, and mental health clinics across two sites (7.1% from Penn State University and 92.9% from Vanderbilt University due to a relocation of the research lab). Seventy eligible participants enrolled. Exclusion criteria included a diagnosis of mania, psychosis, intellectual or developmental disability, and/or a substance use disorder severe enough to require treatment.[1] Current use of antipsychotic medications and mood stabilizers were also excluded. Participants receiving other forms of treatment were included, as long as treatment was stable for 30 days prior to the start of the study (41.4% were taking medication for emotional or behavioral problems and 45.7% were in other types of therapy). Concurrent medication and psychotherapy were examined as predictors and covariates in supplementary analyses (see Tables S4 and S5 in Supplemental Information). Of the 70 enrolled participants, 66 completed the EEG assessment, 56 started CBT, and 37 completed CBT through session 16 and participated in at least 12 sessions (see Figure S1 in the Supplementary Information for the CONSORT diagram). Participants were 65.7% female, 4.3% Hispanic/Latinx, 87.1% White/Caucasian, 4.3% Asian, 7.1% Black/African American, and 1.4% multiracial. Treatment completers did not significantly differ from non-completers in age, sex, race, ethnicity, baseline depressive symptoms, baseline ERPs, concurrent medication, or concurrent psychotherapy ($ps > 0.155$).

Procedure

Study procedures were registered on clinicaltrials.gov (https://clinicaltrials.gov/ct2/show/NCT03154008) and approved by the Institutional Review Boards at both Penn State University and Vanderbilt University. Informed consent was obtained from participants who were 18 years old or from parents or caregivers while assent was obtained from minor participants. Diagnostic clinical interviews and pre-treatment self-report measures were completed during an initial intake assessment. Eligible participants completed the pre-treatment EEG assessment shortly after the intake visit. Once five to 10 adolescents completed intake measures and the EEG assessment, participants were assigned to a CBT group to begin the intervention. Financial compensation was provided for participation in the intake interview, EEG assessment, and completion of questionnaires.

Treatment was administered in a group format[2] using the 16-session (one-and-a-half to two-hour sessions twice weekly for eight weeks) Coping with Depression Course for Adolescents (CWD-A; Clarke et al., 1990). CWD-A is a structured group CBT course that includes psychoeducation, behavioral activation, cognitive restructuring, social skills, relaxation techniques, and problem-solving and communication skills. CWD-A demonstrates significant reductions in depressive symptoms and diagnoses in adolescents post-treatment, with further improvements evident at the six-month follow-up (Lewinsohn et al., 1990). All groups were led by a masters- or doctoral-level clinician with the support of a bachelors- or masters-level co-leader under the supervision of a licensed clinical psychologist. Group leaders completed training in CWD-A with Dr. Paul Rohde, one of the developers of the intervention, and participated in weekly group supervision to ensure adherence to the protocol and to address challenges with group dynamics. Most sessions were completed in person prior to COVID-19, but eight out of 16 sessions for the last group ($n = 5$) moved to Zoom due to the start of the pandemic. On average, participants who started treatment completed 10.91 sessions ($SD = 4.21$, range = 1–16), and 66.1% of participants who started treatment completed the final session of treatment. Participants who missed more than four sessions were withdrawn from treatment to ensure that treatment completers participated in at least 75% of the intervention. Treatment completers were briefly interviewed following session 16 to determine whether they met the criteria for full or partial remission.

Measures

Diagnostic Interview Current and lifetime diagnoses were determined using the DSM-5 version of the Schedule for Affective Disorders and Schizophrenia for School-Aged Children from six to 18 years (K-SADS; Kaufman et al., 2013) administered by clinical psychology doctoral students or masters-level clinicians (for minors, parents were also interviewed). All diagnoses were reviewed and verified by a licensed clinical psychologist. For current depression diagnoses at intake, 28.6% met criteria for both MDD and PDD (i.e., chronic MDD), 24.3% met for MDD only, 32.9% met

[1] When covarying current substance use disorder in our models, results remained consistent with primary analyses, showing RewP significantly predicted depressive symptom changes in both intent-to-treat and treatment completer models $ps < .03$, while reappraisal-related LPP predicted clinician-rated improvement for treatment completers $p = .03$.

[2] We conducted three-level multilevel models to account for clustering within treatment groups. Differences between groups only accounted for a small amount of variance in outcomes and primary results were generally consistent with those presented in the manuscript (see Tables S2 and S3 in the Supplemental Information). As such, we opted to focus on the simpler regression models for the primary results presented here.

for PDD with intermittent major depressive episodes (MDE) including current MDE, 8.6% met for PDD with intermittent MDE without current MDE, and 5.7% met for PDD without a history of MDE. The average age of onset for current depressive episodes was 13.65 years (*SD* = 2.38) with an average episode duration of 110.71 weeks (*SD* = 117.45; range = 3.00–676.00). Regarding severity, 61.4% of participants were classified as moderately ill and 38.6% were classified as markedly ill. Current comorbid diagnoses included 55.7% with at least one anxiety disorder, 17.1% with attention-deficit/hyperactivity disorder, and 4.3% with oppositional defiant or conduct disorder. To evaluate inter-rater reliability, eight audiotaped interviews at baseline were reviewed and coded by an independent interviewer. Inter-rater reliability was excellent (kappa = 1.0 for MDD; kappa = 1.0 for PDD).

Reward Responsiveness At baseline, participants completed a validated eight-item self-report measure of reward responsiveness, adapted from the Behavioral Inhibition System/Behavioral Activation System scales (RR scale; Van den Berg et al., 2010). Items included "If I discover something new I like, I usually continue doing it for a while" and "When I see an opportunity for something I like, I get excited right away," and were rated on a scale from one (strong disagreement) to four (strong agreement). The RR scale demonstrated good internal consistency (Cronbach's alpha = 0.82).

To assess reward responsiveness at the neural level, participants completed a monetary reward task that has been tested extensively across development (e.g., Bress et al., 2015; Kujawa et al., 2018). Two doors were presented on the screen and participants were instructed to select a door, which may have a prize behind it. Participants were informed correct selections would be rewarded with $0.50 while incorrect selections would result in a loss of $0.25, with the potential to earn up to $5.00 throughout the task. Following door selection, a fixation cross (+) was presented for 1000 ms followed by either an upward green arrow, indicating the receipt of monetary reward, or a red downward arrow, indicating monetary loss. Feedback arrows were presented for 1500 ms followed by another fixation cross for 1000 ms. The message "Click for the next round" then appeared on the screen until participants clicked, beginning the next trial. Participants completed two practice trials, one for each type of feedback, followed by 30 reward trials and 30 loss trials presented in a randomized order. All participants earned the full $5.00 following the completion of the task.

Emotion Regulation Participants completed the self-report Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) at baseline. The ERQ is a ten-item measure consisting of two subscales assessing the use of reappraisal and expressive suppression. Only the reappraisal subscale was used for analysis, as it is most similar to the emotion regulation task and cognitive restructuring components of CBT. The reappraisal subscale includes items such as "When I want to feel more positive emotion, I change the way I'm thinking about the situation," which are rated on a scale from one (strongly disagree) to seven (strongly agree). The reappraisal subscale of the ERQ had good internal consistency (Cronbach's alpha = 0.81).

To assess baseline emotion regulation at the neural level, an emotion regulation task was adapted from previous ERP studies (Moser et al., 2014). The experimenter first presented the participant with a description and examples of emotion regulation techniques, with a focus on cognitive reappraisal to change the interpretation of the image (e.g., in response to an image of a young child crying, "This child was upset for a moment, but the mother quickly came and picked the child up and the child was okay."). Next, participants completed four practice trials with two dysphoric images each presented following a "LOOK NEGATIVE" and "DECREASE NEGATIVE" instruction. On look trials, participants were instructed to view the image and to let themselves interpret and respond to it naturally. On decrease trials, participants were instructed to try to think of the picture in a more positive or neutral way to decrease their emotional reaction. After those four practice trials, the experimenter asked the participant to describe the strategies used to decrease their emotional responses on the decrease trial and gave additional examples and explanations of cognitive reappraisal techniques as needed to ensure an understanding of the task. Participants were additionally prompted to rate the intensity of their emotional response from zero (none) to seven (very strong) after each image presentation on all trials. Finally, participants completed six practice trials including the emotional intensity rating before beginning the task. On each trial, participants were first presented with the instruction to "LOOK NEUTRAL", "LOOK NEGATIVE", or "DECREASE NEGATIVE" for 2000 ms. A fixation mark (+) was then presented for 500 ms, followed by a neutral or dysphoric image from the International Affective Picture System (Lang et al., 2005) presented for 6000 ms. Considering the focus on clinical depression, dysphoric images, such as images of people with expressions of emotional pain,[3] were selected, rather than externally threatening stimuli.

[3] Dysphoric IAPS images: 2455, 2053, 2141, 2205, 2276, 2301, 2345, 2375, 2456, 2457, 2700, 2703, 2750, 2799, 2800, 2900, 3180, 3230, 3300, 3350, 6311, 9041, 9220, 9332, 9927.
Neutral IAPS images: 2200, 2190, 2210, 2215, 2221, 2480, 2493, 2512, 2516, 2570, 2840, 5500, 7000, 7002, 7009, 7010, 7020, 7025, 7035, 7050, 7080, 7100, 7150, 7170, 7217.

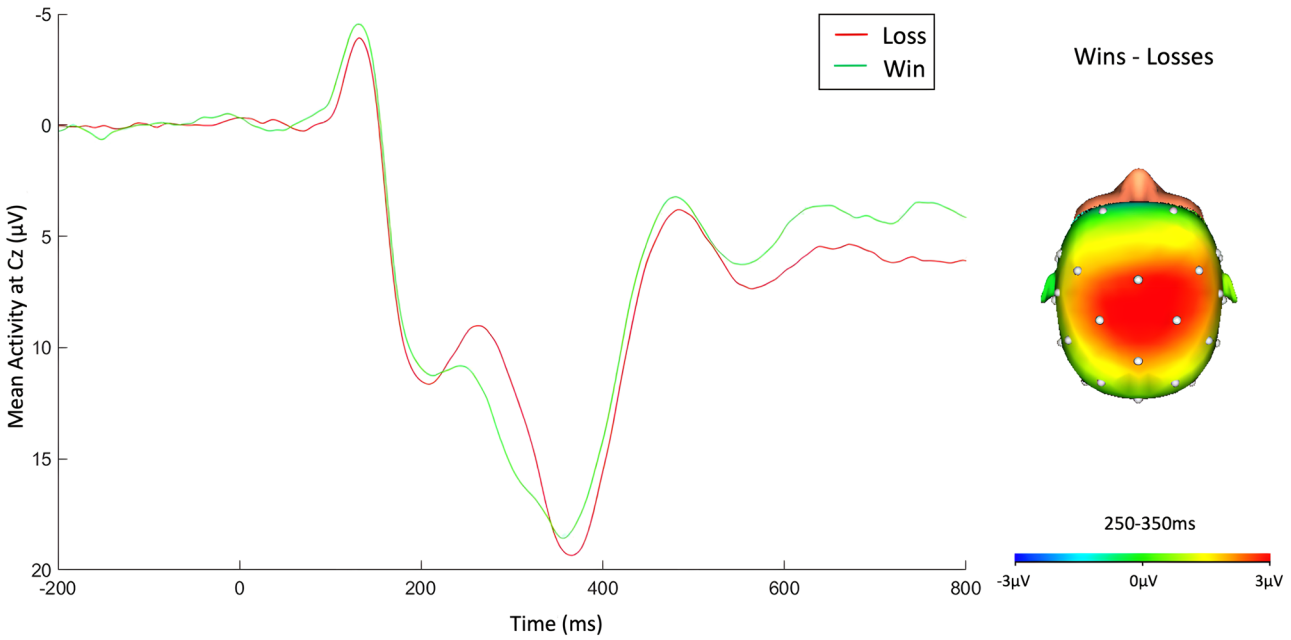

Fig. 1 Waveforms for ERPs in response to win trials (green) and loss trials (red) at Cz. The scalp distribution depicts electrocortical activity to wins minus losses *for illustrative purposes*; unstandardized residual scores for RewP to wins were used in analyses

Neutral images included household objects, nature scenes, and people with neutral expressions. Finally, participants rated the intensity of their emotional response from zero (none) to seven (very strong). A 2500 ms fixation was presented prior to the start of the next trial. The task included 25 trials per condition (75 trials total) presented in random order. Consistent with prior work (Moser et al., 2014), the same set of 25 dysphoric images was used for both look and reappraise trials so that differences in responses between conditions could be attributed to differences in the participant's attempts to regulate responses to the image, rather than differences in the content of the images.

EEG Data Collection and Processing EEG data were continuously recorded using a 32-channel actiCHAmp system from BrainProducts (Munich, Germany). Electrooculogram was recorded using facial electrodes placed one centimeter from the outer corners of each eye, and one centimeter above and below the left eye. Impedances were lowered below 30 kOhm and voltages from active electrodes were referenced online to Cz. Data were digitized at a sampling rate of 1000 ms. Data were processed offline using BrainVision Analyzer software (BrainProducts, Munich, Germany) and re-referenced to the averaged mastoid recordings (TP9/TP10).

RewP data were band pass filtered with cutoffs of 0.10 and 30 Hz and segmented from –200 to 800 ms for feedback presentation. LPP data were band pass filtered with cutoffs at 0.01 and 30 Hz, given evidence that a more stringent high pass cutoff can attenuate later portions of the LPP (Hajcak et al., 2012), and segmented from -200 to 6000 ms for image presentation. Other processing steps were identical for both tasks. Ocular correction was applied using Gratton's algorithm (Gratton et al., 1983) and artifact rejection was handled using semiautomated procedures identifying voltage steps of more than 40 μV between sampling points, maximum voltage differences of 150 μV within a trial, a minimal allowed amplitude of -200 μV and maximal allowed amplitude of 200 μV, and a voltage difference less than 0.50 μV within 100 ms intervals. Data were visually inspected to remove additional artifacts. Segments were averaged within each condition and baseline corrected to -200 ms.

RewP was scored between 250-350 ms at Cz, consistent with prior research and where the component was maximal (see Fig. 1; Ethridge et al., 2017; Nelson et al., 2016). Spearman-Brown coefficients for split-half reliability for RewP to wins and losses were 0.88 and 0.90, respectively. No RewP data needed to be excluded due to excessive noise. RewP was enhanced for win compared to loss feedback, $F(1, 65) = 33.92$, $p < 0.001$, $\eta_p^2 = 0.34$. Regression-based residual scores were calculated for RewP to wins accounting for RewP to losses, with larger residuals reflecting greater reward responsiveness.

Consistent with prior LPP research on emotion regulation, we scored separate poolings of centroparietal (Cz, CP1, CP2, Pz, P3, P4) and frontal electrodes (Fz, F3, F4;Dennis & Hajcak, 2009; Foti & Hajcak, 2008; Moser et al., 2014). The LPP waveforms are presented in Fig. 2. To assess the

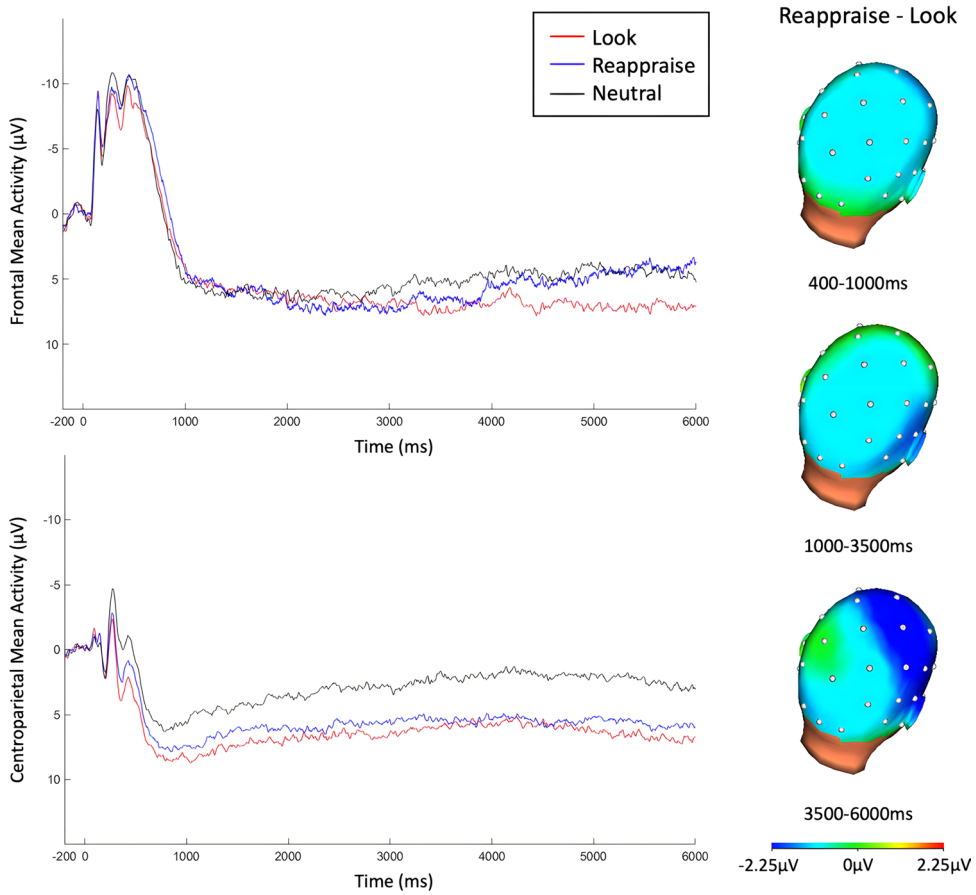

Fig. 2 Waveforms for ERPs while passively viewing neutral images (black), passively viewing dysphoric images (red), and reappraising dysphoric images (blue) across frontal (top) and centroparietal (bottom) sites. Scalp distributions depict relative electrocortical responses for the reappraisal minus passive viewing conditions for dysphoric images across three time windows *for illustrative purposes*; unstandardized residual scores for LPPs during reappraisal were used in analyses

temporal dynamics of emotion regulation and given prior research showing the LPP is composed of several distinct but overlapping positivities (Foti et al., 2009; Pegg et al., 2019), the LPP was scored across three time windows: 400-1000 ms, 1000-3500 ms, and 3500-6000 ms, consistent with prior research (Gupta et al., 2022). Spearman-Brown coefficients for split-half reliabilities for the full LPP window (400-6000 ms) were acceptable to good (0.58-0.74 across electrode poolings and conditions), but reliability varied across the time course of the LPP (reliabilities across frontal sites for reappraisal in the middle and late window and the neutral condition in the late window ranged from 0.53-0.58; all other time windows, conditions, and poolings ranged from 0.65-0.81). Five participants were missing data for LPP analyses due to excessive noise in the EEG data, likely due to the longer stimulus presentation time in the emotion regulation task (6000 ms) compared to the reward task (800 ms). We conducted repeated-measures ANOVAs (see Supplementary Information) to isolate emotion regulation effects in the overall sample and inform scoring for treatment response analyses. LPP amplitudes were reduced during reappraisal compared with passive viewing from 400-1000 ms in the centroparietal pooling, $F(1, 61) = 5.87$, $p = 0.018$, $\eta_p^2 = 0.09$, and from 3500-6000 ms across frontal sites, $F(1, 60) = 4.98$, $p = 0.029$, $\eta_p^2 = 0.08$. Regression-based residual scores for these two LPPs were calculated for the LPP during reappraisal accounting for the LPP during passive viewing, with more negative residuals reflecting greater modulation of the LPP during emotion regulation (Meyer et al., 2017).

Clinician-Rated Treatment Response The primary group leader (with feedback from co-leaders) rated participant improvement dimensionally every two weeks throughout treatment using the CGI improvement scale (CGI-I), which consists of a single item ("Compared to [their] condition at admission, how much have [they] changed?") and measures change in participants' symptoms and functioning on a scale from one (very much improved) to seven (very much

worse). Improvement ratings ranged from 1.0–5.0 ($M = 3.02$) for the last completed treatment session. Participants' last completed treatment session with CGI-I data ranged from 1–16, $M = 12.55$.

Self-Reported Treatment Response Depressive symptoms were assessed using the 33-item Mood and Feelings Questionnaire administered at intake and every two weeks throughout treatment (MFQ; Angold et al., 1995). Items on the MFQ assess the extent to which participants have experienced each symptom in the past two weeks and are rated as either zero (not true), one (sometimes true), or two (true). MFQ scores are computed by summing all items. Intake scores ranged from 4.0–61.0 ($M = 34.52$) while scores ranged from 0.0–59.0 ($M = 23.84$) for the last completed assessment. Participants' last completed treatment session with MFQ data ranged from 1–16, $M = 12.51$. Internal consistencies for each assessment were excellent, with Cronbach's alpha ranging from 0.91-0.95.

Data Analysis

First, overall intervention outcomes were examined using repeated-measures ANOVAs testing the change in depressive symptoms across treatment. Levels of improvement based on CGI-I ratings were also examined. Next, separate regression models were tested to assess the effects of pre-treatment ERPs on both changes in self-reported depressive symptoms and clinician-rated improvement since (the two outcome measures specified in the preregistration on clinicaltrials.gov). Relevant clinical variables (e.g., number of sessions attended, treatment site, comorbid anxiety, and involvement in concurrent treatment outside of the study) were entered as covariates in step one to account for other factors that may contribute to individual differences in treatment response. In models examining depressive symptoms as the outcome, baseline depressive symptoms were also included in step one to isolate the change in symptoms over time. Models examining clinician-rated improvement did not include baseline symptoms because these ratings already reflected the amount of change with treatment. Self-report measures of reward responsiveness or reappraisal were entered in step two. Finally, either RewP or LPP residuals were entered in step three to assess the unique variance accounted for by these neural measures. Models excluding the covariates and self-report measures were also tested. Primary analyses used an intent-to-treat approach with all participants who completed intake assessments ($N = 70$), using the last measure available for each participant, while follow-up models included only treatment completers ($n = 37$). Full-information maximum likelihood was used to estimate missing data in the regression analyses (Enders, 2013), which were conducted with the *lavaan* package in R (Rosseel, 2012). To avoid dichotomizing clinician-rated improvement into responders versus non-responders (Maxwell & Delaney, 1993), CGI-I was treated as a continuous variable based on research indicating maximum likelihood performs comparably to categorical least squares for ordinal variables with five or more categories and approximately symmetric thresholds (Rhemtulla et al., 2012).

Results

Descriptive statistics and bivariate correlations for study variables and within-subjects effects of condition on ERPs are presented in Supplementary Information (Table S1). Notably, RewP and the LPP were not significantly correlated with baseline depressive symptoms, which may be attributable to limited variability in depressive symptoms at intake given the chronicity and severity of depression in our sample.

Treatment Outcomes

Results of paired-sample t-tests examining change in self-reported depressive symptoms across treatment showed depressive symptoms significantly decreased from pre- to post-treatment both in intent-to-treat, $t(54) = 5.56$, $p < 0.001$, Cohen's $d = 0.72$, and treatment completion samples, $t(36) = 4.40$, $p < 0.001$, $d = 0.75$. Reliable change index calculations indicated a change greater than 10.89 on self-reported depressive symptoms reflects reliable improvement with treatment (Jacobson & Truax, 1991). Based on this criterion, 43.6% of participants achieved reliable improvement in depressive symptoms. Based on the last obtained clinician CGI-I rating, 67.9% of participants who began treatment were determined to show at least minimal improvement, and 32.1% were classified as much improved or very much improved. For those who completed treatment, 78.1% demonstrated at least minimal improvement, with 37.5% classified as minimally improved and 40.6% as much improved. Analyses of the effects of comorbid diagnoses, concurrent individual therapeutic or pharmacological treatment, skills development, and treatment engagement on outcomes, as well as analyses examining changes in self-reported anxiety symptoms are presented in the Supplemental Information.

Reward Responsiveness Predicting Treatment Response

Results of the intent-to-treat hierarchical linear regression analyses with RewP predicting treatment response are presented in Table 1. Self-reported reward responsiveness did

Table 1 Hierarchical linear regression models with self-reported reward responsiveness and pre-treatment RewP win residuals predicting self-reported depressive symptoms and clinician-rated improvement

Dependent Variable:	**Self-Reported Depressive Symptoms**				**Clinician-Rated Improvement**			
Variable		***B (SE)***	**95% CI**	***β***		***B (SE)***	**95% CI**	***β***
Step 1:	$R^2 = .37$				$R^2 = .29$			
Baseline depressive symptoms		0.53 (0.11)	[0.32, 0.74]	.58***		-	-	-
Site		-0.26 (5.81)	[-11.65, 11.13]	-.01		0.16 (0.40)	[-0.62, 0.95]	.05
Concurrent Treatment		2.24 (3.35)	[-4.33, 8.81]	.08		0.42 (0.23)	[-0.03, 0.87]	.23^
Comorbid Anxiety Disorder		1.56 (3.12)	[-4.55, 7.67]	.06		-0.17 (0.21)	[-0.58, 0.23]	-.10
Number of Sessions		-0.14 (0.38)	[-0.89, 0.61]	-.04		-0.11 (0.03)	[-0.16, -0.06]	-.50***
Step 2:	$R^2 = .37$				$R^2 = .30$			
Self-reported Reward-Responsiveness		-0.01 (0.42)	[-0.84, 0.82]	-.00		0.02 (0.03)	[-0.03, 0.08]	.12
Step 3:	$R^2 = .42$				$R^2 = .31$			
RewP Win Residuals		0.91 (0.42)	[0.09, 1.74]	.23*		-0.01 (0.03)	[-0.07, 0.05]	-.05

***$p < 0.001$; **$p < 0.01$; *$p < 0.05$; ^$p < .10$

not significantly predict post-treatment depressive symptoms or clinician-rated improvement, $ps > 0.371$. A reduced RewP predicted lower post-treatment depressive symptoms (accounting for baseline symptoms), $z = 2.16$, $p = 0.031$ (see Fig. 3), but was not predictive of clinician-rated improvement, $p = 0.680$. Notably, RewP accounted for a significant amount of unique variance, $\Delta R^2 = 0.05$, $F(1, 63) = 5.42$, $p = 0.023$, in depressive symptoms over and above the variance accounted for by self-reported reward responsiveness and clinical measures. The effect of RewP on depressive symptom change remained significant in models examining treatment completers only ($B = 1.60$, $SE = 0.56$, $\beta = 0.37$, $z = 2.88$, $p = 0.004$) and excluding clinical covariates ($B = 0.92$, $SE = 0.42$, $\beta = 0.23$, $z = 2.20$, $p = 0.028$).

Emotion Regulation Predicting Treatment Response

Regression results for intent-to-treat models examining the early centroparietal LPP and late frontal LPP in the prediction of treatment response are presented in Table 2. Self-reported reappraisal did not significantly predict post-treatment depressive symptoms or clinician-rated improvement, $ps > 0.619$. While the late frontal LPP did not significantly predict clinician-rated improvement in intent-to-treat

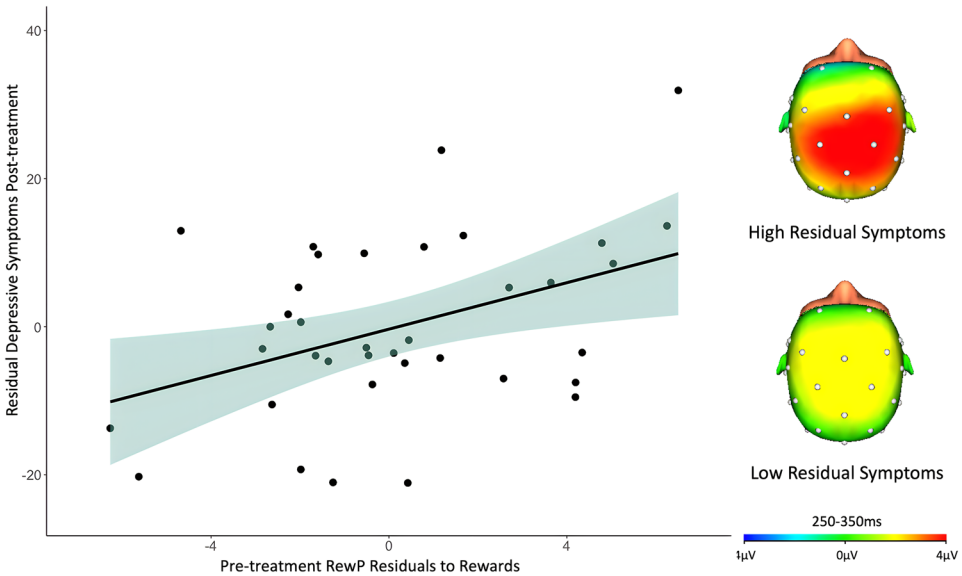

Fig. 3 Scatterplot of pre-treatment RewP residuals to wins predicting residual depressive symptoms post-treatment (unstandardized residuals calculated by predicting post-treatment symptoms from intake symptoms), with scalp distributions depicting RewP for adolescents with high (top) and low (bottom) residual symptoms. Note: The dichotomous split into high and low depressive symptoms was used *for illustrative purposes only*; all analyses used depressive symptoms as a continuous variable

Table 2 Hierarchical linear regression models with self-reported reappraisal and pre-treatment early centroparietal and late frontal LPP residuals during reappraisal predicting self-reported depressive symptoms and clinician-rated improvement

Dependent Variable:	**Self-Reported Depressive Symptoms**				**Clinician-Rated Improvement**			
Variable		***B (SE)***	**95% CI**	***β***		***B (SE)***	**95% CI**	***β***
Step 1:	$R^2 = .37$				$R^2 = .29$			
Baseline depressive symptoms		0.53 (0.11)	[0.32, 0.74]	.58***		-	-	-
Site		-0.26 (5.81)	[-11.65, 11.13]	-.01		0.16 (0.40)	[-0.62, 0.95]	.05
Concurrent Treatment		2.24 (3.35)	[-4.33, 8.81]	.08		0.42 (0.23)	[-0.03, 0.87]	.23^
Comorbid Anxiety Disorder		1.56 (3.12)	[-4.55, 7.67]	.06		-0.17 (0.21)	[-0.58, 0.23]	-.10
Number of Sessions		-0.14 (0.38)	[-0.89, 0.61]	-.04		-0.11 (0.03)	[-0.16, -0.06]	-.50***
Step 2:	$R^2 = .37$				$R^2 = .31$			
Self-reported Reappraisal		-0.11 (0.22)	[-0.55, 0.33]	-.06		-0.01 (0.02)	[-0.04, 0.02]	-.05
Step 3:	$R^2 = .37$				$R^2 = .36$			
Centroparietal LPP Reappraisal Residuals		0.19 (0.51)	[-0.81, 1.19]	.04		0.03 (0.04)	[-0.04, 0.09]	.19
Frontal LPP Reappraisal Residuals		-0.04 (0.28)	[-0.59, 0.51]	-.02		-0.03 (0.02)	[-0.07, -0.01]	-.18

***$p < 0.001$; **$p < 0.01$; *$p < 0.05$; ^$p < .10$

analyses, $p = 0.137$, it did significantly predict clinician-rated improvement for treatment completers, $B = -0.04$, $SE = 0.02$, $\beta = -0.32$, $z = -2.13$, $p = 0.033$. Results indicated larger LPP residuals during reappraisal predicted greater improvement with treatment (see Fig. 4), and reappraisal-related LPPs accounted for a significant amount of unique variance in improvement ratings beyond covariates, $\Delta R^2 = 0.12$, $F(2, 62) = 5.52$, $p = 0.006$. Reappraisal-related LPPs were not predictive of depressive symptom changes, $ps > 0.709$, and the effect on clinician-rated improvement was not significant excluding clinical covariates, $B = -0.02$, $SE = 0.02$, $\beta = -0.13$, $z = -0.93$, $p = 0.352$.

Discussion

The current study tested individual differences in neural and self-report measures of reward responsiveness and emotion regulation in clinically depressed adolescents as predictors of change in depressive symptoms and

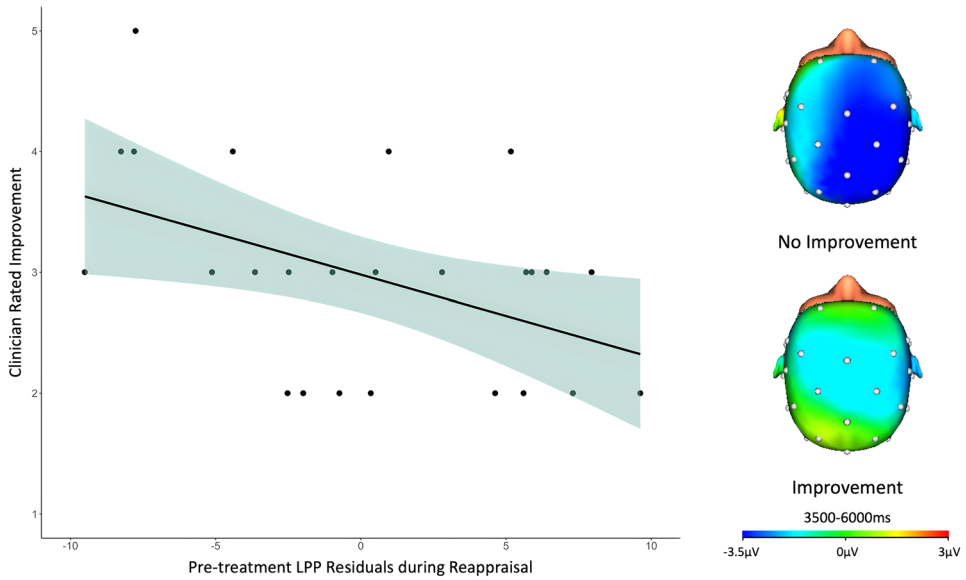

Fig. 4 Scatterplot for pre-treatment LPP residuals during reappraisal predicting clinician-rated improvement (lower scores reflect greater improvement), with scalp distributions depicting the reappraisal-related LPP for adolescents with no improvement (top; CGI-I ≥ 4) and at least minimal improvement (bottom; CGI-I ≤ 3). Note: The dichotomous split into improvement versus no improvement was used *for illustrative purposes only*; all analyses treated clinician-rated improvement as a continuous variable

clinician-rated improvement with CBT. Consistent with previous research (Burkhouse et al., 2016, 2018; Kujawa et al., 2019a, b), adolescents with reduced RewP to reward feedback pre-treatment, indicating blunted neural responsiveness to rewards, reported lower depressive symptoms with treatment, accounting for baseline symptoms. Further, adolescents with larger frontal LPP residuals during the reappraisal of dysphoric images, potentially reflective of impaired emotion regulation abilities, showed greater clinician-rated improvement with CBT among those who completed treatment. Neural measures predicted treatment outcomes over and above clinical and self-report predictors, demonstrating potential clinical utility.

Impairments in positive emotion processing, including alterations in reward responsiveness, are particularly prominent in mood disorders (Admon & Pizzagalli, 2015). Consistent with prior research examining individual CBT (Burkhouse et al., 2016, 2018; Kujawa et al., 2019a, b), the present results indicate a reduced RewP pre-treatment predicts lower self-reported depressive symptoms with treatment and extend prior research to clinically depressed adolescents participating in group CBT. Interestingly, this effect was specific to depressive symptom reductions rather than global ratings of improvement. Although the current study lacks a control group to confirm that these patterns are specific to CBT response, it is important to note that the trajectory of observed symptom changes with treatment is opposite to the pattern found in prospective studies of the association between RewP and depression in community samples. Specifically, prospective longitudinal research using naturalistic follow-up assessments indicates individuals with a reduced RewP show *increased* depressive symptoms over time (Bress et al., 2013; Kujawa et al., 2019a, b; Nelson et al., 2016). However, results are distinct from a recent study which did not find evidence that earlier stages of reward responsiveness indexed by RewP predicted treatment response, but instead, more elaborative processing of rewards captured by the LPP predicted better response to treatment (Webb et al., 2021). These discrepancies are potentially attributable to differences in the reward tasks or clinical characteristics of the sample (e.g., a purely MDD sample versus MDD or PDD with comorbid disorders in our study).

Depression is also characterized by dysfunction in the processing of negative emotions, with specific deficits in emotion regulation (Berking et al., 2014; Joormann & Gotlib, 2010; Kovacs et al., 2009). Reappraisal-related LPP residuals did not predict change in depressive symptoms across treatment but did significantly predict clinician-rated improvement among treatment completers. This differs from prior work showing enhanced LPP reactivity to aversive stimuli predicted larger depressive symptom reductions and CGI-rated response following CBT (Stange et al., 2017), though ours is the first study to examine neural markers of emotion regulation specifically. Our results showed larger frontal LPP residuals during reappraisal at later stages of processing, which is thought to potentially reflect impaired emotion regulation abilities, was associated with more clinician-rated improvement following treatment. These findings could indicate that clinicians may notice early improvements in adolescents' cognitive restructuring abilities and functioning before these skills manifest in symptom changes, although replication of these results is needed before the clinical implications can be determined.

It is important to note that the LPP effects were less robust across models, such that the effects reached significance in treatment completer analyses but not in models excluding clinical covariates or intent-to-treat analyses. The variability could be partially attributable to the lower reliability of the LPP at later stages, which is consistent with prior research parsing the reliability of the LPP (Hill et al., 2022) and highlights the need for replication and additional psychometric-specific research. What remains unclear is whether the low split-half reliability results from poorer psychometric properties of the LPP at such a relatively late stage of processing or meaningful individual differences in the ability to regulate responses across trials, as some types of emotional images may be particularly difficult to regulate emotional responses to relative to others. Additionally, the lack of coherence across self-report and clinician-rated indices of treatment response highlights a critical issue regarding the conceptualization and measurement of treatment response. Distinct processes may underlie observed improvements in session and adolescents' subjective experiences of depressive symptoms.

Our study is the first to directly compare positive and negative emotion processing (i.e., reward responsiveness and emotion regulation) at both the self-report and neural level in the prediction of treatment response for adolescents with clinical depression. In contrast to neural measures, self-report measures of reward responsiveness and use of reappraisal were not predictive of treatment response. Further, self-reported reward responsiveness and reappraisal were not correlated with depressive symptoms, which was surprising; it may result from our sample consisting of all clinically depressed adolescents with at least moderate severity. Neural and self-report measures may capture distinct aspects of emotional processing. Differences may be attributable in part to the time frame assessed or meaningful differences between subjective perceptions and objective function. As such, there may be greater convergence across methods when assessing subjective experiences in the moment through methods like ecological momentary assessment or lab-based manipulations of emotional and cognitive processes. There are also likely important differences between subjective self-perceptions and objective neural functioning which may not be fully reconciled by adjusting the time scale

of assessments. For example, some individuals may underestimate or overestimate a given tendency like their ability to regulate their emotions or level of response to rewards. For reward responsiveness, differences may also stem from the type of reward captured by each measure (e.g., receipt of monetary reward versus broader experiences of rewards in daily life). While neural measures provide a more objective assessment, the development and validation of robust self-report measures with improved coherence with neural functioning are needed.

The current sample was unique in the generally chronic and early onset of depression, and we administered an intensive, empirically supported, manualized CBT intervention. While the findings may help optimize treatment selection for depressed youth, our study has some notable limitations. First, though deficits in reward responsiveness and emotion regulation may predict responses specifically to behavioral activation or cognitive components of CBT, respectively, our study did not directly compare to these treatment components. Research comparing predictors of response to behavioral activation vs. cognitive therapy or other treatment modalities (i.e., antidepressant medications) is needed. We did not exclude participants for concurrent treatment and did not include a control group. Although this limits our ability to definitively conclude that observed changes were the result of treatment, the patterns of associations are opposite to those found in prospective research on reward responsiveness and emotion regulation (Bress et al., 2013; Kujawa et al., 2019a, b; Nelson et al., 2016; Young et al., 2019). There was a high rate of attrition across treatment, which could be due to the relative severity and chronicity of depression in the sample overall, as well as the limited flexibility in scheduling treatment sessions with the group format. Our sample consisted predominantly of White/Caucasian and female participants, limiting the generalizability of these findings to more diverse groups. Given the chronicity and severity of depression in our sample, additional research is also needed to determine if the results generalize to more mildly depressed samples. Finally, the relatively small sample size limits our power to examine moderators of treatment response, such as interactions between ERPs and demographic and clinical variables (e.g., age, sex, participant engagement, anxiety diagnoses).

The current findings demonstrate neurophysiological measures of both positive and negative emotional processing predict distinct outcomes of CBT for adolescents with depression over and above self-report and clinical measures. Research identifying reliable predictors of treatment response is critical given the need to optimize treatment selection due to the substantial proportions of non-responders and the debilitating lifetime course of depressive disorders. Our results replicate previous research by showing reduced responsiveness to rewards pre-treatment predicts depressive symptom reductions following treatment and reveal novel findings that larger LPP residuals during reappraisal, potentially indicating impaired emotion regulation, prior to treatment predicts greater clinician-rated improvement among treatment completers. Further, RewP appeared to be a more robust predictor across models than the LPP, but considering this is among the first studies to examine both ERPs, replication is needed. Taken together, the findings support a need-based model of treatment response when considering brain function, such that adolescents with neural deficits in reward responsiveness and emotion regulation experienced greater improvement with CBT. Although future research is needed to examine changes in ERPs following treatment and extend results to long-term outcomes, with standardization and the establishment of norms, neurophysiological measures could potentially be applied to facilitate precision medicine efforts.

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Data Availability The data that support the findings from this study are available by request to the corresponding author.

Compliance with Ethical Standards

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Ethical Approval Study procedures were registered on clinicaltrials.gov (https://clinicaltrials.gov/ct2/show/NCT03154008) and approved by the Institutional Review Boards at both Penn State University and Vanderbilt University.

Informed Consent Informed consent was obtained from participants who were 18 years old or from parents or caregivers while assent was obtained from minor participants.

Conflict of Interests The author(s) declare no conflicts of interest.

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