Psychophysiology

WILEY

PSYCHOPHYSIOLOGY SPR

ORIGINAL ARTICLE OPEN ACCESS

Differences in Consummatory but Not Anticipatory Reward Processing Predict Depressive Symptoms in Young Adult Women

Ty Lees[1,2] | Lisa M. Gatzke-Kopp[3]

[1]Center for Depression, Anxiety and Stress Research, McLean Hospital, Belmont, Massachusetts, USA | [2]Department of Psychiatry, Harvard Medical School, Boston, Massachusetts, USA | [3]Human Development and Family Studies, The Pennsylvania State University, University Park, Pennsylvania, USA

Correspondence: Lisa M. Gatzke-Kopp (lisakopp@psu.edu)

Received: 26 March 2024 | **Revised:** 27 January 2025 | **Accepted:** 5 February 2025

Funding: This work was supported by Environmental Influences on Child Health (ECHO) Program (Grant UH3 OD023332).

Keywords: depression | ERPs | fb-P3 | FRN | psychopathology | RewP

ABSTRACT

Depression has been postulated to relate to alterations in both anticipatory (i.e., motivation) and consummatory (i.e., hedonic pleasure) stages of reward processing. However, few studies have concurrently examined the various processes of these stages. Furthermore, little attention has been paid to whether these associations are sex-specific, despite increasing evidence of the sex specificity of neural markers of internalizing disorders. The current study examines event-related potentials (ERPs) of reward processing recorded during a monetary incentive delay task among a community sample of $n = 309$ emerging adults in relation to self-reported symptoms of depression. Regression modeling indicated that greater depressive symptom scores were associated with reduced responsivity to reward feedback and increased responsivity to non-reward feedback (as indexed by the Feedback-P3) but only for participants who were identified as female at birth. Individual differences in anticipatory processes (as indexed by both the Cue-P3 and CNV) were not associated with depressive symptoms for either sex. Results of these models suggest that depressive symptoms appear to be associated with consummatory reward processing for young women. It is possible that other dimensions of negative affect could be more poignant for male participants or may provide an additional description of the relationship between reward processing and depressive symptoms.

Behavioral and imaging-based neuroscience research has suggested that aberrations in reward processing may play a role in multiple forms of psychopathology, perhaps most notably depression (Ng et al. 2019; Nusslock and Alloy 2017; Whitton et al. 2015; Zhang et al. 2013). A core symptom of depression is anhedonia, which can manifest as reduced or absent motivation to pursue activities an individual usually enjoys and/or a diminished ability to derive pleasure (i.e., reward) from them (American Psychiatric Association 2022; Gorwood 2008; Treadway and Zald 2011). While "reward processing" is often conceptualized as a general construct, it can be broken down into two distinct temporal stages: an individual's motivation toward or "wanting" of reward, followed by their "liking" or hedonic experience in response to the receipt of a reward (Berridge et al. 2009). Moreover, research has demonstrated that these stages of reward processing (i.e., anticipation and consumption) are supported by distinct anatomical and neurochemical pathways (Liu et al. 2011; Meyer et al. 2021; Nusslock and Alloy 2017). Despite the centrality of reward processes in the purported etiology of depression (Pizzagalli et al. 2011), research has focused more heavily on the consummatory stage of processing (as noted by Glazer et al. 2018), with less consideration of the reward processing system holistically. For instance, depression may be specific to deficits in the hedonic processing of reward, which may

lead to reductions in motivated behavior over time as an individual learns that motivated effort is not effective. Alternatively, it is possible that deficits in either the anticipatory (i.e., motivational) or consummatory (i.e., hedonic) stages are sufficient to contribute to increased symptoms of depression, resulting in etiological heterogeneity within the syndrome. In order to investigate these possible patterns of association, the current study utilizes event-related potentials (ERPs) from a monetary incentive delay (MID) task to quantify individual differences across the temporal sequence of reward processing in a sample of late adolescents/emerging adults in order to evaluate how these components relate to self-reported depression.

1.1 | Anticipatory Reward Processing

Anticipatory reward processing is framed as the process/es that promote/s approach toward reward (Berridge et al. 2009; Berridge and Robinson 2003; Glazer et al. 2018). Underlying this behavior is a diffuse neural circuit centered around the ventral pallidum, the nucleus accumbens, the hypothalamus, and amygdalae (Nguyen et al. 2021), as well as their mesocortical projections to the medial prefrontal, anterior cingulate, insular, and orbitofrontal cortices (Berridge et al. 2009; Haber and Knutson 2010; Knutson et al. 2008; Kringelbach 2005; Leknes and Tracey 2008). Anticipatory reward is a complex cognitive process that involves recognizing the opportunity for reward and deciding to pursue the opportunity to obtain it. As such, the stage includes both evaluative (cue identification) and motor (response preparation) stages (Glazer et al. 2018). The evaluative stage is typically indexed by the cue-P3, a positive-going centro-parietal ERP that typically peaks inside the 300–600-ms post-stimulus window (Polich 2007). Most often, the P3 is associated with working memory context updating and attentional allocation toward, and evaluative processing of, the presented stimuli (Hajcak and Foti 2020; Polich 2007) and is typically larger for valanced stimuli, that is, those indicative of potential reward or loss, relative to neutral stimuli (Novak and Foti 2015).

This evaluative substage is followed by motor preparation, that is, the planning of motor action that serves the motivated pursuit of reward. This stage is indexed by the contingent negative variation (CNV), a broad slow-going negative deflection that occurs between the cue and target stimuli (Brunia et al. 2011; Walter et al. 1964). Some research indicates that CNV amplitude is enhanced by reward incentives (Novak and Foti 2015; Pfabigan et al. 2014; Schevernels et al. 2014; Vuillier et al. 2015). However, other studies have observed no such effect of reward on the CNV amplitude, suggesting that motor preparation may be independent of incentive evaluation (Broyd et al. 2012; Goldstein et al. 2006).

1.2 | Consummatory Reward Processing

Consummatory reward processing broadly refers to the cognitive and affective processes associated with reward attainment or omission (Berridge et al. 2009; Berridge and Robinson 2003; Glazer et al. 2018; Knutson et al. 2001). Anatomically, this stage involves the ventral pallidum and nucleus accumbens and their projections to the frontal cortices (Nguyen et al. 2021). Affectively, this stage is described as the hedonic experience that comes with attaining a reward but is also sensitive to the omission of an expected reward (Berridge and Robinson 2003; Nguyen et al. 2021). One index of reward responding is the reward positivity (RewP), a positive-going fronto-central ERP elicited approximately 250–350 ms after receiving a reward. Notably, this signal tracks valence, with a related peak, the feedback-related negativity (FRN), being observed in the same temporal and anatomical position in response to the omission of a reward (Hajcak et al. 2006; Miltner et al. 1997). Traditionally framed as indexing reward responsivity or prediction errors (Holroyd and Coles 2002), it has also been suggested that these components reflect differences in reward sensitivity (Lange et al. 2012; Threadgill and Gable 2016).

Another common index of rewarding processing is the Feedback-P3 (Fb-P3), a centro-parietal ERP that peaks between 300 and 600 ms directly following the RewP/FRN, which is also implicated in consummatory reward processing (Glazer et al. 2018). Sensitive to reward probability as well as gains and losses, the Fb-P3 is moderated by reward magnitude (Foti, Weinberg, et al. 2011; Hajcak et al. 2005; Pfabigan et al. 2011; San Martín 2012) and is thought to index the processes by which outcome-related information is categorized to maximize future reward (Donchin 1981; Polich 2007) and/or the motivational salience of reward feedback (San Martín 2012).

1.3 | Reward Processing and Its Association With Depression

Numerous studies have investigated depression-related changes in both anticipatory and consummatory reward processing ERPS, although the results presented for both stages are not always consistent. Starting with anticipatory ERPs, several studies have reported reduced cue P3 amplitude among individuals with major depressive disorder relative to healthy controls (Santopetro et al. 2021; White et al. 2021; Zhou et al. 2019). In contrast, studies that have examined dimensional measures of depressive symptoms among community-based samples have reported no association between cue-P3 amplitude and depression severity (Ait Oumeziane et al. 2019; Novak et al. 2016; Umemoto and Holroyd 2017), suggesting that depression may only be associated with lower attention to reward in clinical contexts.

Alternatively, some researchers have suggested that depression may not be associated with a change in the desirability of reward but rather a lesser willingness to exert effort to obtain rewards (Treadway et al. 2012). Consistent with this postulation, several studies have reported reduced CNV amplitudes among individuals with major depressive disorder (Ashton et al. 1988; Van derhasselt et al. 2014), as well as patient samples characterized by having attempted suicide (Ashton et al. 1994; Hansenne et al. 1996). However, a multitude of studies with similar sample sizes and clinical characteristics have failed to detect effects associated with CNV amplitude (Ait Oumeziane et al. 2019; Knott and Lapierre 1987; Köhler et al. 2011). Altogether, evidence suggests that deficits in anticipatory processing of reward stimuli may be more likely to manifest among individuals with clinically significant depression rather than as a linear association. However, it is possible that variations in anticipatory

reward processing are necessary but not sufficient mechanisms of depression. For instance, many individuals could evidence a blunted response to reward opportunities but only develop depression in the context of an additional vulnerability. Therefore, it is important to consider a holistic approach to understanding how reward processes predict depression.

Consummatory processes have been the primary focus of most research investigating depression and reward processing (see Foti and Weinberg (2018) and Glazer et al. (2018) for commentary). Across this literature, several studies have observed a reduction in RewP amplitude when comparing child (Belden et al. 2016) and mostly female adult patients with current and remitted MDD (Foti et al. 2014; Klawohn et al. 2021; Liu et al. 2014; Whitton et al. 2016) to matched healthy controls. Dimensional assessments of female (Bress and Hajcak 2013; Foti, Kotov, et al. Foti et al. 2011; Nelson, Perlman, et al. 2016) and mixed-sex adolescent cohorts (Bress et al. 2012, 2015; Kujawa et al. 2014), as well as emerging adults (Foti and Hajcak 2009; Umemoto and Holroyd 2017; Weinberg et al. 2015), have reported comparable findings, and similar depression-related reductions in the Fb-P3 amplitude have also been reported (Ait Oumeziane et al. 2019; Luking et al. 2021). However, null results (Padrão et al. 2013) or even potentiated RewP amplitudes have also been described in MDD (Mies et al. 2011; Mueller et al. 2015) or with increasing depressive symptoms (Ait Oumeziane and Foti 2016). Meta-analyses of the consummatory reward literature similarly suggest that the association between changes in ERP indices of reward responding and depression may not necessarily be robust (Clayson et al. 2020) or may depend upon other factors like the task used in the study (Moran et al. 2017) or the age of study participants (Keren et al. 2018).

Beyond the response to reward feedback, a smaller body of research has investigated the FRN as a measure of sensitivity to loss or negative feedback and how individual differences in this process may be associated with depression. Interestingly, unlike the other components, the FRN literature has largely converged around the negativity bias that is observed in depression, such that clinically depressed adolescent girls (Webb et al. 2017) and adults (Cavanagh et al. 2011; Tucker et al. 2003), as well as adults with remitted depression (Santesso et al. 2008), all report enhanced neural responses to loss or negative feedback. Altogether, as with anticipatory processing, the evidence for consummatory processing is mixed. While potentially more robust, this literature does further highlight the need for a holistic approach in examining reward processing.

Few studies to date have taken such a holistic approach by examining both anticipatory and consummatory reward processing simultaneously (see Glazer et al. (2018) and Meyer et al. (2021) for relevant commentary). Two such studies, Novak et al. (2016) and Umemoto and Holroyd (2017), found that higher depressive symptoms were associated with only diminished consummatory processing (i.e., RewP amplitude), whereas anticipatory processes appear unrelated. It is worth noting, however, that in the instance of the former study, this was only true when accounting for individual differences in impulsivity as a moderator. More recently, Ait Oumeziane et al. (2019), Luking et al. (2021), and Thompson et al. (2023) each observed that indices of both anticipatory (i.e., the Cue-P3 or stimulus preceding negativity) and consummatory (i.e., the RewP or Fb-P3) reward processing were independently associated with increased depressive symptoms in adolescents. Collectively, these few studies generally suggest that changes in anticipatory and consummatory reward processing are separately associated with depression but may be moderated by other factors.

Although there is no clear explanation for the mixed findings in the literature, some researchers have offered evidence for moderating factors, including those related to participant (e.g., impulsivity; Novak et al. 2016) or task design (e.g., gambling vs. other; Moran et al. 2017). Interestingly, little research has specifically examined whether participant sex plays a moderating role in this association. While most studies reported here were conducted with mixed-sex samples, few of them approach an even distribution, with 20 of 34 studies reporting at least 60% female participants and only 3 reporting 60% or more male participants. Moreover, across the studies reviewed above, approximately 70% of the included participants were identified as female, aligning with the demographics reported in the meta-analyses of Keren et al. (2018). Sample composition is often skewed toward females in studies of internalizing disorders, consistent with the epidemiological evidence that women are more likely to garner a diagnosis of depression (Daly 2022). Interestingly, there is increasing evidence of the sex-specificity of associations among ERPs and internalizing disorders. This has most notably been reported for anxiety, where meta-analyses have found that frequently reported associations of the error-related negativity and anxiety are specific to females, demonstrating the importance of examining sex as a moderator (Moser et al. 2016). More recent literature has observed similar effects in depression (Harold et al. 2023), and this potential moderation is further emphasized by the heterogeneous or absent findings observed in studies with mixed-sex samples (e.g., Ait Oumeziane and Foti 2016; Nelson, Kessel, et al. 2016). And so, given these noted effects, it is important for research to examine participant sex as a moderator of the relationship between neural indices of reward processing indices and depression.

1.4 | The Present Study

Understanding if and how anticipatory and consummatory reward processes intersect as multiple vulnerabilities that together predict symptom severity and the role of participant sex in this association may provide a more nuanced insight into depressive psychopathology than investigating each process separately. As such, the first aim of the present study was to use ERP indices of both anticipatory and consummatory reward processing to concurrently examine how these processes predict self-reported depressive symptoms in a sample of $n = 309$ late adolescents/emerging adults.

In line with the existing literature, as we conducted a dimensional assessment of depressive symptoms in a community sample, we hypothesized that higher self-reported symptoms would (1) not be associated with the amplitude of the Cue-P3 but (2) would be associated with lower CNV amplitude; (3) be associated with a reduced response to reward, that is, a decreased RewP or Fb-P3 to positive feedback amplitude; and (4) be associated with a potentiated response to negative feedback, that is, an increased FRN or Fb-P3 to negative feedback amplitude. We

then sought to evaluate if/how these associations are moderated by participant sex (defined as sex assigned at birth). Based on an increasing body of literature demonstrating sex-specific effects among ERPs and internalizing symptoms, we hypothesized that the associations among reward processing ERPs and depressive symptoms would only be observed for female participants.

2 | Methods

2.1 | Participants

The data used in the present study were captured from $n = 358$ participants (52.23% female) aged $M = 18.55$, SD = 0.49 years who were part of the Family Life Project (FLP) and completed the most recent round of data collection. Briefly, the FLP was an epidemiological study that followed $n = 1292$ children and their families recruited at the time of the child's birth (approximately 2003), in regions of Pennsylvania ($n = 519$) and North Carolina ($n = 773$), to investigate the effects of poverty and rurality on early child development; additional details regarding the recruitment and maintenance of the FLP sample have been reported elsewhere (Vernon-Feagans et al. 2013).

Between 2022 and 2023, $n = 358$ participants agreed to participate in a clinic visit. EEG data were available for $n = 309$ of these participants, who ranged in age at the time of assessment from 17.52 to 19.66 years ($M = 18.55$, SD = 0.49). Of these participants, 50.16% were identified as female at birth, $n = 92$ as African American or Black, $n = 193$ as White or Caucasian, and $n = 24$ were identified as bi- or multi-racial. Of the $n = 49$ participants (65.31% female; $n = 16$ African American or Black, $n = 29$ White, and $n = 4$ bi- or multi-racial) who completed the clinic visit but did not have usable EEG data, the data were missing for the following reasons: $n = 7$ had no data recorded due to equipment failure; $n = 9$ had no data after processing due to poor or missing mastoid electrodes; and $n = 33$ elected not to complete the EEG component of the visit. Finally, it should be noted that the $n = 49$ participants without EEG data (either unusable or not recorded) were more likely to be female ($\chi^2(1) = 6.99$, $p = 0.008$) but did not differ with respect to their age at the clinic visit, racial identity, or self-reported depression scores (p's ≥ 0.85) when compared to the $n = 309$ participants with EEG data.

2.2 | Study Procedure

All study procedures were approved by the NYU Langone School of Medicine Institutional Review Board, with reliance from the IRBs of both The Pennsylvania State University and the University of North Carolina, Chapel Hill.

Due to restrictions during the Covid-19 outbreak, initial waves of data collection were conducted entirely online. All FLP participants were asked to complete a total of three online surveys distributed between 2020 and 2023. Beginning in 2022, participants were invited to complete an in-person clinic visit that consisted of multiple assessments including anthropometric measurement, cognitive function assessment, collection of biological samples, and an EEG protocol. Of note, this wave of data collection represented the first time in the history of the FLP study that participants were asked to travel to a data collection site rather than having research assistants conduct assessments in their home. To facilitate this, clinics were established within one of the original recruitment counties in Pennsylvania and North Carolina. Participants were provided gift cards for fuel prorated by the approximate mileage they would be required to travel. In addition, participants were paid $200 for completing this visit, and an additional $125 if they consented to providing biological samples. As described below, for the EEG assessment participants were told that they would be playing a game and had the opportunity to win "up to $50" in addition to their other payments. Informed participant consent, and if appropriate, assent, was obtained electronically prior to the clinic visit.

2.3 | Patient-Reported Outcomes Measurement Information System (PROMIS)—Depressive Symptoms

Depression symptoms were assessed in each of the three online surveys using an age-appropriate form (i.e., if a participant completed the survey prior to their 18th birthday they completed the pediatric version of the scale, otherwise they completed the adult scale) of the PROMIS—Depressive Symptoms scale. These scales were initially developed based on a sample representative of the general US population (Irwin et al. 2010; Pilkonis et al. 2011) and have since been validated across additional clinical (e.g., pediatric oncology; Reeve et al. 2020) and general population samples (Klaufus et al. 2021; Pilkonis et al. 2014). They have been shown to possess excellent reliability (> 0.85; Irwin et al. 2010) and moderate-to-high internal consistency and test–retest reliability (Varni et al. 2014). Participants were asked to rate items on a 5-point ordinal rating scale of "Never," "Rarely," "Sometimes," "Often," and "Always". A total of 8 items (possible score range of 8–40) assessed symptoms of depression over the past 7 days, including negative mood (e.g., sadness, guilt), views of self (e.g., self- criticism, worthlessness), social cognition (e.g., loneliness, interpersonal alienation), and decreased positive affect and engagement (e.g., loss of interest, meaning, and purpose). Given the differences in the timing of online survey completion and the clinic visits, to best estimate the concurrent experience of depression, for each participant, we used the PROMIS data from the online survey that was closest in time to their EEG assessment. The majority of participants completed the symptom questionnaire prior to the EEG assessment (median: −130 days; IQR: −271.5 to −31.5 days; range: −1050 to 232 days).

Of the 309 participants with EEG data, self-reported depressive symptom scores were available for $n = 292$ participants, with a sample average score of $M = 18.00$, SD = 8.83, and scores across the full possible range of the scale (8–40). PROMIS measures generate T-scores that are based on responses provided by a reference population and have a mean of 50 and standard deviation of 10. The average symptom score of our sample corresponds to a T-score of $M = 55.7$, SD = 3.1 (range 35.30–82.40) a value slightly above the population norm (i.e., a T-score of 50). Notably, there was a significant difference in depression scores between female ($M = 20.07$, SD = 8.76; T-score = 57.9) and male ($M = 15.95$, SD = 8.44; T-score = 53.3) participants ($t = 4.09$, $p < 0.001$).

2.4 | EEG Protocol and Recording

EEG data were recorded using a BioSemi ActiveTwo system (BioSemi, Amsterdam, Netherlands) with DC amplifiers set at a gain of 1 and a 24-bit A-D conversion resolution. This system was paired with a 34-lead unipolar montage (which included additional FC_z and I_z electrodes) of sintered silver electrodes placed symmetrically at standard extended 10–20 locations over the whole head using BioSemi electrode caps. Additional electrodes were placed on the left and right mastoid processes, the left suborbital ridge below the pupil level, and on the left radial styloid process (to capture ECG data that are not analyzed in the present work). Data for most recordings were digitized at 1024 Hz according to the BioSemi zero reference principle (individual electrode voltage is quantified relative to the common mode sense and driven right leg loop). However, $n = 33$ recordings were digitized at 512 Hz, and so in pre-processing, all recordings were downsampled to 512 Hz. Throughout the recordings, electrode DC offsets were maintained below 40 μV as a substitute for traditional impedance measures.

During the electrode cap setup, RAs used graduated plastic syringes to apply conductive gel into each electrode holder and recorded the total volume of gel (mL) used when placing the EEG cap and electrodes on each participant's head. The purpose behind this measurement was to quantify individual differences in hair volume, which may introduce systematic bias in ERP amplitudes and account for these in our analysis (Lees et al. 2024). After placing the cap and electrodes, the participant was asked to sit quietly for a 3-min baseline recording not included in the present work. Following this initial baseline, participants completed the electrophysiological Monetary Incentive Delay task (eMID; Broyd et al. 2012), which was presented using Presentation (Version 21.21.0, Build: 06.06.19). After the completion of the eMID task, participants were paid the full $50, and the clinic visit was concluded.

2.4.1 | Electrophysiological Monetary Incentive Delay Task

The MID task was originally developed to separate the neural response to different stages of reward processing in fMRI research (Knutson et al. 2000, 2001), with subsequent studies adapting the protocol for EEG (Broyd et al. 2012). The general structure of each trial is presented in Figure 1 and proceeds as follows: participants were first presented with a cue stimulus (either a blue plus sign, minus sign, or circle) that denoted the nature of the trial (i.e., gain—where participants are able to win points, loss—where participants may lose points, neutral—where participants neither gain nor lose points but still receive true feedback) for 250 ms, followed approximately 2 s (randomly jittered ±50 ms) later by a target stimulus (a white square) to which participants were required to respond via a single-button press as quickly as possible. Following a second static 1500-ms ISI, participants were shown a feedback stimulus indicating the outcome of the trial for 1500 ms, and this was followed by an inter-trial interval of 1000 ms.

Regardless of the trial type, the feedback presented on each trial was determined by comparing the participants' response time on that trial to a threshold time. If the trial response time was below the threshold value, participants received positive feedback (i.e., a green check), but if the response time was above the threshold value or participants responded prior to the presentation of the target, they received negative feedback (i.e., a red X). The implications of a positive performance feedback varied by trial type: Gain trials = won points, Loss trials = did not lose points; Neutral trials = no effect on points. Similarly,

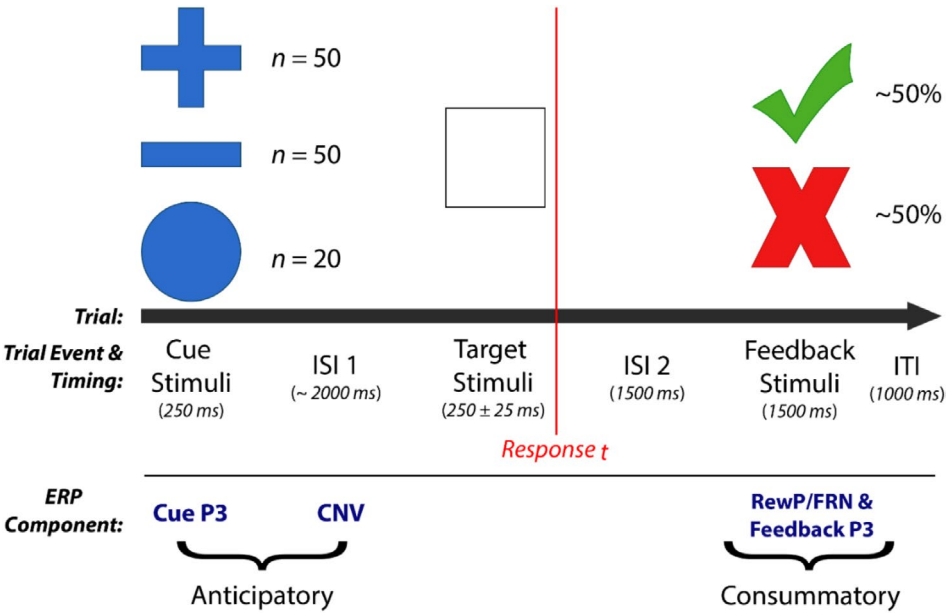

FIGURE 1 | Trial format of the electrophysiology monetary incentive delay (eMID) task. On each trial, participants are initially presented with a cue stimulus indicating the trial type (i.e., gain, loss, neutral) and possible outcomes (i.e., gain points, fail to gain points, lose points, avoid losing points, no loss or gain). Following the first interstimulus interval (ISI), participants were shown a target stimulus to which participants were required to respond as quickly as possible. After a second ISI, participants were shown a feedback stimulus indicating the outcome of the trial. CNV, contingent negative variation; FRN, feedback-related negativity; RewP, reward positivity.

negative performance feedback resulted in: Gain trials = did not win points; Loss trials = lost points; Neutral trials = no effect on points.

Additionally, the threshold value was dynamically adjusted between each trial based upon the outcome of the previous trial, that is, if participants received positive feedback, the threshold would be lowered by 25 ms to increase the difficulty of the subsequent trial, but if they received negative feedback, it would be increased by 25 ms to make the subsequent trial easier. This adjustment was implemented to force an individual error rate of approximately 50% and balance the number of successful (i.e., positive feedback) and unsuccessful (i.e., negative feedback) trials.

Prior to starting the task, research staff provided a detailed explanation of the task, ensuring participants understood the difference in the trial type cues (i.e., the circle indicated neutral trials, the plus sign indicated gain trials, and the minus sign indicated loss trials). Most importantly, participants were informed that their performance (i.e., successfully winning points and avoiding losing points) was tied to the size of the cash prize they would receive upon completing the task (all participants received the full $50 for completing the EEG protocol regardless of their task score). After receiving these instructions, participants completed a short practice round of 15 trials (5 trials of each type) to familiarize themselves with the task. These trials were also used to establish the initial length of the dynamic response time threshold. Participants were free to complete this practice round as many times as were necessary to feel comfortable.

Following the practice trials, participants completed the full eMID task. In the task, participants completed a total of 120 trials (50 gain, 50 loss, 20 neutral trials) split as evenly as possible into 3 blocks: block 1 consisted of 17 gain trials, 16 loss trials, and 7 neutral trials; block 2 consisted of 17 gain trials, 17 loss trials, and 6 neutral trials; and block 3 consisted of 16 gain trials, 17 loss trials, and 7 neutral trials. Individual trials were presented in a manner such that no two successive trials were identical. Additionally, between blocks, a 1-min break was programmed to provide participants with a short rest. During this rest period, participants were provided general feedback on their performance in the form of a score that was displayed on the monitor. After completing the third block of trials, participants were shown a screen that presented their final score.

2.5 | EEG Data Processing

EEG pre-processing was completed in Matlab R2019a using the EEGLAB (v2021.1; Delorme and Makeig 2004) and ERPLAB (v8.30; Lopez-Calderon and Luck 2014) toolboxes. As a first step, the raw eMID task data were imported, and channel coordinate information was added. Following this, as previously mentioned, the recording was downsampled to 512 Hz, temporarily re-referenced to Cz, and then parsed through a bad channel detection algorithm that is embedded in the FASTER plug-in (v1.2.3b; Nolan et al. 2010). This algorithm uses three parameters: (1) the mean Pearson correlation between the channel of interest and other channels, (2) the variance of the channel of interest relative to other channels, and (3) the Hurst exponent to identify and flag bad channels using an associated *Z*-score and a threshold value of 3. Identified bad channels were removed from the recording before continuing with the data processing; on average, $M = 2.31$, SD = 0.95 channels were removed. As a note, given the size and relatively low density of our electrode montage, we chose not to interpolate removed channels.

After removing bad channels, the recordings were DC-corrected and subsequently band-pass-filtered using a 2nd-order Butterworth infinite impulse response (IIR) filter with a 12 dB per octave roll-off and half-amplitude cut-off values of 0.1 and 30 Hz. After filtering, data were re-referenced to the average of the mastoid channels (M_1 and M_2) and then decomposed using Independent Component Analysis (specifically the implementation via the "*runica*" EEGLab function). Using the ICLabel plug-in (v1.3; Pion-Tonachini et al. 2019), the computed independent components were classified, and those marked as eye- and muscle-activity artifacts, as well as channel/line noise with a classification threshold of 90% or greater, were removed. For some recordings, this threshold failed to identify any components to remove, and in these instances ($n = 9$), the ICA decomposition was manually reviewed for artifactual components. On average, $M = 1.73$, SD = 0.77 components were removed; of these, 98.80% were classified as eye activity, 0.45% as channel noise, and 0.75% as muscle activity.

Following the removal of artifactual ICA components, the recording was separately segmented around the Cue stimuli and the feedback stimuli; the specific details regarding each component and its derivation are described in the ERP measures and quantification section. After segmentation, all segments containing a voltage step > 100 μV between successive 200-ms windows (with a 50% overlap), a 30 μV or greater change between sampling points, or a voltage value beyond −100 to 100 μV were marked as artifacts and removed.

2.5.1 | ERP Measures and Quantification

Given our focus on reward processing, neutral and loss trials were not examined in the current analysis. Using the gain trials, the Cue-P3 and CNV were derived as indices of anticipatory reward processing. For the Cue-P3, data were segmented from −200 to 1000 ms around the gain-cue stimuli and baseline-corrected across the −200- to 0-ms window. Then, the Cue-P3 was quantified at Pz as the mean activity across the 250- to 550-ms post-stimuli window. After removing artifactual trials, the mean number of trials used in deriving the Cue-P3 was $M = 40.09$, SD = 10.95. To derive the CNV, data were segmented from −200 to 2500 ms around the gain-cue stimuli, baseline-corrected across the −200- to 0-ms pre-stimuli window, and then averaged. After averaging, the CNV was quantified at the Cz electrode as the mean amplitude over the 1750–1950-ms post-stimulus window, which coincides with the 200 ms preceding the participants' response. After removing artifactual trials, the mean number of trials used in deriving the CNV was $M = 34.06$, SD = 13.65.

To index consummatory reward processing, separate ERPs for both the positive and negative feedback stimuli were derived.

First, data were segmented from −200 to 1000 ms around the feedback stimuli and baseline-corrected across the −200 to 0-ms window. The segmented data were then entered into a temporal principal component analysis (PCA) to determine the timing of early (i.e., the RewP/FRN) and late (i.e., Feedback-P3) components (see supplement for more details). This analysis identified two temporal components that aligned with the ERP components. The first, TF02, was a positive deflection peaking at 280 ms that resembled the RewP/FRN and represented 19.74% of the variance. The second component, TF03, resembled the Fb-P3 and was a positive deflection that represented 13.82% of the variance with a peak at 444 ms. The peak latencies of these temporal components were then used to define the measurement windows for our components. For the positive feedback response, the initial evaluative response to reward (i.e., the RewP) was quantified as the mean activity across the 230–330-ms post-feedback stimuli window at the Cz electrode. In addition, the later reward salience response was indexed by the Fb-P3 and quantified as the mean activity across the 344–544-ms post-positive feedback stimuli window at the Pz electrode. The FRN and Fb-P3 for negative feedback were both derived in the same way but using data segmented around the negative feedback stimulus. After removing artifactual trials, the mean number of trials used in deriving the RewP/Fb-P3 for positive feedback was $M = 19.33$, SD = 7.54, and the mean number of trials for the FRN/Fb-P3 for negative feedback was $M = 16.97$, SD = 7.45. The mean amplitudes for each ERP component can be found in Table 1, and the grand average waveforms and associated topographies for the anticipatory (i.e., the Cue-P3 and CNV) and consummatory (the RewP/FRN and Fb-P3) ERP components are presented in Figures 2 and 3, respectively.

Finally, while not a primary focus of the present study, residualized difference waves that contrast conditions (e.g., positive minus negative feedback as is common for the RewP) were generated, and zero-order bivariate correlations among these components and depressive symptom scores were examined and found to be non-significant (Table S1). As these were non-significant, and previous work has suggested that subtraction-based difference scores have the potential to mask suppressor effects when investigating individual differences in psychopathology (Meyer et al. 2017), they were not included in any further analysis.

2.6 | Data Analysis

Zero-order Pearson's correlations among all variables of interest were examined to characterize simple effects. Subsequently, a series of ordinary least squares regression models were examined predicting the raw self-reported depression symptom scores. Our first set of models examined the main effects of reward processing ERPs in predicting depression scores and if these associations were moderated by participant sex assigned at birth (dummy coded such that female was coded 0 and male was 1). These models included participant sex, cue-P3 amplitude, CNV amplitude, either the RewP/FRN or Fb-P3 amplitudes, and the interaction terms between participant sex and each ERP amplitude. Additionally, participant age at recording and the volume of electrode gel used in setting up the EEG cap were both included as covariates. We estimated separate models for the RewP/FRN and Fb-P3 to ensure that each described stage of reward processing (i.e., cue evaluation, motivated action toward reward, and response to reward) was indexed by only a single ERP component.

Based on the results of these first models, we then estimated a second set of models, separated by participant sex, to examine these associations separately within each sex. These models were estimated by including only the ERPs implicated in the prior models and the interactions among these components, as well as participant age at recording and the volume of electrode gel as covariates.

TABLE 1 | Descriptive statistics of zero-order bivariate correlations among electrode gel volume, depressive symptom score, and ERP amplitudes.

	1	2	3	4	5	6	7	8	9
1. Age	—								
2. Gel volume	0.30***	—							
3. Depression score	0.08	−0.09	—						
4. Cue-P3	0.01	−0.05	−0.02	—					
5. CNV	−0.03	0.06	0.03	0.40***	—				
6. RewP	−0.02	−0.15*	−0.03	0.14*	−0.28***	—			
7. FRN	−0.05	−0.22***	0.03	0.07	−0.33***	0.74***	—		
8. $\text{Fb-P3}_{\text{Positive}}$	−0.03	−0.01	0.02	0.17**	−0.16*	0.47***	0.31***	—	
9. $\text{Fb-P3}_{\text{Negative}}$	−0.01	−0.15*	0.06	0.09	−0.24***	0.28***	0.47***	0.62***	—
Mean	18.55	15.76	18.00	5.01	−5.30	16.63	14.18	7.96	8.24
SD	0.49	10.02	8.83	4.38	5.98	7.66	7.51	5.68	5.75

Abbreviations: CNV, contingent negative variation; Fb-P3, feedback-P3; FRN, feedback-related negativity; RewP, reward positivity.
*$p < 0.05$;
**$p < 0.01$;
***$p < 0.001$.

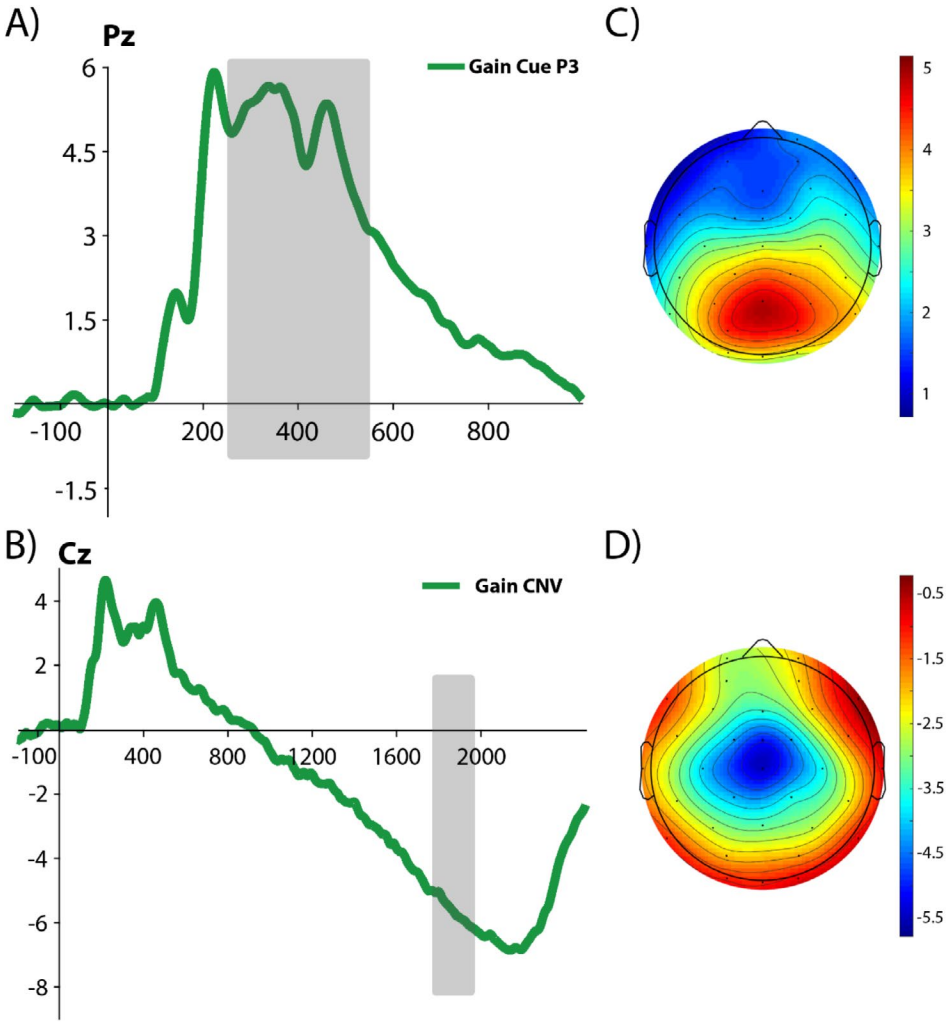

FIGURE 2 | Grand average waveforms for the Cue-P3 and CNV anticipatory reward processing ERP components. Plot A presents the grand average Cue-P3 waveform as derived at the Pz electrode, and Plot B presents the grand average CNV waveform as derived at the Cz electrode. In both plots, the waveform for the gain condition is presented, and polarity is such that positive values are plotted up. The shaded region indicates the window of measurement for each ERP component: 250–550 and 1750–1950 ms post-stimulus for the Cue-P3 and CNV, respectively. Plots C and D present the scalp topography across the measurement windows of the Cue-P3 and CNV, respectively. CNV, contingent negative variation.

Ordinary least-squares regression models were estimated in R using the "*stats*" package (R Core Team 2013), and model figures were created using the "*ggplot2*" package (Wickham 2016). Prior to being entered into the models, age at clinic visit, gel volume, and the ERP amplitude parameters were all mean-centered (using either the full-sample (Table 2) or sex-specific means (Table 3)). Lastly, statistical significance was evaluated at $p < 0.05$.

3 | Results

Zero-order correlations among the ERP amplitudes of interest, participant age, depression score, and EEG gel volume are reported in Table 1. As expected, most of the ERPs were significantly correlated (r's $\geq |0.14|$, p's ≤ 0.03) with one another, with the notable exception of the Cue-P3, which was not associated with the FRN or Fb-P3 for negative feedback amplitude (p's ≥ 0.27), suggesting that an individual's initial attention toward potential reward is not associated with their response to negative feedback and/or loss sensitivity. Moreover, it is worth noting that while the RewP and Fb-P3 for positive feedback ($r = 0.47$, $p < 0.001$) and the FRN and Fb-P3 for negative feedback ($r = 0.47$, $p < 0.001$) were positively associated, there is also variance unique to each component, which suggests that, in some capacity, they represent unique processes. As such, the regression models were estimated separately for both the RewP/FRN and Fb-P3.

Unexpectedly, depression scores were not significantly associated with any of the investigated ERPs (p's ≥ 0.45), suggesting that, at a simple level, depression scores are not associated with changes in the separate indices of reward processing in this sample of emerging adults. It is also worth noting that the RewP ($r = -0.15$, $p = 0.02$), FRN ($r = -0.22$, $p < 0.001$), and Fb-P3 for negative feedback ($r = -0.15$, $p = 0.02$) were significantly correlated with the volume of electrode gel used, confirming its inclusion as a covariate in our models.

3.1 | Reward Processing and Depression

The first aim of the present study was to concurrently examine if anticipatory and consummatory ERP indices of reward processing

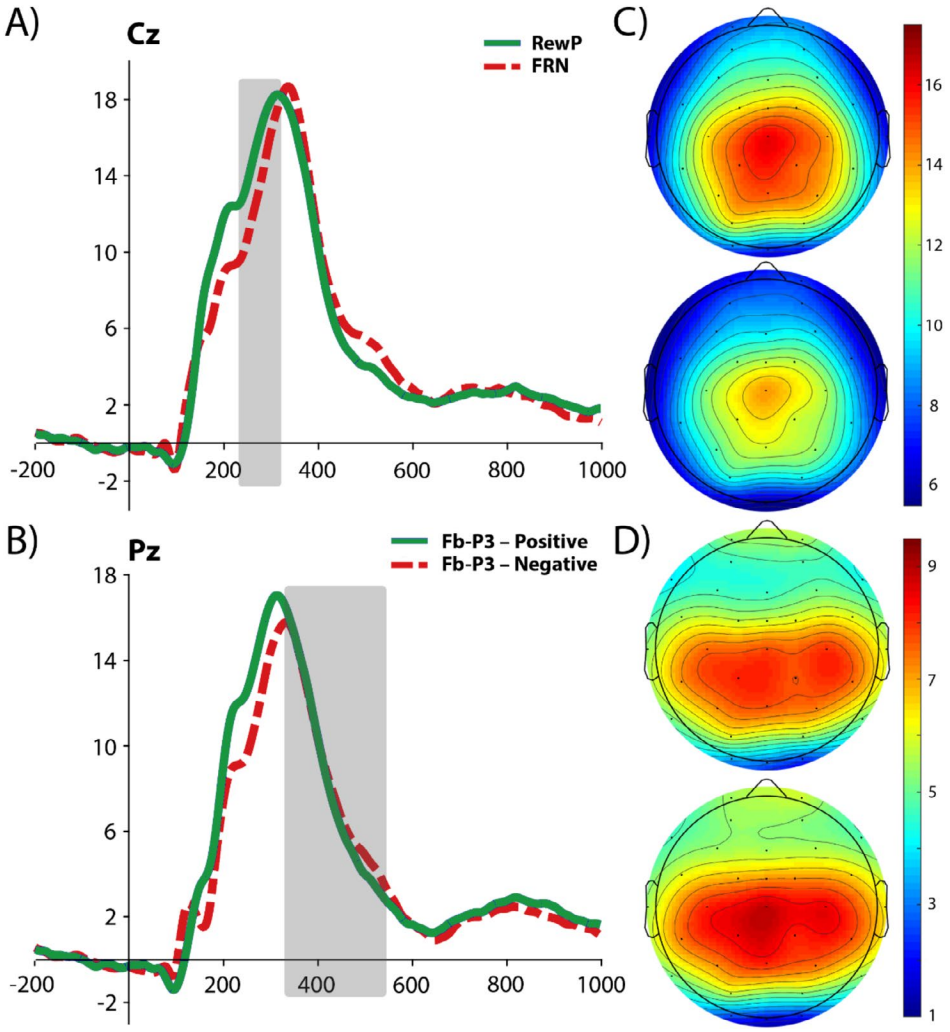

FIGURE 3 | Grand average waveforms for the RewP/FRN and feedback-P3 consummatory reward processing ERP components. Plot A presents the grand average RewP and FRN waveforms as derived at the Cz electrode, and Plot B presents the grand average feedback-P3 waveforms as derived at the Pz electrode. In both plots, the waveforms for both the positive (green, solid-line) and negative (red, dashed-line) feedback conditions are presented, and polarity is such that positive values are plotted up. The shaded region indicates the window of measurement for each ERP component: 230–330 and 344–544 ms post-stimulus for the RewP/FRN and Feedback-P3, respectively. Plot C presents the scalp topography across the measurement window for the RewP (top) and FRN (bottom), and Plot D presents the topography for the positive (top) and negative (bottom) feedback-P3 across its measurement window. Fb-P3, feedback-P3; FRN, feedback-related negativity; RewP, reward positivity.

are related to depression symptom scores. Additionally, we also wanted to see if these associations were moderated by sex. The models evaluating these aims are presented in Table 2. Across both models, the main effect for participant sex was significant (t's ≤ -2.61, p's ≤ 0.01) indicating that, on average, male participants scored an estimated 3.03–3.21 points lower than their female counterparts. In line with our hypotheses, the main effect for the anticipatory Cue-P3 was non-significant (p's ≥ 0.44). Interestingly, the main effects for the CNV were non-significant (p's ≥ 0.14) in contrast to our hypotheses. Moreover, the interactions between the anticipatory ERP amplitudes and participant sex were also non-significant (p's > 0.41). Collectively, these results suggest that, on average, anticipatory reward processes are not associated with depressive symptom scores.

With respect to consummatory reward processing, contrary to our hypotheses, the main effects for the RewP and the FRN (left column of Table 2) were non-significant (p's > 0.25) as were their interaction with participant sex (p's > 0.65), indicating that on average, the initial response to reward was not associated with depressive symptom scores. However, with respect to later stages of consummatory reward processing (right column of Table 2), the main effect for Fb-P3 amplitude for positive feedback was significant ($b = -0.56$, $p = 0.006$), indicating that for female participants a diminished response to positive feedback was associated with an elevated depressive symptom score. Moreover, the interaction term between Fb-P3 amplitude for positive feedback stimuli and participant sex was also significant ($b = 0.83$, $p = 0.004$) indicating that being male moderates this association. To further probe this effect, we plotted the sex-specific regressions (Figure 4A) and conducted a simple slope analysis, finding that the response to positive feedback was negatively associated with depression symptoms for female participants ($b = -0.56$, $t = -2.77$, $p = 0.006$) and non-significant for male participants ($b = 0.26$, $t = 1.33$, $p = 0.18$).

TABLE 2 | Results from the ordinary least-squares regression models examining how reward processing ERPs predict depressive symptoms.

	RewP/FRN			Fb-P3	
Fixed effects	***b* (SE)**	***t***	**Fixed effects**	***b* (SE)**	***t***
Intercept	19.89 (0.86)	23.01***	Intercept	19.94 (0.88)	22.55***
Age	1.36 (1.35)	1.01	Age	0.99 (1.31)	0.76
Gel volume	−0.10 (0.06)	−1.54	Gel volume	−0.11 (0.06)	−1.73†
Sex (male)	−3.41 (1.21)	−2.83**	Sex (male)	−3.21 (1.23)	−2.61**
Cue-P3	−0.21 (0.27)	−0.77	Cue-P3	−0.12 (0.24)	−0.48
CNV	0.23 (0.19)	1.21	CNV	0.25 (0.17)	1.47
RewP	−0.17 (0.19)	−0.92	$\text{Fb-P3}_{\text{Positive}}$	−0.56 (0.20)	−2.77**
FRN	0.20 (0.18)	1.15	$\text{Fb-P3}_{\text{Negative}}$	0.45 (0.23)	1.94†
Sex × Cue-P3	0.16 (0.35)	0.45	Sex × Cue-P3	−0.02 (0.33)	−0.07
Sex × CNV	−0.21 (0.26)	−0.83	Sex × CNV	−0.16 (0.24)	−0.67
Sex × RewP	0.10 (0.24)	0.40	$\text{Sex} \times \text{Fb-P3}_{\text{Positive}}$	0.83 (0.28)	2.91**
Sex × FRN	−0.11 (0.25)	−0.46	$\text{Sex} \times \text{Fb-P3}_{\text{Negative}}$	−0.50 (0.29)	−1.75†
R^2, *p*	0.07	0.22	R^2, *p*	0.10	0.02
df, *F*	11, 195	1.31	*df*, *F*	11, 195	2.08

Note: Unstandardized coefficients. Prior to being entered into the model, all continuous variables (age, gel volume, and ERP amplitudes) were mean-centered using the full-sample average, and sex was dummy-coded such that female was set to 0 to serve as the reference group. Models were fit with complete data from $n = 205$ subjects.
Abbreviations: CNV, contingent negative variation; Fb-P3, feedback-P3; FRN, feedback-related negativity; RewP, reward positivity.
†$p < 0.1$;
**$p < 0.01$;
***$p < 0.001$.

TABLE 3 | Results from the ordinary least-squares regression models examining how the Fb-P3 predicts depressive symptoms in female and male participants separately.

	Females ($n = 101$)		Males ($n = 107$)	
Fixed effects	***b* (SE)**	***t***	***b* (SE)**	***t***
Intercept	20.03 (0.93)	21.58***	16.62 (0.97)	17.20***
Age	1.93 (1.77)	1.09	−0.61 (2.01)	−0.30
Gel volume	−0.05 (0.09)	−0.49	−0.12 (0.08)	−1.47
$\text{Fb-P3}_{\text{Positive}}$	−0.52 (0.20)	−2.65**	0.27 (0.20)	1.38
$\text{Fb-P3}_{\text{Negative}}$	0.38 (0.21)	1.75†	−0.11 (0.18)	−0.62
$\text{Fb-P3}_{\text{Positive}} \times \text{Fb-P3}_{\text{Negative}}$	−0.00 (0.02)	−0.18	−0.00 (0.03)	−0.08
R^2, *p*	0.10	0.08†	0.04	0.52
df, *F*	5, 95	2.05	5, 101	0.84

Note: Unstandardized coefficients. Prior to being entered into the model, all continuous variables (age, gel volume, and ERP amplitudes) were mean-centered using the sex-specific average.
Abbreviation: Fb-P3, feedback-P3.
†$p < 0.1$;
**$p < 0.01$;
***$p < 0.001$.

Additionally, the main effect for the Fb-P3 response to negative feedback was very near to significant ($b = 0.45$, $p = 0.05$), indicating that in female participants, a greater response to negative feedback was associated with greater depressive symptoms. Moreover, the interaction term between Fb-P3 amplitude for negative feedback stimuli and participant sex also trended toward significance ($b = -0.50$, $p = 0.08$), indicating that this association differed for male participants. Follow-up probes (Figure 4B)

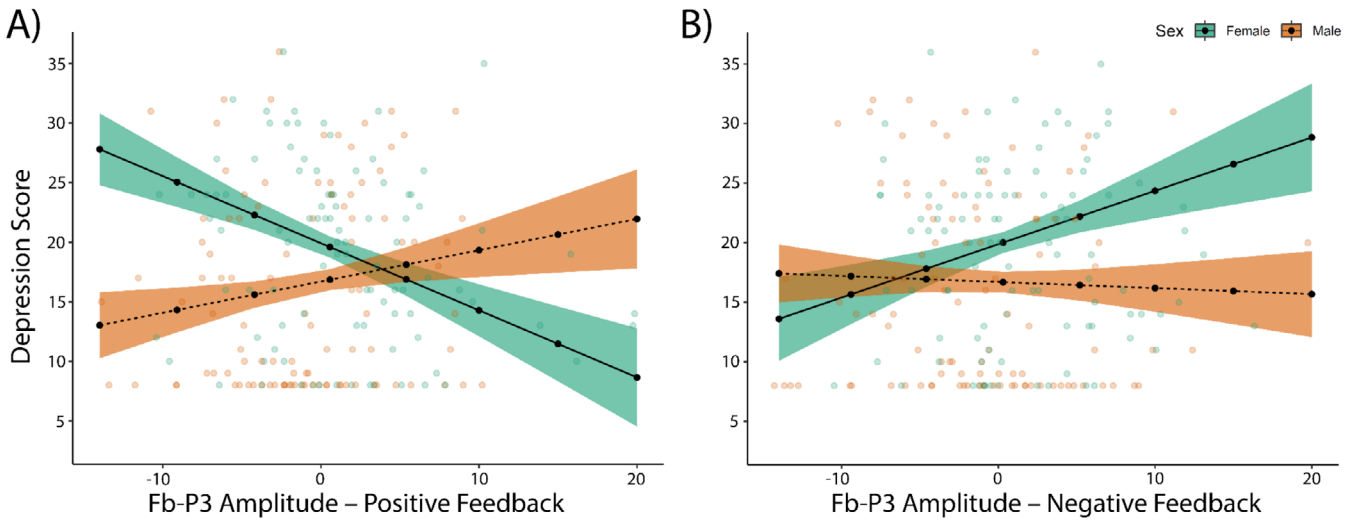

FIGURE 4 | Sex-specific associations between feedback-P3 amplitude to positive and negative feedbacks and depressive symptom scores. Plot A presents the associations between depressive symptom scores and the amplitude of the feedback-P3 ERP to positive feedback, and Plot B presents the associations between depressive symptom scores and the amplitude of the feedback-P3 ERP to negative feedback. In both plots, the association is presented separately for male (orange, dashed-line) and female participants (green, solid-line). Together, these plots indicate that only female participants demonstrated associations between higher depressive symptom scores and reduced responsivity to positive feedback and greater responsivity to negative feedback.

again revealed that the association between the response to negative feedback and depressive symptoms (Figure 4B) was very near to significant and positive for female participants ($b = 0.45$, $t = 1.94$, $p = 0.05$) and non-significant for male participants ($b = -0.05$, $t = -0.30$, $p = 0.76$). Together, the combined results of these models suggest that changes in the later stages of consummatory reward processing are associated with depressive symptom scores but only in young women.

3.2 | Multidimensional Reward Processing in Depression

As the significant effects presented in Table 2 were confined to the model including the Fb-P3, we restricted our subsequent analysis to this later stage of consummatory processing. Moreover, as this first model observed separate effects for male and female participants, to ensure parsimony, our subsequent analyses estimated sex-specific models (Table 3).

With respect to female participants (Table 3; left column), the Fb-P3-specific model retained the significant main effect for the response to positive feedback ($t = -2.65$, $p = 0.009$) and a trending effect for negative feedback ($t = 1.75$, $p = 0.08$) that was observed in the prior model. Notably, the interaction term between the two Fb-P3 parameters was not significant ($p = 0.85$), and alongside the main effects indicates that in young women, greater depressive symptoms were independently predicted by a blunted response to reward and a potentiated response to negative feedback. For male participants (Table 3; right column), the model revealed no association between positive and negative Fb-P3 amplitudes, including their interaction term, and depressive symptom scores (p's ≥ 0.17), indicating that the depressive symptom scores of these young men were not predicted by individual differences in reward processing.

4 | Discussion

The present study sought to concurrently investigate how anticipatory and consummatory reward processing predict self-reported depression in a sample of late adolescents/emerging adults. Overall, we found that anticipatory reward processes, as indexed by the Cue-P3 and CNV, were not associated with self-reported depressive symptom scores. With respect to consummatory reward, the initial response to reward (as indexed by the RewP/FRN) was surprisingly not associated with depression scores. However, the later stage of consummatory reward processing, as indexed by the Fb-P3, was associated with depressive symptom scores, but only for those participants who were identified as female at birth. These results suggest that depression-related alterations in reward processing could be confined to consummatory processes, and these changes may be specific to women.

With respect to anticipatory processes, the present lack of association between cue-P3 amplitude and depressive symptoms is generally consistent with existing literature. Studies that have reported blunted cue-P3 amplitudes were conducted with adult participants meeting clinical diagnostic criteria for major depression in comparison to healthy controls (Santopetro et al. 2021; White et al. 2021; Zhou et al. 2019). Whereas comparable adult studies examining depression as a continuous dimension tend to generally not detect associations with Cue-P3 (Ait Oumeziane et al. 2019; Novak et al. 2016; Umemoto and Holroyd 2017), two more recent studies in young and middle adolescents have observed reductions in Cue-P3 amplitude (Luking et al. 2021; Thompson et al. 2023). As such, given that we conducted a dimensional assessment of a large community-based mostly adult sample, rather than clinical/diagnostic categorizations, it is not unexpected to have not found any association between the Cue-P3 and depressive symptoms.

The absence of an association between the CNV (representative of the motivated pursuit of reward) and depressive symptoms is contrary to our hypothesis as well as other studies (Ashton et al. 1988, 1994; Van derhasselt et al. 2014) However, given the lack of evidence of an association for the preceding anticipatory process represented by the Cue-P3, it is perhaps not surprising to not detect associations with preparatory mechanisms. Indeed, this is in line with work from Ait Oumeziane et al. (2019) who also found no association between depressive symptoms and CNV amplitude, suggesting that there is no deficit in this aspect of anticipatory reward processing. That said, other studies have observed that the association between anticipatory ERPs and depression may depend on other person-level characteristics such as impulsivity (Novak et al. 2016). It is possible that such characteristics also function as a vulnerability for depression, and hence when combined with altered anticipatory reward processing, an association emerges. Accordingly, future efforts investigating indices of motivated effort toward reward and depression may wish to evaluate and analyze such person-level factors.

Turning to consummatory reward processes, the results associated with the indices of initial response to reward, that is, the RewP and FRN, tell a similar story. In contrast to past literature that observed diminished RewP and potentiated FRN amplitudes with increasing depressive symptoms and/or depression status (Bress et al. 2015; Klawohn et al. 2021; Nelson, Perlman, et al. 2016; Proudfit 2015; Whitton et al. 2016), the present analysis found no such association for either male or female participants. This lack of association suggests that individual differences in evaluating an outcome as better or worse than expected were not associated with self-reported depressive symptoms in our sample. While unexpected considering the breadth of literature implicating changes in the RewP in depression (Proudfit 2015), these results do align with recent meta-analytical results that have suggested that the collective evidence for this association may not be robust (Clayson et al. 2020) or is confined to adolescents and absent in people over the age of 18 years (Keren et al. 2018), like the majority of participants included in the present study.

Interestingly, while these earlier consummatory processes were not associated with depressive symptoms, a later stage indexed by the Fb-P3 was. We observed that with increasing depressive symptoms, the Fb-P3 amplitude was diminished for positive feedback and potentiated for negative feedback in young women, and these associations were non-significant in young men. Invoked as a marker of outcome salience, the Fb-P3 is thought to, separately from the RewP, capture the motivational salience and categorization of feedback and its integration with working memory to maximize future reward (Glazer et al. 2018). And so, in line with past comparable research (Ait Oumeziane et al. 2019; Luking et al. 2021), these associations suggest that while their initial evaluative response to reward remains intact, young women who report higher levels of depressive symptoms weigh positive feedback as less salient and negative feedback as more salient; an interpretation supported by both the diminished response to reward and negativity bias frameworks of depression (Pizzagalli and Roberts 2022; Whitton et al. 2015). It is worth noting that these findings do not imply that depression is associated with both reduced response to reward and increased response to non-reward but rather suggest that either could be sufficient to increase susceptibility to depressive symptoms, at least among females.

Focusing on the sex-specific nature of the present findings, past literature has emphasized the importance of examining the potential moderating effects of participant sex (Gatzke-Kopp 2016). This is particularly important in psychopathology research, given that a greater prevalence of depression has been noted among adolescent and adult women (Daly 2022). Indeed, increasing evidence indicates moderation by sex of the associations between ERP components and symptoms of anxiety and depression (Harold et al. 2023; Moser et al. 2016). It is not clear why males do not demonstrate this association. Despite having a lower average depression score, males did evidence the same full range of depression severity as females, and so sufficient variance was available. Thus, for the same severity of depressive symptoms, a different neural mechanism may underlie depression pathology for males. It could be that males and females manifest anhedonic tendencies in different ways, particularly at subthreshold levels of symptom severity. For instance, males may be more inclined to lose interest in social reward at low levels of depression but continue to engage in the reward domains incorporated in the present experiments (e.g., performance evaluation and monetary reward). Social reward processes are being increasingly recognized as offering unique insights for depression research (Kujawa 2024), and future studies would do well to consider if these processes and their role in depression may differ by sex.

Alternatively, it is possible that biological sex moderates a multifinal phenotype for blunted reward processing such that females are more likely to display depression and withdrawal and males are more likely to display irritability and impulsivity. Indeed, blunted reward processing has been implicated in externalizing behavior problems (Beauchaine and Gatzke-Kopp 2012)—a form of psychopathology more prevalent among males and characterized by impulsivity. With this in mind, wider or more direct assessments of other affective dimensions, like impulsivity, might offer future studies more information regarding the relationship between reward processing and psychopathology in male participants.

4.1 | Limitations and Future Directions

Within our participants, while a full range of self-reported symptom scores on the PROMIS—Depressive Symptoms survey was observed, there was a degree of flooring, whereby the mean and median values are nearer to the minimum possible score than to the middle of the possible range. It is therefore possible that our results would not necessarily directly translate to and/or align with cohorts that report higher depressive symptoms, particularly changes in anhedonia, or those recruited based upon an existing clinical depressive diagnosis. Moreover, our symptom scores and EEG data were temporally separated, and while we did not have a strong hypothesis regarding the temporal direction of causation in the relationship between depression and changes in reward processing, it is worth acknowledging that this could contribute noise to the data.

Furthermore, while the PROMIS questionnaire used in the present study broadly evaluates depression, it does not provide

a nuanced evaluation of the various symptom dimensions of depression. It has been postulated that aberrations in reward processing and anhedonia mark an endophenotype that may underlie depressive symptoms (Pizzagalli 2014; Whitton et al. 2015), and so, it may be that the relationship between reward processing and depression is dependent on a specific symptom experience, and future studies should consider widening the breadth of dimensions of depression they evaluate. In a similar vein, the heterogeneity of depression speaks to the possibility that different neural profiles could be associated with similar (i.e., equifinal) or different (i.e., multifinal) profiles of depressive symptoms. Concurrently examining multiple stages of reward processing is only a first step in investigating this possibility, and future research should consider how to evaluate neural profiles and how these could be used to describe an individual's unique experience of depression.

Lastly, only a single rating of depressive symptoms was used, and this was across two versions of the PROMIS, one pediatric and one adult. While these versions were completed in an age-appropriate manner and evaluate the same construct, it is a difference to be acknowledged. Additionally, neither of the versions captured information regarding lifetime depressive status. Given that reward-related impairments observed in acute MDD persist into remission (Whitton et al. 2016) and symptom severity moderates these changes (Pizzagalli et al. 2008; Whitton et al. 2021), it may be possible that an individual can possess the underlying neural biomarker and/or vulnerability while not reporting current symptoms, thus obscuring the association. Therefore, we would encourage future studies to capture and account for participant lifetime history of depression when investigating changes in reward processing.

4.2 | Conclusions

Overall, in a sample of late adolescents/emerging adults, the association between reward processing and depressive symptoms appears to be confined to the consummatory processes of young women. It may be that this sex-specific association is attributable to an experience of negative affect that centers on sadness and worthlessness and is perhaps more common in women or at least more acceptably expressed by women. Furthermore, it may be that examining other dimensions of negative affectivity, for example, anger and irritability, could be more poignant for male participants or, at the very least, provide an additional description of the relationship between reward processing and depressive symptoms.

Author Contributions

Ty Lees: conceptualization, data curation, formal analysis, methodology, visualization, writing – original draft, writing – review and editing. **Lisa M. Gatzke-Kopp:** conceptualization, funding acquisition, methodology, project administration, resources, supervision, writing – original draft, writing – review and editing.

Acknowledgments

We wish to extend our thanks to the study participants and their families for providing a detailed and extended look at their daily lives and to the many research assistants who helped obtain such rich data.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

References

Ait Oumeziane, B., and D. Foti. 2016. "Reward-Related Neural Dysfunction Across Depression and Impulsivity: A Dimensional Approach." *Psychophysiology* 53, no. 8: 1174–1184. https://doi.org/10.1111/psyp.12672.

Ait Oumeziane, B., O. Jones, and D. Foti. 2019. "Neural Sensitivity to Social and Monetary Reward in Depression: Clarifying General and Domain-Specific Deficits." *Frontiers in Behavioral Neuroscience* 13: 1–16. https://doi.org/10.3389/fnbeh.2019.00199.

American Psychiatric Association. 2022. "Depressive Disorders." In *Diagnostic and Statistical Manual of Mental Disorders*. American Psychiatric Association Publishing. https://doi.org/10.1176/appi.books.9780890425787.x04_Depressive_Disorders.

Ashton, C. H., J. F. Golding, V. R. Marsh, J. W. Thompson, F. Hassanyeh, and S. P. Tyrer. 1988. "Cortical Evoked Potentials and Clinical Rating Scales as Measures of Depressive Illness." *Psychological Medicine* 18, no. 2: 305–317. https://doi.org/10.1017/S0033291700007856.

Ashton, C. H., E. F. Marshall, F. Hassanyeh, V. R. Marsh, and S. Wright-Honari. 1994. "Biological Correlates of Deliberate Self-Harm Behaviour: A Study of Electroencephalographic, Biochemical and Psychological Variables in Parasuicide." *Acta Psychiatrica Scandinavica* 90, no. 5: 316–323. https://doi.org/10.1111/j.1600-0447.1994.tb01600.x.

Beauchaine, T. P., and L. M. Gatzke-Kopp. 2012. "Instantiating the Multiple Levels of Analysis Perspective in a Program of Study on Externalizing Behavior." *Development and Psychopathology* 24, no. 3: 1003–1018. https://doi.org/10.1017/S0954579412000508.

Belden, A. C., K. Irvin, G. Hajcak, et al. 2016. "Neural Correlates of Reward Processing in Depressed and Healthy Preschool-Age Children." *Journal of the American Academy of Child and Adolescent Psychiatry* 55, no. 12: 1081–1089. https://doi.org/10.1016/j.jaac.2016.09.503.

Berridge, K. C., and T. E. Robinson. 2003. "Parsing reward." *Trends in Neurosciences* 26, no. 9: 507–513. https://doi.org/10.1016/S0166-2236(03)00233-9.

Berridge, K. C., T. E. Robinson, and J. W. Aldridge. 2009. "Dissecting Components of Reward: 'Liking', 'Wanting', and Learning." *Current Opinion in Pharmacology* 9, no. 1: 65–73. https://doi.org/10.1016/j.coph.2008.12.014.

Bress, J. N., and G. Hajcak. 2013. "Self-Report and Behavioral Measures of Reward Sensitivity Predict the Feedback Negativity." *Psychophysiology* 50, no. 7: 610–616. https://doi.org/10.1111/psyp.12053.

Bress, J. N., A. Meyer, and G. H. Proudfit. 2015. "The Stability of the Feedback Negativity and Its Relationship With Depression During Childhood and Adolescence." *Development and Psychopathology* 27, no. 4: 1285–1294. https://doi.org/10.1017/S0954579414001400.

Bress, J. N., E. Smith, D. Foti, D. N. Klein, and G. Hajcak. 2012. "Neural Response to Reward and Depressive Symptoms in Late Childhood to Early Adolescence." *Biological Psychology* 89, no. 1: 156–162. https://doi.org/10.1016/j.biopsycho.2011.10.004.

Broyd, S. J., H. J. Richards, S. K. Helps, G. Chronaki, S. Bamford, and E. J. S. Sonuga-Barke. 2012. "An Electrophysiological Monetary Incentive Delay (e-MID) Task: A Way to Decompose the Different Components of Neural Response to Positive and Negative Monetary Reinforcement."

Journal of Neuroscience Methods 209, no. 1: 40–49. https://doi.org/10.1016/j.jneumeth.2012.05.015.

Brunia, C. H. M., G. J. M. van Boxtel, and K. B. E. Böcker. 2011. "Negative Slow Waves as Indices of Anticipation: The Bereitschaftspotential, the Contingent Negative Variation, and the Stimulus-Preceding Negativity." In *The Oxford Handbook of Event-Related Potential Components*, edited by E. S. Kappenman and S. J. Luck. Oxford University Press. https://doi.org/10.1093/oxfordhb/9780195374148.013.0108.

Cavanagh, J. F., A. J. Bismark, M. J. Frank, and J. J. B. Allen. 2011. "Larger Error Signals in Major Depression Are Associated With Better Avoidance Learning." *Frontiers in Psychology* 2: 331. https://doi.org/10.3389/fpsyg.2011.00331.

Clayson, P. E., K. A. Carbine, and M. J. Larson. 2020. "A Registered Report of Error-Related Negativity and Reward Positivity as Biomarkers of Depression: P-Curving the Evidence." *International Journal of Psychophysiology* 150: 50–72. https://doi.org/10.1016/j.ijpsycho.2020.01.005.

Daly, M. 2022. "Prevalence of Depression Among Adolescents in the U.S. From 2009 to 2019: Analysis of Trends by Sex, Race/Ethnicity, and Income." *Journal of Adolescent Health* 70, no. 3: 496–499. https://doi.org/10.1016/j.jadohealth.2021.08.026.

Delorme, A., and S. Makeig. 2004. "EEGLAB: An Open Source Toolbox for Analysis of Single-Trial EEG Dynamics Including Independent Component Analysis." *Journal of Neuroscience Methods* 134, no. 1: 9–21. https://doi.org/10.1016/j.jneumeth.2003.10.009.

Donchin, E. 1981. "Surprise!? Surprise?" *Psychophysiology* 18, no. 5: 493–513. https://doi.org/10.1111/j.1469-8986.1981.tb01815.x.

Foti, D., J. M. Carlson, C. L. Sauder, and G. H. Proudfit. 2014. "Reward Dysfunction in Major Depression: Multimodal Neuroimaging Evidence for Refining the Melancholic Phenotype." *NeuroImage* 101: 50–58. https://doi.org/10.1016/j.neuroimage.2014.06.058.

Foti, D., and G. Hajcak. 2009. "Depression and Reduced Sensitivity to Non-rewards Versus Rewards: Evidence From Event-Related Potentials." *Biological Psychology* 81, no. 1: 1–8. https://doi.org/10.1016/j.biopsycho.2008.12.004.

Foti, D., R. Kotov, D. N. Klein, and G. Hajcak. 2011a. "Abnormal Neural Sensitivity to Monetary Gains Versus Losses Among Adolescents at Risk for Depression." *Journal of Abnormal Child Psychology* 39, no. 7: 913–924. https://doi.org/10.1007/s10802-011-9503-9.

Foti, D., and A. Weinberg. 2018. "Reward and Feedback Processing: State of the Field, Best Practices, and Future Directions." *International Journal of Psychophysiology* 132: 171–174. https://doi.org/10.1016/j.ijpsycho.2018.08.006.

Foti, D., A. Weinberg, J. Dien, and G. Hajcak. 2011b. "Event-Related Potential Activity in the Basal Ganglia Differentiates Rewards From Nonrewards: Temporospatial Principal Components Analysis and Source Localization of the Feedback Negativity." *Human Brain Mapping* 32, no. 12: 2207–2216. https://doi.org/10.1002/hbm.21182.

Gatzke-Kopp, L. M. 2016. "Diversity and Representation: Key Issues for Psychophysiological Science." *Psychophysiology* 53, no. 1: 3–13. https://doi.org/10.1111/psyp.12566.

Glazer, J. E., N. J. Kelley, N. Pornpattananangkul, V. A. Mittal, and R. Nusslock. 2018. "Beyond the FRN: Broadening the Time-Course of EEG and ERP Components Implicated in Reward Processing." *International Journal of Psychophysiology* 132: 184–202. https://doi.org/10.1016/j.ijpsycho.2018.02.002.

Goldstein, R. Z., L. A. Cottone, Z. Jia, T. Maloney, N. D. Volkow, and N. K. Squires. 2006. "The Effect of Graded Monetary Reward on Cognitive Event-Related Potentials and Behavior in Young Healthy Adults." *International Journal of Psychophysiology* 62, no. 2: 272–279. https://doi.org/10.1016/j.ijpsycho.2006.05.006.

Gorwood, P. 2008. "Neurobiological Mechanisms of Anhedonia." *Dialogues in Clinical Neuroscience* 10, no. 3: 291–299. https://doi.org/10.31887/DCNS.2008.10.3/pgorwood.

Haber, S. N., and B. Knutson. 2010. "The Reward Circuit: Linking Primate Anatomy and Human Imaging." *Neuropsychopharmacology* 35, no. 1: 4–26. https://doi.org/10.1038/npp.2009.129.

Hajcak, G., and D. Foti. 2020. "Significance? ... Significance! Empirical, Methodological, and Theoretical Connections Between the Late Positive Potential and P300 as Neural Responses to Stimulus Significance: An Integrative Review." *Psychophysiology* 57, no. 7: e13570. https://doi.org/10.1111/psyp.13570.

Hajcak, G., C. B. Holroyd, J. S. Moser, and R. F. Simons. 2005. "Brain Potentials Associated With Expected and Unexpected Good and Bad Outcomes." *Psychophysiology* 42, no. 2: 161–170. https://doi.org/10.1111/j.1469-8986.2005.00278.x.

Hajcak, G., J. S. Moser, C. B. Holroyd, and R. F. Simons. 2006. "The Feedback-Related Negativity Reflects the Binary Evaluation of Good Versus Bad Outcomes." *Biological Psychology* 71, no. 2: 148–154. https://doi.org/10.1016/j.biopsycho.2005.04.001.

Hansenne, M., W. Pitchot, A. Gonzalez Moreno, I. Urcelay Zaldua, and M. Ansseau. 1996. "Suicidal Behavior in Depressive Disorder: An Event-Related Potential Study." *Biological Psychiatry* 40, no. 2: 116–122. https://doi.org/10.1016/0006-3223(95)00372-X.

Harold, R., K. R. Donaldson, D. Rollock, R. Kotov, G. Perlman, and D. Foti. 2023. "Abnormal Neurophysiological Sensitivity to Rewards in Depression Is Moderated by Sex and Age in Middle Adulthood." *Biological Psychology* 182: 108623. https://doi.org/10.1016/j.biopsycho.2023.108623.

Holroyd, C. B., and M. G. H. Coles. 2002. "The Neural Basis of Human Error Processing: Reinforcement Learning, Dopamine, and the Error-Related Negativity." *Psychological Review* 109, no. 4: 679–709. https://doi.org/10.1037/0033-295X.109.4.679.

Irwin, D. E., B. Stucky, M. M. Langer, et al. 2010. "An Item Response Analysis of the Pediatric PROMIS Anxiety and Depressive Symptoms Scales." *Quality of Life Research* 19, no. 4: 595–607. https://doi.org/10.1007/s11136-010-9619-3.

Keren, H., G. O'Callaghan, P. Vidal-Ribas, et al. 2018. "Reward Processing in Depression: A Conceptual and Meta-Analytic Review Across fMRI and EEG Studies." *American Journal of Psychiatry* 175, no. 11: 1111–1120. https://doi.org/10.1176/appi.ajp.2018.17101124.

Klaufus, L. H., M. A. J. Luijten, E. Verlinden, et al. 2021. "Psychometric Properties of the Dutch-Flemish PROMIS® Pediatric Item Banks Anxiety and Depressive Symptoms in a General Population." *Quality of Life Research* 30, no. 9: 2683–2695. https://doi.org/10.1007/s11136-021-02852-y.

Klawohn, J., K. Burani, A. Bruchnak, N. Santopetro, and G. Hajcak. 2021. "Reduced Neural Response to Reward and Pleasant Pictures Independently Relate to Depression." *Psychological Medicine* 51, no. 5: 741–749. https://doi.org/10.1017/S0033291719003659.

Knott, V. J., and Y. D. Lapierre. 1987. "Electrophysiological and Behavioral Correlates of Psychomotor Responsivity in Depression." *Biological Psychiatry* 22, no. 3: 313–324. https://doi.org/10.1016/0006-3223(87)90149-1.

Knutson, B., G. W. Fong, C. M. Adams, J. L. Varner, and D. Hommer. 2001. "Dissociation of Reward Anticipation and Outcome With Event-Related fMRI." *Neuroreport* 12, no. 17: 3683–3687. https://journals.lww.com/neuroreport/toc/2001/12040.

Knutson, B., A. Westdorp, E. Kaiser, and D. Hommer. 2000. "FMRI Visualization of Brain Activity During a Monetary Incentive Delay Task." *NeuroImage* 12, no. 1: 20–27. https://doi.org/10.1006/nimg.2000.0593.

Knutson, B., G. E. Wimmer, C. M. Kuhnen, and P. Winkielman. 2008. "Nucleus Accumbens Activation Mediates the Influence of Reward Cues on Financial Risk Taking." *Neuroreport* 19, no. 5: 509–513. https://doi.org/10.1097/WNR.0b013e3282f85c01.

Köhler, S., C. H. Ashton, R. Marsh, A. J. Thomas, N. A. Barnett, and J. T. O'Brien. 2011. "Electrophysiological Changes in Late Life Depression and Their Relation to Structural Brain Changes." *International Psychogeriatrics* 23, no. 1: 141–148. https://doi.org/10.1017/S1041610210001250.

Kringelbach, M. L. 2005. "The Human Orbitofrontal Cortex: Linking Reward to Hedonic Experience." *Nature Reviews Neuroscience* 6, no. 9: 691–702. https://doi.org/10.1038/nrn1747.

Kujawa, A. 2024. "Reduced Reward Responsiveness and Depression Vulnerability: Consideration of Social Contexts and Implications for Intervention." *Psychophysiology* 61, no. 6: e14528. https://doi.org/10.1111/psyp.14528.

Kujawa, A., G. H. Proudfit, and D. N. Klein. 2014. "Neural Reactivity to Rewards and Losses in Offspring of Mothers and Fathers With Histories of Depressive and Anxiety Disorders." *Journal of Abnormal Psychology* 123, no. 2: 287–297. https://doi.org/10.1037/a0036285.

Lange, S., A. Leue, and A. Beauducel. 2012. "Behavioral Approach and Reward Processing: Results on Feedback-Related Negativity and P3 Component." *Biological Psychology* 89, no. 2: 416–425. https://doi.org/10.1016/j.biopsycho.2011.12.004.

Lees, T., N. Ram, M. M. Swingler, and L. M. Gatzke-Kopp. 2024. "The Effect of Hair Type and Texture on Electroencephalography and Event-Related Potential Data Quality." *Psychophysiology* 61, no. 3: e14499. https://doi.org/10.1111/psyp.14499.

Leknes, S., and I. Tracey. 2008. "A Common Neurobiology for Pain and Pleasure." *Nature Reviews Neuroscience* 9, no. 4: 314–320. https://doi.org/10.1038/nrn2333.

Liu, W., L. Wang, H. Shang, et al. 2014. "The Influence of Anhedonia on Feedback Negativity in Major Depressive Disorder." *Neuropsychologia* 53: 213–220. https://doi.org/10.1016/j.neuropsychologia.2013.11.023.

Liu, X., J. Hairston, M. Schrier, and J. Fan. 2011. "Common and Distinct Networks Underlying Reward Valence and Processing Stages: A Meta-Analysis of Functional Neuroimaging Studies." *Neuroscience & Biobehavioral Reviews* 35, no. 5: 1219–1236. https://doi.org/10.1016/j.neubiorev.2010.12.012.

Lopez-Calderon, J., and S. J. Luck. 2014. "ERPLAB: An Open-Source Toolbox for the Analysis of Event-Related Potentials." *Frontiers in Human Neuroscience* 8: 213. https://doi.org/10.3389/fnhum.2014.00213.

Luking, K. R., K. Gilbert, D. Kelly, et al. 2021. "The Relationship Between Depression Symptoms and Adolescent Neural Response During Reward Anticipation and Outcome Depends on Developmental Timing: Evidence From a Longitudinal Study." *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging* 6, no. 5: 527–535. https://doi.org/10.1016/j.bpsc.2020.11.001.

Meyer, A., M. D. Lerner, A. De Los Reyes, R. D. Laird, and G. Hajcak. 2017. "Considering ERP Difference Scores as Individual Difference Measures: Issues With Subtraction and Alternative Approaches." *Psychophysiology* 54, no. 1: 114–122. https://doi.org/10.1111/psyp.12664.

Meyer, G. M., J. Marco-Pallarés, P. Boulinguez, and G. Sescousse. 2021. "Electrophysiological Underpinnings of Reward Processing: Are We Exploiting the Full Potential of EEG?" *NeuroImage* 242: 118478. https://doi.org/10.1016/j.neuroimage.2021.118478.

Mies, G. W., F. M. van der Veen, J. H. M. Tulen, T. K. Birkenhäger, M. W. Hengeveld, and M. W. van der Molen. 2011. "Drug-Free Patients With Major Depression Show an Increased Electrophysiological Response to Valid and Invalid Feedback." *Psychological Medicine* 41, no. 12: 2515–2525. https://doi.org/10.1017/S0033291711000778.

Miltner, W. H. R., C. H. Braun, and M. G. H. Coles. 1997. "Event-Related Brain Potentials Following Incorrect Feedback in a Time-Estimation Task: Evidence for a "Generic" Neural System for Error Detection." *Journal of Cognitive Neuroscience* 9, no. 6: 788–798. https://doi.org/10.1162/jocn.1997.9.6.788.

Moran, T. P., H. S. Schroder, C. Kneip, and J. S. Moser. 2017. "Meta-Analysis and Psychophysiology: A Tutorial Using Depression and Action-Monitoring Event-Related Potentials." *International Journal of Psychophysiology* 111: 17–32. https://doi.org/10.1016/j.ijpsycho.2016.07.001.

Moser, J. S., T. P. Moran, C. Kneip, H. S. Schroder, and M. J. Larson. 2016. "Sex Moderates the Association Between Symptoms of Anxiety, but Not Obsessive Compulsive Disorder, and Error-Monitoring Brain Activity: A Meta-Analytic Review." *Psychophysiology* 53, no. 1: 21–29. https://doi.org/10.1111/psyp.12509.

Mueller, E. M., P. Pechtel, A. L. Cohen, S. R. Douglas, and D. A. Pizzagalli. 2015. "Potentiated Processing of Negative Feedback in Depression Is Attenuated by Anhedonia." *Depression and Anxiety* 32, no. 4: 296–305. https://doi.org/10.1002/da.22338.

Nelson, B. D., E. M. Kessel, F. Jackson, and G. Hajcak. 2016a. "The Impact of an Unpredictable Context and Intolerance of Uncertainty on the Electrocortical Response to Monetary Gains and Losses." *Cognitive, Affective, & Behavioral Neuroscience* 16, no. 1: 153–163. https://doi.org/10.3758/s13415-015-0382-3.

Nelson, B. D., G. Perlman, D. N. Klein, R. Kotov, and G. Hajcak. 2016b. "Blunted Neural Response to Rewards as a Prospective Predictor of the Development of Depression in Adolescent Girls." *American Journal of Psychiatry* 173, no. 12: 1223–1230. https://doi.org/10.1176/appi.ajp.2016.15121524.

Ng, T. H., L. B. Alloy, and D. V. Smith. 2019. "Meta-Analysis of Reward Processing in Major Depressive Disorder Reveals Distinct Abnormalities Within the Reward Circuit." *Translational Psychiatry* 9, no. 1: 293. https://doi.org/10.1038/s41398-019-0644-x.

Nguyen, D., E. E. Naffziger, and K. C. Berridge. 2021. "Positive Affect: Nature and Brain Bases of Liking and Wanting." *Current Opinion in Behavioral Sciences* 39: 72–78. https://doi.org/10.1016/j.cobeha.2021.02.013.

Nolan, H., R. Whelan, and R. B. Reilly. 2010. "FASTER: Fully Automated Statistical Thresholding for EEG Artifact Rejection." *Journal of Neuroscience Methods* 192, no. 1: 152–162. https://doi.org/10.1016/j.jneumeth.2010.07.015.

Novak, B. K., K. D. Novak, D. R. Lynam, and D. Foti. 2016. "Individual Differences in the Time Course of Reward Processing: Stage-Specific Links With Depression and Impulsivity." *Biological Psychology* 119: 79–90. https://doi.org/10.1016/j.biopsycho.2016.07.008.

Novak, K. D., and D. Foti. 2015. "Teasing Apart the Anticipatory and Consummatory Processing of Monetary Incentives: An Event-Related Potential Study of Reward Dynamics." *Psychophysiology* 52, no. 11: 1470–1482. https://doi.org/10.1111/psyp.12504.

Nusslock, R., and L. B. Alloy. 2017. "Reward Processing and Mood-Related Symptoms: An RDoC and Translational Neuroscience Perspective." *Journal of Affective Disorders* 216, no. 1: 3–16. https://doi.org/10.1016/j.jad.2017.02.001.

Padrão, G., A. Mallorquí, D. Cucurell, J. Marco-Pallares, and A. Rodriguez-Fornells. 2013. "Neurophysiological Differences in Reward Processing in Anhedonics." *Cognitive, Affective, & Behavioral Neuroscience* 13, no. 1: 102–115. https://doi.org/10.3758/s13415-012-0119-5.

Pfabigan, D. M., J. Alexopoulos, H. Bauer, and U. Sailer. 2011. "Manipulation of Feedback Expectancy and Valence Induces Negative and Positive Reward Prediction Error Signals Manifest in Event-Related Brain Potentials." *Psychophysiology* 48, no. 5: 656–664. https://doi.org/10.1111/j.1469-8986.2010.01136.x.

Pfabigan, D. M., E. M. Seidel, R. Sladky, et al. 2014. "P300 Amplitude Variation Is Related to Ventral Striatum BOLD Response During Gain and Loss Anticipation: An EEG and fMRI Experiment." *NeuroImage* 96: 12–21. https://doi.org/10.1016/j.neuroimage.2014.03.077.

Pilkonis, P. A., S. W. Choi, S. P. Reise, A. M. Stover, W. T. Riley, and D. Cella. 2011. "Item Banks for Measuring Emotional Distress From the Patient-Reported Outcomes Measurement Information System (PROMIS®): Depression, Anxiety, and Anger." *Assessment* 18, no. 3: 263–283. https://doi.org/10.1177/1073191111411667.

Pilkonis, P. A., L. Yu, N. E. Dodds, K. L. Johnston, C. C. Maihoefer, and S. M. Lawrence. 2014. "Validation of the Depression Item Bank From the Patient-Reported Outcomes Measurement Information System (PROMIS®) in a Three-Month Observational Study." *Journal of Psychiatric Research* 56: 112–119. https://doi.org/10.1016/j.jpsychires.2014.05.010.

Pion-Tonachini, L., K. Kreutz-Delgado, and S. Makeig. 2019. "ICLabel: An Automated Electroencephalographic Independent Component Classifier, Dataset, and Website." *NeuroImage* 198: 181–197. https://doi.org/10.1016/j.neuroimage.2019.05.026.

Pizzagalli, D. A. 2014. "Depression, Stress, and Anhedonia: Toward a Synthesis and Integrated Model." *Annual Review of Clinical Psychology* 10, no. 1: 393–423. https://doi.org/10.1146/annurev-clinpsy-050212-185606.

Pizzagalli, D. A., D. G. Dillon, R. Bogdan, and A. J. Holmes. 2011. "Reward and Punishment Processing in the Human Brain: Clues From Affective Neuroscience and Implications for Depression Research." In *Neuroscience of Decision Making*, edited by O. Vartanian and D. R. Mandel, 1st ed., 199–220. Psychology Press.

Pizzagalli, D. A., D. Iosifescu, L. A. Hallett, K. G. Ratner, and M. Fava. 2008. "Reduced Hedonic Capacity in Major Depressive Disorder: Evidence From a Probabilistic Reward Task." *Journal of Psychiatric Research* 43, no. 1: 76–87. https://doi.org/10.1016/j.jpsychires.2008.03.001.

Pizzagalli, D. A., and A. C. Roberts. 2022. "Prefrontal Cortex and Depression." *Neuropsychopharmacology* 47, no. 1: 225–246. https://doi.org/10.1038/s41386-021-01101-7.

Polich, J. 2007. "Updating P300: An Integrative Theory of P3a and P3b." *Clinical Neurophysiology* 118, no. 10: 2128–2148. https://doi.org/10.1016/j.clinph.2007.04.019.

Proudfit, G. H. 2015. "The Reward Positivity: From Basic Research on Reward to a Biomarker for Depression." *Psychophysiology* 52, no. 4: 449–459. https://doi.org/10.1111/psyp.12370.

R Core Team. 2013. *A Language and Environment for Statistical Computing*. R Foundation for Statistical Computing.

Reeve, B. B., M. McFatrich, J. W. Mack, et al. 2020. "Expanding Construct Validity of Established and New PROMIS Pediatric Measures for Children and Adolescents Receiving Cancer Treatment." *Pediatric Blood & Cancer* 67, no. 4: e28160. https://doi.org/10.1002/pbc.28160.

San Martín, R. 2012. "Event-Related Potential Studies of Outcome Processing and Feedback-Guided Learning." *Frontiers in Human Neuroscience* 6: 304. https://doi.org/10.3389/fnhum.2012.00304.

Santesso, D. L., K. T. Steele, R. Bogdan, et al. 2008. "Enhanced Negative Feedback Responses in Remitted Depression." *Neuroreport* 19, no. 10: 1045–1048. https://doi.org/10.1097/WNR.0b013e3283036e73.

Santopetro, N. J., C. J. Brush, A. Bruchnak, J. Klawohn, and G. Hajcak. 2021. "A Reduced P300 Prospectively Predicts Increased Depressive Severity in Adults With Clinical Depression." *Psychophysiology* 58, no. 4: e13767. https://doi.org/10.1111/psyp.13767.

Schevernels, H., R. M. Krebs, P. Santens, M. G. Woldorff, and C. N. Boehler. 2014. "Task Preparation Processes Related to Reward Prediction Precede Those Related to Task-Difficulty Expectation." *NeuroImage* 84: 639–647. https://doi.org/10.1016/j.neuroimage.2013.09.039.

Thompson, B., N. J. Santopetro, C. J. Brush, D. Foti, and G. Hajcak. 2023. "Neural Deficits in Anticipatory and Consummatory Reward Processing Are Uniquely Associated With Current Depressive Symptoms During Adolescence." *Psychophysiology* 60, no. 7: e14257. https://doi.org/10.1111/psyp.14257.

Threadgill, A. H., and P. A. Gable. 2016. "Approach-Motivated Pregoal States Enhance the Reward Positivity." *Psychophysiology* 53, no. 5: 733–738. https://doi.org/10.1111/psyp.12611.

Treadway, M. T., N. A. Bossaller, R. C. Shelton, and D. H. Zald. 2012. "Effort-Based Decision-Making in Major Depressive Disorder: A Translational Model of Motivational Anhedonia." *Journal of Abnormal Psychology* 121, no. 3: 553–558. https://doi.org/10.1037/a0028813.

Treadway, M. T., and D. H. Zald. 2011. "Reconsidering Anhedonia in Depression: Lessons From Translational Neuroscience." *Neuroscience & Biobehavioral Reviews* 35, no. 3: 537–555. https://doi.org/10.1016/j.neubiorev.2010.06.006.

Tucker, D. M., P. Luu, G. Frishkoff, J. Quiring, and C. Poulsen. 2003. "Frontolimbic Response to Negative Feedback in Clinical Depression." *Journal of Abnormal Psychology* 112, no. 4: 667–678. https://doi.org/10.1037/0021-843X.112.4.667.

Umemoto, A., and C. B. Holroyd. 2017. "Neural Mechanisms of Reward Processing Associated With Depression-Related Personality Traits." *Clinical Neurophysiology* 128, no. 7: 1184–1196. https://doi.org/10.1016/j.clinph.2017.03.049.

Van derhasselt, M.-A., R. De Raedt, A. De Paepe, et al. 2014. "Abnormal Proactive and Reactive Cognitive Control During Conflict Processing in Major Depression." *Journal of Abnormal Psychology* 123: 68–80. https://doi.org/10.1037/a0035816.

Varni, J. W., B. Magnus, B. D. Stucky, et al. 2014. "Psychometric Properties of the PROMIS® Pediatric Scales: Precision, Stability, and Comparison of Different Scoring and Administration Options." *Quality of Life Research* 23, no. 4: 1233–1243. https://doi.org/10.1007/s11136-013-0544-0.

Vernon-Feagans, L., M. Cox, and The Family Life Project Key Investigators. 2013. "The Family Life Project: An Epidemiological and Developmental Study of Young Children Living in Poor Rural Communities." *Monographs of the Society for Research in Child Development* 78, no. 5: 1–150. https://doi.org/10.1111/mono.12046.

Vuillier, L., D. Whitebread, and D. Szucs. 2015. "ERP Evidence of Cognitive Strategy Change in Motivational Conditions With Varying Level of Difficulty." *Neuropsychologia* 70: 126–133. https://doi.org/10.1016/j.neuropsychologia.2015.02.025.

Walter, W. G., R. Cooper, V. J. Aldridge, W. C. McCallum, and A. L. Winter. 1964. "Contingent Negative Variation: An Electric Sign of Sensori-Motor Association and Expectancy in the Human Brain." *Nature* 203, no. 4943: 380–384. https://doi.org/10.1038/203380a0.

Webb, C. A., R. P. Auerbach, E. Bondy, C. H. Stanton, D. Foti, and D. A. Pizzagalli. 2017. "Abnormal Neural Responses to Feedback in Depressed Adolescents." *Journal of Abnormal Psychology* 126, no. 1: 19–31. https://doi.org/10.1037/abn0000228.

Weinberg, A., H. Liu, G. Hajcak, and S. A. Shankman. 2015. "Blunted Neural Response to Rewards as a Vulnerability Factor for Depression: Results From a Family Study." *Journal of Abnormal Psychology* 124, no. 4: 878–889. https://doi.org/10.1037/abn0000081.

White, E. J., M. Nacke, E. Akeman, et al. 2021. "P300 Amplitude During a Monetary Incentive Delay Task Predicts Future Therapy Completion in Individuals With Major Depressive Disorder." *Journal of Affective Disorders* 295: 873–882. https://doi.org/10.1016/j.jad.2021.08.106.

Whitton, A. E., P. Kakani, D. Foti, et al. 2016. "Blunted Neural Responses to Reward in Remitted Major Depression: A High-Density Event-Related Potential Study." *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging* 1, no. 1: 87–95. https://doi.org/10.1016/j.bpsc.2015.09.007.

Whitton, A. E., P. Kumar, M. T. Treadway, et al. 2021. "Mapping Disease Course Across the Mood Disorder Spectrum Through a Research Domain Criteria Framework." *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging* 6, no. 7: 706–715. https://doi.org/10.1016/j.bpsc.2021.01.004.

Whitton, A. E., M. T. Treadway, and D. A. Pizzagalli. 2015. "Reward Processing Dysfunction in Major Depression, Bipolar Disorder and Schizophrenia." *Current Opinion in Psychiatry* 28, no. 1: 7–12. https://doi.org/10.1097/YCO.0000000000000122.

Wickham, H. 2016. *ggplot2*. Springer. https://doi.org/10.1007/978-0-387-98141-3.

Zhang, W.-N., S.-H. Chang, L.-Y. Guo, K.-L. Zhang, and J. Wang. 2013. "The Neural Correlates of Reward-Related Processing in Major Depressive Disorder: A Meta-Analysis of Functional Magnetic Resonance Imaging Studies." *Journal of Affective Disorders* 151, no. 2: 531–539. https://doi.org/10.1016/j.jad.2013.06.039.

Zhou, L., G. Wang, C. Nan, H. Wang, Z. Liu, and H. Bai. 2019. "Abnormalities in P300 Components in Depression: An ERP-sLORETA Study." *Nordic Journal of Psychiatry* 73, no. 1: 1–8. https://doi.org/10.1080/08039488.2018.1478991.

Supporting Information

Additional supporting information can be found online in the Supporting Information section.