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Interaction Effects of Irritability and Anhedonia on ERP and Time–Frequency Measures of Reward Sensitivity

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ABSTRACT

Irritability and anhedonia are two prevalent, co-occurring, and impairing symptoms of major depression that are proposed to result from dysfunctions in reward processing. While irritability is associated with *heightened* sensitivity to reward receipt, anhedonia is linked to *blunted* sensitivity to reward receipt. Given these supposed paradoxical effects on reward sensitivity, it is noteworthy that no studies have examined how both symptoms interact to affect reward sensitivity. In a community sample of young adults ($N=73$, $M_{age}=21.21 \pm 2.27$, 56.16% females), we evaluated the interacting effects of dimensionally assessed irritability and anhedonia symptoms on neural measures of reward and loss sensitivity during a well-established reward paradigm (the Doors task). Event-related potential and time–frequency measures were analyzed using both univariate and a multivariate method based on generalized eigendecomposition to isolate delta- and theta-band activity in response to feedback. Linear-mixed models tested the unique and interacting effects of irritability and anhedonia on EEG measures of reward sensitivity (FRN/N2, theta, and delta power). Participants with above-average irritability and anhedonia displayed larger (more negative) FRN/N2 amplitudes across feedback valence, and reduced theta power following reward, whereas no associations were observed at lower symptom levels. These findings suggest that individuals with co-occurring irritability and anhedonia may perceive feedback overall (reward and loss) as more salient, while interpreting rewards as less motivationally relevant. These effects remained significant when controlling for broader depressive symptoms, gender, and income. These results provide preliminary evidence that co-occurring irritability and anhedonia jointly influence neural sensitivity to feedback, in individuals without clinical levels of symptoms. Awaiting validation in clinical samples, our results highlight the importance of considering symptom interactions to better understand reward-related mechanisms in depression.

1 | Introduction

Depressive disorders, including major depressive disorder (MDD), are a leading cause of disability worldwide (Lopez et al. 2006; GBD 2018). Despite its significant public health impact and decades of neuroscience research, advances in understanding MDD's pathophysiology and biomarkers remain limited (Kapur et al. 2012; Winter et al. 2024). The largest and most recent neuroimaging meta-analyses relying on comparisons between patients with MDD and healthy controls have yielded modest or null effects (Müller et al. 2017; Ng et al. 2019; Schmaal et al. 2017, 2020). Symptomatic heterogeneity in MDD samples (Fried, Coomans, et al. 2020; Fried and Nesse 2015), where individuals can present contrasting features (e.g., weight/appetite increase vs. decrease, insomnia vs. hypersomnia) (Zimmerman et al. 2015), likely limits progress. A symptom-based approach

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may improve our understanding of depression (Fried 2017; Insel et al. 2010), as individual MDD symptoms are linked to distinct risk factors (Fried et al. 2014), inflammatory markers (Frank et al. 2021; Fried, Von Stockert, et al. 2020; van Eeden et al. 2020), and brain structures (Freichel et al. 2024; Hilland et al. 2020).

Dysfunction in reward processing is a leading candidate mechanism in depression (Halahakoon et al. 2020; Keren et al. 2018; Mackin et al. 2021; Proudfit 2015). Reward processing involves many subcomponents, including reward anticipation, motivation, consumption, and learning (Berridge and Robinson 2003). Specifically, *reward sensitivity* (i.e., the hedonic/consummatory response to reward receipt) is blunted in MDD (e.g., Brush et al. 2018; Cavanagh et al. 2021; Foti et al. 2014; Foti and Hajcak 2009; Hager et al. 2022). Most of the symptom-specific explorations of reward sensitivity have focused on anhedonia (Borsini et al. 2020; Pizzagalli 2022), linking greater anhedonia severity with reduced reward sensitivity and lower reward approach among both MDD and nonclinical populations (Banica et al. 2022; Borsini et al. 2020; Liu et al. 2014; Stoy et al. 2012; Wang et al. 2020; Whitton et al. 2015), although this association is not always replicated in nonclinical samples (e.g., Hager et al. 2022; Umemoto et al. 2019). Neuroimaging studies link anhedonia with reduced ventral striatum (VS) responses to reward (e.g., Borsini et al. 2020), while pharmacologically induced VS activity reduces anhedonia symptoms (Pizzagalli et al. 2020).

Another common MDD symptom—irritability, defined as an increased proneness to experience anger and frustration—has also been proposed to result from dysfunctional reward processing (Brotman et al. 2017). Although not a DSM-5 criterion for MDD in adults, irritability is reported by 40%–60% of patients with MDD (Fava et al. 2010; Judd et al. 2013; Rizk et al. 2025). Irritability is also common in the adult general population, particularly among young adults (Perlis et al. 2024), although direct prevalence comparisons across clinical vs. nonclinical samples are limited as studies in MDD and the general population use different measurement tools. Irritability is associated with greater severity and duration of the depressive episode in MDD patients (Fava et al. 2010; Judd et al. 2013; Perlis et al. 2009), as well as psychosocial impairment, suicidality, and lower life satisfaction in both clinical and community samples (Jha et al. 2020; Liu et al. 2023; Perlis et al. 2024), highlighting its clinical relevance.

Interestingly, there is evidence that irritability and anhedonia frequently co-occur in adult MDD. Among patients with MDD, anhedonia is reported by 88.2% of those with irritability (Fava et al. 2010), and individuals with irritable mood are 6.7 times more likely to experience anhedonia than those without irritable mood (Liu and Cole 2021). Additionally, cross-sectional and longitudinal associations between irritability and anhedonia are observed in the general population (Losiewicz et al. 2023; Sistiaga et al. 2024). Despite the frequent co-occurrence of both symptoms, they are associated with contrasting patterns of neural sensitivity measures. Whereas anhedonia has been linked with blunted reward sensitivity (e.g., Borsini et al. 2020), pediatric irritability has been linked with increased reward sensitivity and behavioral approach tendencies (Bebko et al. 2014; Kessel et al. 2016; Perlman et al. 2015). This higher sensitivity to reward might contribute to a lower threshold for experiencing frustration in pediatric irritability (Brotman et al. 2017). However, it remains unclear whether adult irritability is associated with atypical reward sensitivity. As brain regions supporting reward processing undergo important changes throughout adolescence (Galvan 2010), the neural mechanisms underlying irritability may vary across developmental stages. To the best of our knowledge, only one event-related potential (ERP) study has investigated reward sensitivity in irritability in young adults and found that irritability was unrelated to Reward Positivity (RewP) amplitudes, but was related to reduced neural response to loss during a frustrating task (Deveney 2019). However, important differences between the paradigm in that study and those typically used to probe reward sensitivity in anhedonia and depression (e.g., the Doors task; Foti et al. 2011; Proudfit 2015) complicate direct comparisons between studies. Greater methodological alignment is needed, as evidence shows higher trait anger (a construct closely linked to irritability) in young adults was linked to larger RewP amplitudes on the Doors task (Tsypes et al. 2019).

Taken together, studies suggest that irritability and anhedonia may have opposite patterns of reward sensitivity; whereas irritability is associated with *heightened* sensitivity to reward receipt, anhedonia is linked to *blunted* sensitivity to reward receipt. This raises the question of how these two apparently paradoxical mechanisms can co-exist within the same individuals reporting both symptoms. This also raises questions about whether findings on anhedonia and reward responsivity have been influenced by the levels of co-occurring irritability in study samples, and, conversely, whether the presence and severity of anhedonia may have influenced irritability-related results in reward responsivity tasks. Zisner and Beauchaine (2016) proposed that hypofunctioning of the mesolimbic dopamine reward system could be a mechanism explaining the comorbidity between irritability and anhedonia. However, to the best of our knowledge, this has not been empirically evaluated, and how this would manifest at the macroscopic neural level remains unclear. To begin addressing this question, the present study aimed to evaluate the shared and unique effects of anhedonia and irritability on neural measures of feedback sensitivity, using an established reward paradigm—the Doors task (Foti et al. 2011; Proudfit 2015). Given that irritability and depressive symptoms are especially prevalent in 18–24-year-olds (Collier Villaume et al. 2023; Perlis et al. 2024) and most mental health problems develop before age 24 (Solmi et al. 2022), we investigated those reward-related mechanisms in young adults. Understanding these symptom-specific influences on feedback sensitivity could help identify precise targets for treatment and prevention in this population.

To examine these processes, research has often focused on the feedback-related negativity (FRN), a frontocentral ERP that occurs 250–350 ms after feedback presentation. The FRN is more negative (i.e., larger) to negative versus to positive feedback and to unexpected relative to expected outcomes (Paul et al. 2025). Although interpretations of the FRN continue to evolve (Cavanagh and Holroyd 2026), it is thought to reflect an obligatory neural response to improbable, task-relevant events, analogous to the N200 elicited by rare stimuli in oddball paradigms. In the case of negative feedback, the associated negative reward prediction error (RPE) may slightly enhance the FRN/N2 amplitude. In

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contrast, positive feedback elicits a reward-related component, the RewP, which suppresses the underlying FRN/N2 (Holroyd et al. 2008). The RewP is selectively elicited by reward or goal attainment and reflects sensitivity to positive RPE (Cavanagh and Holroyd 2026; Gheza, Paul, et al. 2018; Proudfit 2015). This framework supports the use of the difference-wave approach, as it removes the obligatory component (the FRN/N2), isolating the valence-related signal that primarily reflects reward processing (the RewP). Evidence from topographic and source analyses further supports the existence of two independent, but temporally overlapping, processes (Cavanagh and Holroyd 2026). For example, the FRN/N2 has been linked to a frontocentral topography and a dorsal anterior cingulate cortex (ACC) generator while the RewP had a more central topography and a likely posterior cingulate cortex generator (Gheza, Paul, et al. 2018). Following this conceptualization, the present study examined how irritability and anhedonia relate to FRN/N2 amplitudes, using feedback valence (reward vs. loss) as a fixed factor and additionally employed the RewP (difference-wave approach) to isolate positive feedback signals and facilitate comparisons with previous research on reward sensitivity using the Doors task.

This traditional ERP approach is useful but overlooks information present in the EEG signal. Because ERP components reflect overlapping frequency activities (e.g., delta and theta) linked to different cognitive processes (Bernat et al. 2015), time–frequency (TF) analyses provide complementary insights by capturing temporal dynamics of power across frequency bands. For example, simultaneous ERP and TF studies (Gheza, De Raedt, et al. 2018) show that the FRN/N2 is sensitive to unexpected nonreward, supporting its role in signaling RPE. Theta power (4-8 Hz) over frontocentral areas, originating from the ACC (Cohen et al. 2008; Foti et al. 2015), tracks outcomes and expectations more broadly regardless of feedback valence: it is larger for negative feedback but also responds to positive expectancy violations, indexing a cognitive control mechanism that signals the need to adjust behavior following events such as worse-than-expected outcomes (Cavanagh and Frank 2014; Holroyd and Umemoto 2016). In contrast, delta power (1-3 Hz) over centroparietal areas is sensitive to predictable outcomes and secondary attributes of the stimulus (e.g., magnitude of the outcome; Bernat et al. 2015; Foti et al. 2015). Both delta and theta signals have been investigated in depression. Reduced delta power in response to reward was concurrently (Jin et al. 2019) and prospectively (Nelson et al. 2018) associated with higher depressive symptoms. Increased theta power in response to loss has been observed in female adolescents experiencing a current major depressive episode compared to healthy controls (Webb et al. 2017), and was associated with higher depressive symptoms (Jin et al. 2019). However, the extent to which individual symptoms (i.e., anhedonia and irritability) modulate delta and theta power in response to reward and loss is unknown.

Most EEG studies on reward sensitivity and depressive symptoms use *univariate* analyses, considering each electrode as a separate statistical unit. However, each electrode comprises mixed signals from multiple neural generators spread via volume conduction. This motivates the use of *multivariate* approaches that leverage the multidimensional information distributed across a set of electrodes to decompose and isolate the EEG data into task-relevant statistical sources (Cohen 2017a, 2022). This approach aligns well with the investigation of anhedonia, which is likely predicted by spatially distributed brain networks (Lynch et al. 2024), and the RewP, which is likely generated from multiple sources (Pirrung et al. 2025). The multivariate method used here, generalized eigendecomposition (GED), is a source-separation method that creates a spatial filter (i.e., a weighted combination of data channels, called "components") specifically designed to isolate relevant (e.g., task- or frequency-specific) from irrelevant patterns in the data (Cohen 2022). Compared to methods like principal or independent component analysis, GED allows for interindividual differences in topography (e.g., due to electrode placement, cortical folding), reducing the impact of individual variability on statistical power. Additionally, GED components are not constrained to be orthogonal, which allows for better separation of sources that are correlated in the channel space (Cohen 2017a). Importantly, GED supports hypothesis-driven analyses by allowing researchers to select a priori data features of interest based on specific research aims. This allows for the extraction of neural component(s) that capture task-related brain activity associated with the cognitive process of interest. These components can then be linked to clinical symptoms (e.g., hallucinations in schizophrenia; Ohki et al. 2024), offering valuable clinical insights.

Taken together, the aim of this study was to evaluate the unique and interacting effects of irritability and anhedonia on neural measures of reward and loss sensitivity on the Doors task (Foti et al. 2011; Proudfit 2015) using a combination of univariate ERP and TF measures of reward sensitivity, as well as a multivariate method (GED) isolating patterns of delta and theta activity in response to win and loss feedback. According to a wealth of past literature (Bernat et al. 2015; Foti et al. 2015; Proudfit 2015; Williams et al. 2021), we expected the Doors task to elicit (1) a larger (i.e., more negative) FRN/N2 following loss versus win, (2) increased delta power following win compared to loss, and (3) increased theta power following loss compared to win. Additionally, we hypothesized that irritability and anhedonia would have interacting effects on neural measures of win and loss sensitivity, although no specific directional hypotheses were formulated given the past mixed findings and the novelty of the present study.

2 | Method

2.1 | Participants

A total of 91 young adults (ages 18–25 years) from the community were recruited in 2022–2023 through flyers in New Haven, Connecticut. The study was approved by the Institutional Review Board of Yale University (HIC#: 2000031088). An initial phone survey determined eligibility: 18–25 years old, no major medical illnesses, no psychiatric condition other than depression or anxiety, no substance dependence, and no loss of consciousness > 5 min. Participants were paid $15/h and were informed that they could earn an additional $10 during the Doors task. Data from 13 participants (14.29%) were excluded due to our EEG equipment being incompatible with participants' hair types or hairstyles ($n = 5$), excessive EEG artifacts ($n = 5$), hardware issue ($n = 2$), and sickness during the experimental session ($n = 1$). The percentage of excluded participants is similar to previous reward sensitivity studies (Bowers et al. 2018; Williams et al. 2021).

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Prior to analyses, five outliers were detected on our irritability measure ($n = 4$) and our anhedonia measure ($n = 1$) using a conservative outlier detection method ±3 Median Absolute Deviation (Leys et al. 2013). These participants were excluded from the primary analyses given that linear regression coefficients are highly sensitive to outliers (Montgomery et al. 2021). However, the results from the full sample can be found in the Supplement (S1). Thus, our final sample consisted of 73 young adults ($M_{age} = 21.21 \pm 2.27$, 56.16% females, see Table 1 for sociodemographic characteristics).

TABLE 1 | Sociodemographic characteristics of the final sample ($N = 73$).

Variables	n (%)
Gender	
Female	41 (56.16%)
Male	31 (42.47%)
Refuse to answer/Don't know	1 (1.37%)
Education	
Partial High School Degree	1 (1.32%)
High School Degree	25 (32.89%)
Technical School Degree/Partial College	19 (25.00%)
College/University Degree	27 (35.53%)
Postgraduate Degree	4 (5.26%)
Occupation	
Student	60 (82.19%)
Professional	7 (9.59%)
Laborer	2 (1.37%)
Other	5 (6.85%)
Race	
Asian	15 (20.55%)
Black or African American	2 (2.74%)
White or Caucasian	49 (67.12%)
More than one race	6 (8.22%)
Refuse to answer/Don't know	1 (1.37%)
Ethnicity	
Hispanic/Latinx	13 (17.81%)
Not Hispanic/Latinx	60 (82.19%)
Family annual income	
Less than \$14.999	3 (4.11%)
\$15,000 through \$19,999	2 (2.74%)
\$20,000 through \$49,999	7 (9.59%)
\$50,000 through \$74,999	15 (20.55%)
\$75,000 through \$99,999	8 (10.96%)
\$100,000 and greater	36 (49.31%)
Refuse to answer/Don't know	2 (2.74%)
Individual annual income	
Less than \$14.999	52 (71.23%)
\$15,000 through \$19,999	2 (2.74%)
\$20,000 through \$24,999	1 (1.37%)
\$25,000 through \$49,999	15 (20.55%)
\$50,000 through \$74,999	2 (2.74%)
Refuse to answer/Don't know	1 (1.37%)

2.2 | Materials

2.2.1 | Brief Irritability Test (BITe)

The BITe (Holtzman et al. 2015) is a 5-item self-report questionnaire, rated on a 6-point Likert-type scale (1 = never, 2 = rarely, 3 = sometimes, 4 = often, 5 = very often, 6 = always), designed to assess irritability over the past 2 weeks. The total score is calculated by summing the score of each item, with a higher total score corresponding to a higher level of irritability. In the current sample, the BITe demonstrated good internal consistency (Cronbach's $\alpha = 0.80$) and is thought to be the most precise and reliable tool for measuring irritability in adults (Saatchi et al. 2023; Toohey and DiGiuseppe 2017). The mean BITe score in our sample (10.44) was consistent with levels observed in the general population (Holtzman et al. 2015; Perlis et al. 2024).

2.2.2 | Dimensional Anhedonia Rating Scale (DARS)

The 17-item DARS (Rizvi et al. 2015) assesses the dimensions of interest, motivation, effort, and consummatory pleasure across four major reward types: hobbies, food/drinks, social activities, and sensory experiences. Participants provide examples of their own favorite activities and are asked to rate how each item applies to them "right now" on a 5-point Likert-type scale ranging from 0 (not at all) to 4 (very much). The subscale scores are the sum of all the items within a reward type, and the total score is the sum of the score obtained for each subscale. Higher scores on the DARS reflect high interest, motivation, effort, and pleasure (i.e., a low level of anhedonia). The mean DARS score in our sample (55.22) was substantially higher than that typically observed in individuals with a major depressive episode (30.2; Rizvi et al. 2015). To enhance the interpretability of our findings, we reverse-coded DARS scores so that higher scores reflect greater levels of anhedonia. In the current sample, the DARS demonstrated good internal consistency ($\alpha = 0.89$).

2.2.3 | Doors Task

Participants completed the Doors task (Foti et al. 2011; Proudfit 2015), a traditional reward sensitivity task in which participants are presented with two doors, are instructed to choose one of the two doors, and receive feedback about this choice. Participants were informed that they should try to discern a rule/pattern about which door was going to award them the most monetary reward, and that the wins accumulated would translate into actual monetary reward. Two practice trials

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were followed by 60 trials: half of them followed by a monetary win (50¢) indicated by an upward green arrow and the other half followed by a monetary loss (25¢) indicated by a downward red arrow. Win and loss trials were presented randomly for a total of 30 wins and 30 losses. The doors stimuli appeared on the screen until a button was pressed by the participant, followed by a 1000 ms fixation cross, a 2000 ms feedback presentation, and a 1500 ms intertrial interval.

2.3 | Procedure

Young adults were asked to complete the BITe and DARS questionnaires and were then seated about 70 cm in front of the presentation computer to complete the Doors task. After the Doors task, they completed a frustration task that is not reported in this manuscript. At the end of the experimental session, participants were given the maximum reward ($10) regardless of their actual earnings during the task.

2.4 | Data Analyses

2.4.1 | EEG Recording and Preprocessing

EEG was recorded using a 128 Ag/AgCl electrode nets (HydroCel Geodesic Sensor Net) using a Net Amps 200 Amplifier and running Net Station 4.2.2 software (Electrical Geodesics Inc. [EGI], Eugene, OR). The reference electrode was placed at Cz, and electrodes were arranged according to the 10–20 system. Signals were sampled at 500 Hz. During recording, a 100 Hz low-pass filter was applied; the cutoff corresponds to the half-power point (−3 dB) with a standard roll-off. Electrode impedances were maintained below 50 kΩ throughout data acquisition. EEG analyses were conducted using custom scripts on MATLAB 2022b (MathWorks Inc., Natick, MA) and EEGLAB Toolbox (Delorme and Makeig 2004). Our preprocessing pipeline was adapted from previous work (Bowers et al. 2018) that used similar EEG equipment and experimental paradigm as in the current study. Data were downsampled to 200 Hz. Data were high-pass filtered at 0.3 Hz and low-pass filtered at 49 Hz. Noisy channels were identified via visual inspection of the channel power spectra and removed ($M = 1.45$, SD = 1.35, range 0–6). Channels were marked as "noisy" if they showed persistent excessive high-frequency noise, flatlining, or poor signal quality across the recording. Trials were epoched from −1000 to 2000 ms relative to feedback onset. Epochs were visually inspected, and those containing motion artifacts, muscle activity, or abrupt voltage shifts exceeding physiological ranges were rejected. This manual artifact rejection procedure was chosen because it proved to be more effective and less biased than automatic methods (Cohen 2017b). To further identify artifacts in the data, independent components analysis (ICA) was performed on a copy of the dataset that was filtered with a 1 Hz high-pass filter. ICA weights from the ICA run on the copied (1 Hz) data set were then copied back to the 0.3 Hz high-passed data. ICA artifact identification was guided by the ICLabel MATLAB plug-in (Pion-Tonachini et al. 2019), and components with clearly identifiable nonbrain artifacts (e.g., eye-blinks or heartbeats) were removed from the data ($M = 4.38$, SD = 1.30, range 1–7). Missing or removed channels were interpolated using a spherical spline interpolation, and data were re-referenced to the average of all electrodes. ERP amplitudes were baseline-corrected using the −200 to 0 ms prestimulus window. Participants had an average of 27.92 loss trials (SD = 3.68, range 12–30), and 28.15 win trials (SD = 3.37, range 17–30).

2.4.2 | ERP Analysis

Given the high interindividual variability in the peak time of the FRN/N2 and following published guidelines (Williams et al. 2021), we selected the peak amplitude for each participant within the 200 to 400 ms time window of the difference wave between win and loss feedback. Then, we systematically increased the window size (from 10 to 200 ms) to calculate the mean amplitude around the peak and identified the inflection point of the resulting mean amplitude curve at 50 ms (see Figure S3). Following Williams et al. (2021)'s recommendation, we used this time window of 50 ms centered around the peak, encompassing 10 time bins at 200 Hz. The FRN/N2 to loss and to win were computed as the mean amplitude in this time window post feedback on loss and win trials, respectively, and by averaging eight frontocentral electrodes surrounding FCz (electrode numbers on 128-ch. EGI net: 5, 6, 7, 11, 12, 13, 106, 112). For each participant, the FRN/N2 values were averaged across trials within each feedback valence, resulting in one value per participant per feedback valence. The grand average ERPs are shown in Figure 1. Split-half reliability showed a mean Spearman-Brown corrected reliability of 0.71 for FRN/N2 amplitudes to win trials, 0.67 for loss trials, and 0.60 for the RewP difference score.

2.4.3 | Time-Frequency Analysis

2.4.3.1 | Univariate Time–Frequency Analysis. We performed univariate TF analysis on data recorded at electrode FCz. TF measures were computed by multiplying the power spectrum of the epoched trials with a fast Fourier power spectrum of a set of complex Morlet wavelets. This results in a time series that reflects the similarities between the epoched EEG data and a selected wavelet, from which TF power can be derived to quantify the energy present at specific timepoints and frequencies. These wavelets were Gaussian-windowed complex sine waves that increased by 100 logarithmically spaced steps from 0.5 to 0.20 Hz. As frequency increased, the width of each frequency band was set to increase from $2/(2\pi f)$ to $10/(2\pi f)$. Then, the inverse fast Fourier transform was computed to recover the time series. For each feedback valence, the power at each timepoint was calculated by averaging the squared absolute value of the instantaneous amplitude. Averaged power by feedback valence was then converted to a decibel (dB) scale, to allow comparison between different frequency bands. The baseline was defined as the averaged power from −500 to −200 ms before feedback receipt. The analysis code was adapted from Cavanagh et al. (2021).

2.4.3.2 | Multivariate Source-Separation Analysis. To isolate theta and delta power in response to feedback receipt, we used GED, a hypothesis-driven multivariate source separation method (see Cohen 2022 for details), to create two (independent) spatial filters that maximize power in the theta and delta band,

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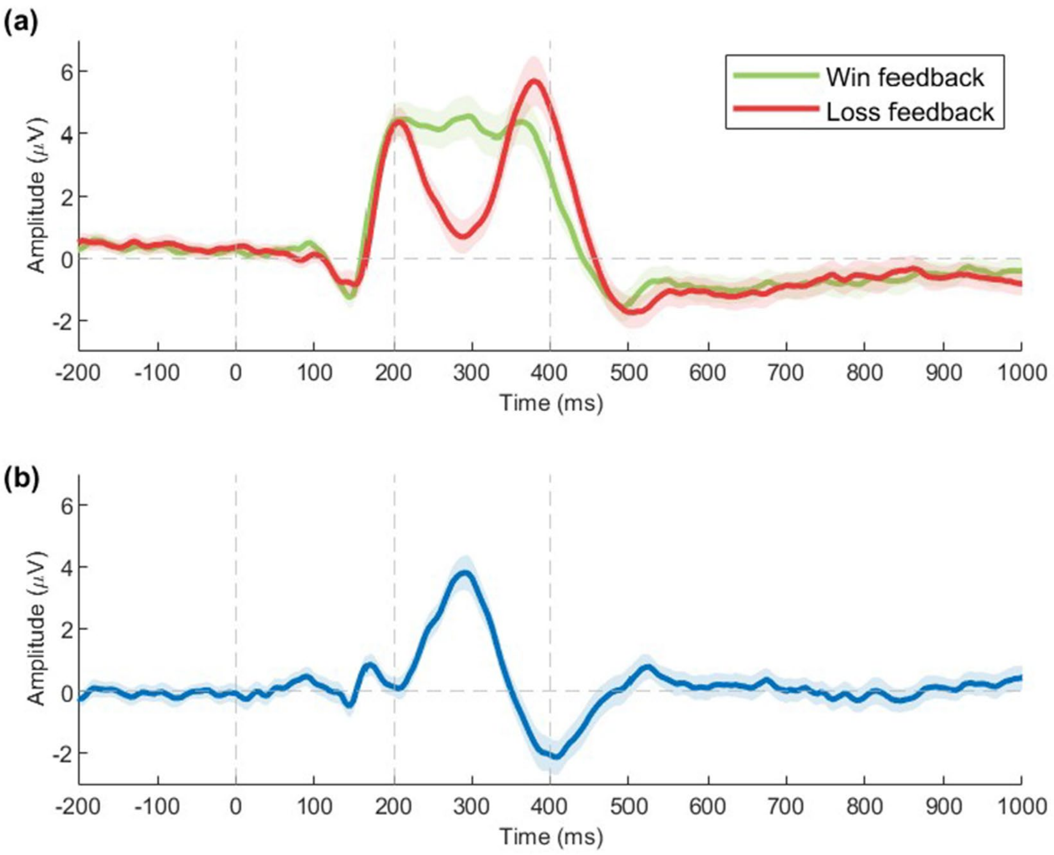

FIGURE 1 | Grand average ERPs in response to feedback at the average of eight frontocentral electrodes around FCz. Time = 0 ms corresponds to feedback presentation. (a) Grand average waveform per feedback valence with 95% confidence intervals. (b) Grand average difference waveform (computed by subtracting the FRN/N2 to loss from the FRN/N2 to win) with 95% confidence intervals.

respectively. This procedure was based on the approach used in previous work (Duprez et al. 2020; Zuure et al. 2020). Choice of theta and delta was guided by prior work on reward processing (e.g., Bernat et al. 2015; Williams et al. 2021).

A spatial filter is constructed by finding a set of weights for the data channels that maximizes a researcher-specified contrast between two data features. These two data features are contained in two covariance matrices, named S for signal and R for reference, encoding all pairwise linear interactions across channels. GED aims to find a set of channel weights contained in a vector **w** that maximizes the power ratio between S and R. This can be expressed as the generalized Rayleigh quotient, with λ being the generalized eigenvalue corresponding to the vector w:

$$\lambda = \frac{\mathrm{w}^{\mathrm{T}}\mathrm{S}\mathrm{w}}{\mathrm{w}^{\mathrm{T}}\mathrm{R}\mathrm{w}} \qquad (1)$$

In our case, we aimed to maximally separate theta and delta power from broadband activity. This involved the construction of 2 spatial filters (one for delta and one for theta), and therefore 2 sets of S and R matrices. We computed the S matrices as channel*channel covariance matrices derived from the signal narrowband filtered in delta or theta band around individual-specific peak frequencies. This was done to account for the high interindividual variability in those peaks (Haegens et al. 2014; Williams et al. 2021). Peak frequencies were identified for each participant by finding the maximum power based on the average of all trials within theta (4–8 Hz, 0–700 ms) and delta (1–3.9 Hz, 0–400 ms) bands from the previously described TF decomposition at FCz. Averaging across all trials was used to avoid overfitting and to ensure that subsequent statistical contrasts between feedback valences (win vs. loss) are orthogonal to the maximization criteria and unbiased (Cohen 2022). These peaks were then used as the center frequency for the Gaussian narrowband filters (full-width at half-maximum = 3 Hz for theta and 1.5 Hz for delta) applied to the broadband data. The resulting delta- and theta-filtered data were then used to compute the S matrices, defined within time windows of 800 ms for theta and 1200 ms for delta around their respective power peaks. The time windows were defined to incorporate multiple theta and delta cycles. We computed the R matrices as the channel*channel covariance matrices derived from broadband unfiltered data from the same time windows. The R matrices were regularized with 1% shrinkage ($\gamma = 0.01$) to reduce noise and improve stability and matrix separability.

The solution to Equation (1) involves performing a GED on S and R, defined by the equation SW = RWΛ, which gives a matrix of eigenvectors W and a diagonal matrix of eigenvalues Λ. The eigenvector with the highest associated generalized eigenvalue serves as the spatial filter that maximally separates S from R because eigenvectors with larger eigenvalues explain more variance. We performed 2 GEDs on these matrices, which yielded

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128 spatial filters for theta and 128 spatial filters for delta. To select the best component for each frequency band, we used the following selection procedure, inspired by Zuure et al. (2020). For theta, the 15 spatial filters with the highest eigenvalues were applied to the unfiltered data, generating 15 components. The topography of each component's activation was then compared to a predefined EEG template consisting of a Gaussian centered on midfrontal electrode FCz (Figure 3A1), where theta power is typically maximal. For each component, we computed the shared spatial variance (R^2_i) with this template. To ensure that the selected component not only resembled the expected topography but also optimally separated theta activity from other signals, R^2 values were weighted by the normalized eigenvalue of each component using the formula:

$$\text{Eigenvalue-weighted index}_i = \left(\frac{\lambda_i}{\sum_{j=1}^{N} \lambda_j} \right) * R_i^2$$

where λ_i is the eigenvalue of component i, N is the total number of components, and R^2_i is the shared spatial variance between the component's topography and the template. The component with the highest eigenvalue-weighted index was selected as the best theta spatial filter. The same procedure was applied to select the best delta spatial filter, except that the template was centered on Cz (Figure 3B1), where delta activity is typically maximal.

Finally, the selected theta and delta components were TF decomposed through complex Morlet wavelet convolution as described previously. Based on examination of the grand average TF representations, power values between 0 and 700 ms and 4–7 Hz for theta activity, and power values between 0 and 500 ms and 1.5–3.9 Hz for delta activity were exported at the participant level for statistical analyses. For each participant, power values were averaged across trials within each feedback valence, resulting in one theta and one delta value per participant per feedback valence. Spearman–Brown split-half reliability was moderate for win-related theta ($\rho = 0.56$) and good for loss-related theta ($\rho = 0.72$). Delta-band activity showed moderate-to-good reliability for both win ($\rho = 0.63$) and loss ($\rho = 0.58$).

2.4.4 | Statistical Analyses

2.4.4.1 | Descriptive Analyses. Means and standard deviations of the study variables were computed, and bivariate, zero-order correlations between them were examined.

2.4.4.2 | Effect of Feedback Valence on ERP and Time–Frequency Measures. We conducted three separate linear-mixed models (LMMs) to investigate the within-subject effect of feedback valence (win vs. loss) on FRN/N2 amplitude, theta power, and delta power. The models included feedback valence (win vs. loss) as a fixed effect and random intercepts for each subject to account for within-subject variability.

2.4.4.3 | Effect of Irritability, Anhedonia, and the Irritability × Anhedonia Interaction on ERP and Time–Frequency Measures. A set of 3 LMMs was conducted to evaluate the interaction effects between feedback valence (win vs. loss), irritability, and anhedonia, on each neural measure: FRN/N2 (Model 1), theta (Model 2), and delta (Model 3). For the RewP (difference-wave approach), a separate linear model was conducted and reported in the Supplement (S2). Independent variables were mean-centered prior to analysis, and random intercepts were calculated for participants. For all models, the corrected variance inflation factors were between 1.15 [95% CI 1.03, 1.92] and 1.57 [95% CI 1.32, 2.00] for the BITe and between 1.13 [95% CI 1.02, 1.98] and 1.54 [95% CI 1.30, 1.97] for the DARS, which suggested acceptable levels of multicollinearity for regression analysis. We explored the significant interactions using the Johnson–Neyman technique, a robust method for identifying regions of significance within an interaction (Bauer and Curran 2005; Johnson and Neyman 1936).

The fits of the LMMs with and without covariates of interest (i.e., gender, combined [family + individual] annual income, and depression score) were compared using ANOVA, to test whether including these covariates significantly improved model fit. We included the depression scores as measured by the depression subscale of the Depression Anxiety and Stress Scale (Lovibond and Lovibond 2011) to test whether the effects were specific to anhedonia and irritability or could be explained by broader depressive symptomatology (e.g., sadness, low positive affect, hopelessness, low self-esteem, worthlessness). Beforehand, we removed two items (i.e., item 5: "I found it difficult to work up the initiative to do things" and item 16: "I was unable to become enthusiastic about anything") from the total score of the depression subscale, as these items overlap with the construct of anhedonia. This revised scale demonstrated good internal consistency (Cronbach's $\alpha = 0.81$).

2.4.4.4 | Sample Size Justification and Sensitivity Power Analysis. The planned sample size was informed by prior work by Deveney (2019), which indicated that 62 participants would be sufficient to detect significant effects of a feedback manipulation on FRN/N2 amplitudes to loss versus gain. Power calculations suggested that 65 participants would provide 80% power to detect a moderate effect size ($f = 0.20$). We aimed to collect approximately 70 participants with usable EEG data.

For the present analyses, sensitivity power simulations were conducted using the powerSim function in R on mixed-effects models to estimate the observed power of the predictors. The interaction term irritability × anhedonia had an effect size of −0.030, $\alpha = 0.05$, with estimated power of approximately 72.8% (95% CI: 68.7%–76.7%). The three-way interaction feedback valence × irritability × anhedonia had an effect size of −0.014, with the same α, yielding an estimated power of approximately 65.6% (95% CI: 61.3%–69.8%). These results indicate moderate power to detect the observed effects, suggesting that smaller effects may have gone undetected.

3 | Results

3.1 | Descriptive and Correlation Results

Means, standard deviations, and bivariate correlations among variables are described in Table 2. There was a small positive correlation between BITe and DARS ($r = 0.27$, $p < 0.05$). There

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TABLE 2 | Means, standard deviations, and correlations with 95% confidence intervals among variables.

Variable	*M*	SD	1	2	3	4	5	6
1. BITe	10.44	3.00						
2. DARS	12.78	7.90	0.27*					
			[0.05, 0.47]					
3. BITe × DARS	6.40	27.74	0.31**	0.28*				
			[0.08, 0.50]	[0.05, 0.48]				
4. DASS depression	1.22	1.93	0.28*	0.38**	0.11			
			[0.05, 0.48]	[0.16, 0.56]	[−0.12, 0.34]			
5. FRN/N2	2.81	3.40	−0.10	−0.06	−0.25**	−0.06		
			[−0.25, 0.07]	[−0.22, 0.10]	[−0.39, −0.09]	[−0.22, 0.10]		
6. Theta	2.77	1.53	−0.10	−0.14	−0.16	−0.09	−0.25**	
			[−0.25, 0.07]	[−0.29, 0.03]	[−0.32, 0.00]	[−0.25, 0.07]	[−0.39, −0.09]	
7. Delta	1.48	1.44	−0.16	0.05	−0.08	−0.04	0.03	0.00
			[−0.31, 0.01]	[−0.12, 0.21]	[−0.24, 0.08]	[−0.20, 0.13]	[−0.13, 0.19]	[−0.16, 0.17]

Note: Degree of freedom for all correlations = 71.

Abbreviations: BITe, Brief Irritability Test; BITe × DARS, interaction term between BITe and DARS; DARS, Dimensional Anhedonia Rating Scale. DARS scores were reverse-coded so that higher scores correspond to higher anhedonia; DASS depression, depression subscale of the Depression Anxiety and Stress Scale; FRN/N2, feedback-related negativity.

**$p < 0.01$.

*$p < 0.05$.

were no linear associations between symptoms and neural measures averaged across feedback valences, except between the FRN/N2 and the interaction term irritability × anhedonia ($r = -0.25$, $p < 0.05$). For the correlations between ERP and TF measures, the FRN/N2 was negatively correlated with theta power ($r = -0.27$, $p < 0.01$). No other correlations were significant.

3.2 | Effect of Feedback Valence on ERP and Time–Frequency Measures

3.2.1 | ERP Results

The FRN/N2 to loss feedback was significantly larger (i.e., more negative) than the FRN/N2 to win feedback, $b = 3.99$, $\mathrm{SE} = 0.26$, $t(72) = 15.12$, $p < 0.001$.

3.2.2 | Time–Frequency Results

The univariate TF decomposition at electrode FCz (Figure 2) showed a clear increase in power compared to baseline in the theta and delta band. The theta power increase was visibly stronger in response to loss feedback relative to win feedback. Conversely, the delta power increase was slightly stronger in response to win feedback relative to loss feedback.

We applied GED to find two spatial filters that best isolate theta and delta activity from the broadband activity. Figure 3A2,B2 show the topographies of the activation patterns of the selected theta and delta spatial filters averaged across participants. For the GED-derived theta component, there was a clear activation around midfrontal electrodes. Theta power extracted from the TF decomposition of this component revealed that theta power to loss feedback (Figure 3A3; $M = 3.32$, $\mathrm{SD} = 1.55$) was significantly higher than theta power to win feedback (Figure 3A4; $M = 2.21$, $\mathrm{SD} = 1.30$), $b = -1.11$, $\mathrm{SE} = 0.16$, $t(72) = -6.70$, $p < 0.001$.

For the GED-derived delta component, an activation can be seen around central electrodes. Delta power extracted from the TF decomposition of this component revealed that delta power to win feedback (Figure 3B3; $M = 1.53$, $\mathrm{SD} = 1.41$) did not differ from delta power to loss feedback (Figure 3B4; $M = 1.42$, $\mathrm{SD} = 1.48$), $b = -0.11$, $\mathrm{SE} = 0.18$, $t(72) = -0.65$, $p = 0.52$.

3.3 | Effect of Irritability, Anhedonia, and the Irritability × Anhedonia Interaction on ERP and Time–Frequency Measures

Results of the LMMs predicting ERP and TF measures are shown in Table 3. The LMM with *feedback valence* (win vs. loss), irritability, and anhedonia as predictors of FRN/N2 (model 1) showed that neither irritability nor anhedonia showed significant main effects, nor did their interactions with feedback valence. There was a significant two-way interaction between irritability and anhedonia ($\beta = -0.03$, $\mathrm{SE} = 0.01$, $t(99) = -2.47$, $p = 0.015$; Figure 4a,b), indicating that the combined influence of these symptoms modulated FRN/N2 amplitude. This interaction was not dependent on feedback valence, as the three-way interaction with feedback valence was not significant ($p = 0.96$).

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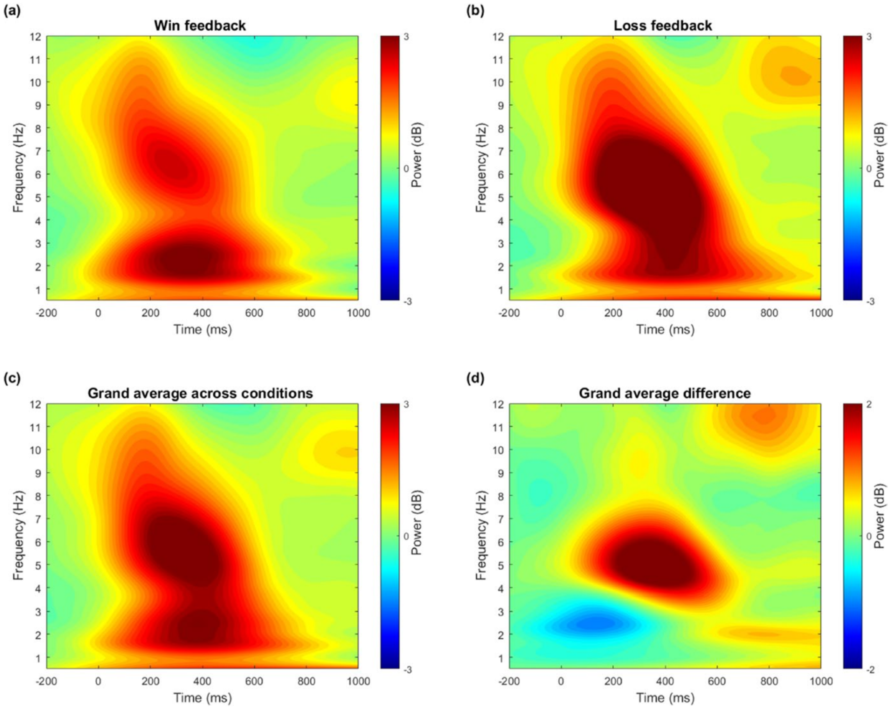

FIGURE 2 | Time–frequency power (decibel transformed) at electrode FCz. (a) Average of win trials. (b) Average of loss trials. (c) Grand average across conditions. The subject-wise average across conditions was used to find the individual-specific theta and delta frequency peaks within a time–frequency window of 4–8 Hz and 0–700 ms for theta and a time–frequency window of 1–3.9 Hz and 0–500 ms for delta. This was done on the grand average across conditions to not bias the statistical test on feedback valence difference. (d) Grand average difference computed by subtracting the averaged power of win trials from the averaged power of loss trials.

The significant irritability × anhedonia interaction on FRN/N2 amplitude was further explored using simple slopes analysis. At levels of anhedonia above the mean (+1 SD), irritability was significantly associated with larger (i.e., more negative) FRN/N2 amplitude ($b = -0.28$, $\mathrm{SE} = 0.13$, $p = 0.03$). In contrast, when anhedonia was low (−1 SD) or average, irritability was not significantly related to FRN/N2 ($b = 0.19$, $\mathrm{SE} = 0.17$, $p = 0.25$; $b = -0.04$, $\mathrm{SE} = 0.11$, $p = 0.69$, respectively). These group-level differences are illustrated in Figure 5a, which shows the FRN/N2 waveforms for four groups created using a mean split of irritability and anhedonia. We found no effect of irritability, anhedonia, or their interaction on the RewP amplitudes (S2).

The LMM with *feedback valence* (win vs. loss), irritability, and anhedonia as predictors of theta power (model 2) revealed no significant main effects of irritability ($p = 0.51$) or anhedonia ($p = 0.97$). The feedback valence × anhedonia interaction was marginally significant, $b = -0.04$, $\mathrm{SE} = 0.02$, $t(69) = -1.80$, $p = 0.076$. Importantly, the three-way interaction between feedback valence, irritability, and anhedonia was significant, $b = -0.014$, $\mathrm{SE} = 0.006$, $t(69) = -2.33$, $p = 0.023$ (Figure 4c,d), indicating that the association between feedback-related theta activity and feedback valence varied as a function of irritability and anhedonia levels. The exploratory probe of the three-way interaction revealed that the association between irritability and theta activity depended on anhedonia levels, but only in response to win feedback. Specifically, irritability was significantly associated with reduced theta power for participants with levels of anhedonia above the mean ($b = -0.18$, $\mathrm{SE} = 0.09$, $p < 0.05$). At low (−1 SD) or average levels of anhedonia, irritability was not significantly related to theta power ($b = 0.12$, $\mathrm{SE} = 0.09$, $p = 0.17$; $b = 0.01$, $\mathrm{SE} = 0.06$, $p = 0.90$, respectively). In response to loss feedback, no significant associations were found between irritability and theta at any levels of anhedonia. These patterns are reflected in Figure 5b, which depicts theta power time courses for the four mean-split groups in response to win feedback.

The LMM with *feedback valence* (win vs. loss), irritability, and anhedonia as predictors of delta power (model 3) revealed no main effects of irritability or anhedonia, or any significant two-way or three-way interactions (all $ps > 0.14$).

For all models, the inclusion of covariates (i.e., gender, combined annual income, and depression score) did not significantly improve model fit for FRN/N2, $\chi^2(3) = 4.14$, $p = 0.25$, theta, $\chi^2(3) = 1.57$, $p = 0.67$, or delta, $\chi^2(3) = 0.83$, $p = 0.84$. In all cases, the extended models showed higher AIC/BIC values, and changes in explained variance were negligible: FRN/N2 (marginal $R^2 = 0.39 \rightarrow 0.40$), theta ($0.20 \rightarrow 0.20$), and delta ($0.02 \rightarrow 0.03$), suggesting no additional explanatory value of the covariates. The effects reported above remained statistically significant when controlling for those covariates.

4 | Discussion

This study aimed to evaluate the unique and interacting effects of irritability and anhedonia symptoms on neural measures of

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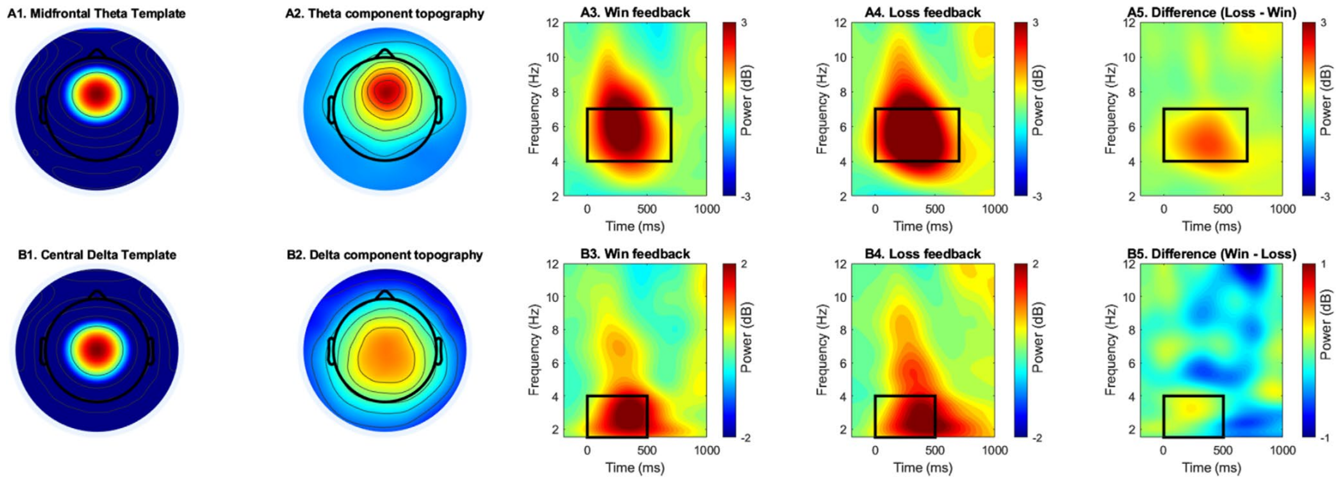

FIGURE 3 | (A1) Template topography used to select midfrontal theta components. (A2) Topographical map showing the activation pattern of the theta spatial filter defined by GED. Time–frequency power (decibel transformed) of the theta component defined by GED for win trials (A3), loss trials (A4), and the difference computed by subtracting the averaged power of win trials from the averaged power of loss trials (A5). (B1) Template topography used to select central delta components. (B2) Topographical map showing the activation pattern of the delta spatial filter defined by GED. Time–frequency power (decibel transformed) of the delta component defined by GED for win trials (B3), loss trials (B4), and the difference computed by subtracting the averaged power of loss trials from the averaged power of win trials (B5). Black rectangles show the time–frequency windows used to extract theta activity to win and loss feedback (A3–A4) and delta activity to win and loss feedback (B3–B4).

reward sensitivity in young adults. To this end, we measured time-domain (FRN/N2) and TF (theta and delta power) EEG responses to win and loss in a traditional reward sensitivity task using both univariate and multivariate methods. This was the first study looking at the extent to which individual symptoms (i.e., anhedonia and irritability) modulate EEG responses to reward and loss. We found interacting effects of anhedonia and irritability on theta power to win and the FRN/N2. These interaction effects remained significant when controlling for sociodemographic characteristics as well as general depressive symptoms, suggesting that these effects may be specific to irritability and anhedonia symptoms. These findings provide preliminary evidence of an interactive effect of both symptoms on neural sensitivity to reward and loss in a community sample of young adults. Irritability may moderate the association between anhedonia and reward sensitivity, and vice versa. This highlights the importance of considering symptom interactions when studying reward sensitivity.

Our task-related results replicate prior findings in both the time and the TF literature on reward responsivity. That is, the expected effect of feedback valence (win vs. loss) on the FRN/N2 amplitude was observed, such that the FRN/N2 was more negative in response to loss than to win (e.g., Bowers et al. 2018; Proudfit 2015; Williams et al. 2021). In addition, we examined neural activity in response to feedback by performing a multivariate source separation method and identified, individually for each participant, a midfrontal theta component and a central delta component. Time–frequency decomposition of the GED-derived theta component revealed higher theta power in response to loss compared to win feedback, which is consistent with studies showing that theta power is primarily sensitive to negative outcomes (Bernat et al. 2015). For the GED-derived delta component, TF decomposition showed that delta power in response to win was slightly higher than in response to loss feedback, although this difference was not statistically significant. These findings suggest that the task worked as expected and help to contextualize the results associated with irritability and anhedonia.

Importantly, our findings suggest that neural measures of reward sensitivity on the Doors task are influenced by the interaction between irritability and anhedonia, with some effects being valence-specific (i.e., theta power) and others valence-independent (i.e., FRN/N2). In particular, the association between theta power and win feedback depended on individual differences in both irritability and anhedonia. Specifically, individuals presenting with both irritability and anhedonia displayed reduced theta power to win feedback. Theta oscillations in response to (both win and loss) feedback are proposed to index the motivational salience of outcomes that enhance the engagement of top-down cognitive control mechanisms that facilitate and guide goal-directed behaviors (Cavanagh and Frank 2014). Notably, theta power correlates with trial-by-trial behavioral adjustments (Cavanagh and Shackman 2015). While theta power is typically enhanced following negative feedback, it also appears in response to reward or positive reinforcement (e.g., Doñamayor et al. 2012), suggesting that theta power reflects an unsigned prediction error that encodes outcome salience, rather than outcome valence (Cavanagh et al. 2012). Theta power following rewarding stimuli has also been shown to correlate with subsequent behavioral adaptation (Cavanagh et al. 2009) and higher engagement in reward-seeking behaviors (Gibney et al. 2023). Therefore, a potential interpretation for our finding could be that individuals with anhedonia and mean-to-high levels of irritability may interpret rewards as less motivationally relevant. This may in turn lead to reduced engagement in reward-seeking behaviors. This would be consistent with the characterization of anhedonia as a loss of interest and reduced approach motivation and effort expenditure for pleasurable activities (Horne et al. 2021). Furthermore, a network analysis examining the associations between irritability

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TABLE 3 | Linear mixed model results predicting neural measures from irritability and anhedonia.

Model with predictors	*β*	SE	Df	*t*	*p*	95% CI
Model 1: FRN/N2						
Intercept	**1.01**	**0.32**	**98.53**	**3.12**	**0.002**	**0.39 to 1.63**
Feedback valence	**3.99**	**0.28**	**69.00**	**14.36**	**<0.001**	**3.45 to 4.52**
Irritability	−0.04	0.11	98.53	−0.39	0.69	−0.26 to 0.17
Anhedonia	0.01	0.04	98.53	0.21	0.83	−0.07 to 0.09
Feedback valence × Irritability	0.03	0.09	69.00	0.34	0.73	−0.15 to 0.22
Feedback valence × Anhedonia	−0.01	0.03	69.00	−0.24	0.81	−0.08 to 0.06
Irritability × Anhedonia	**−0.03**	**0.01**	**98.53**	**−2.46**	**0.01**	**−0.05 to −0.01**
Feedback valence × Irritability × Anhedonia	0.00	0.01	69.00	0.04	0.96	−0.02 to 0.02
Model 2: Theta						
Intercept	**3.32**	**0.17**	**106.13**	**19.54**	**<0.001**	**2.99 to 3.65**
Feedback valence	**−1.02**	**0.16**	**69.00**	**−6.29**	**<0.001**	**−1.33 to −0.70**
Irritability	−0.04	0.06	106.13	−0.66	0.51	−0.15 to 0.07
Anhedonia	0.00	0.02	106.13	0.04	0.96	−0.04 to 0.04
Feedback valence × Irritability	0.05	0.06	69.00	0.83	0.41	−0.06 to 0.15
Feedback valence × Anhedonia	−0.04	0.02	69.00	−1.79	0.07	−0.08 to 0.00
Irritability × Anhedonia	0.00	0.00	106.13	0.01	0.99	−0.01 to 0.01
Feedback valence × Irritability × Anhedonia	**−0.01**	**0.00**	**69.00**	**−2.33**	**0.02**	**−0.03 to −0.002**
Model 3: Delta						
Intercept	1.53	0.17	115.01	8.78	<0.001	1.20 to 1.87
Feedback valence	−0.07	0.18	69.00	−0.38	0.70	−0.42 to 0.28
Irritability	−0.09	0.06	115.01	−1.46	0.15	−0.21 to 0.03
Anhedonia	0.02	0.02	115.01	0.70	0.49	−0.03 to 0.06
Feedback valence × Irritability	0.02	0.06	69.00	0.27	0.79	−0.11 to 0.14
Feedback valence × Anhedonia	0.01	0.02	69.00	0.33	0.74	−0.04 to 0.05
Irritability × Anhedonia	0.00	0.01	115.01	0.02	0.98	−0.01 to 0.01
Feedback valence × Irritability × Anhedonia	−0.01	0.01	69.00	−0.98	0.33	−0.02 to 0.01

Note: DARS scores were reverse-coded so that higher scores correspond to higher anhedonia. Bold font indicates significant predictors.

and various aspects of hedonic response (e.g., pleasure, desire) revealed that irritability symptoms are specifically linked to a self-reported reduced willingness to exert effort across multiple reward types (Sistiaga et al. 2024). While this aligns with the hypothesis of shared hypofunctioning of the reward system underlying both irritability and anhedonia (Zisner and Beauchaine 2016), this needs to be formally tested with paradigms that measure approach motivation and effort expenditure. Such paradigms would clarify whether the reduced theta power in response to reward effectively leads to diminished effort or energy expenditure to obtain rewards, and whether these effects are modulated by irritability and anhedonia. This was not assessed in the reward paradigm used in the present study, which precludes us from drawing conclusions about this specific mechanism.

The irritability × anhedonia interaction effect on theta power in response to reward was identified using the novel multivariate method GED. Leveraging whole-brain dense-array EEG measurements, GED allowed us to extract neural components that specifically isolate task-related frequency activity during reward receipt, separately for each participant. We found a personalized theta component that showed associations with symptoms of anhedonia and irritability. Alongside recent findings by Ohki et al. (2024), our results further suggest the potential clinical utility of the individual-specific GED component,

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as evidenced by their associations with dimensionally assessed clinical symptoms. Applying this method to extract personalized frequency peaks linked to specific aspects of reward processing offers a promising avenue to understand interindividual differences in reward mechanisms and their implications for clinical symptoms.

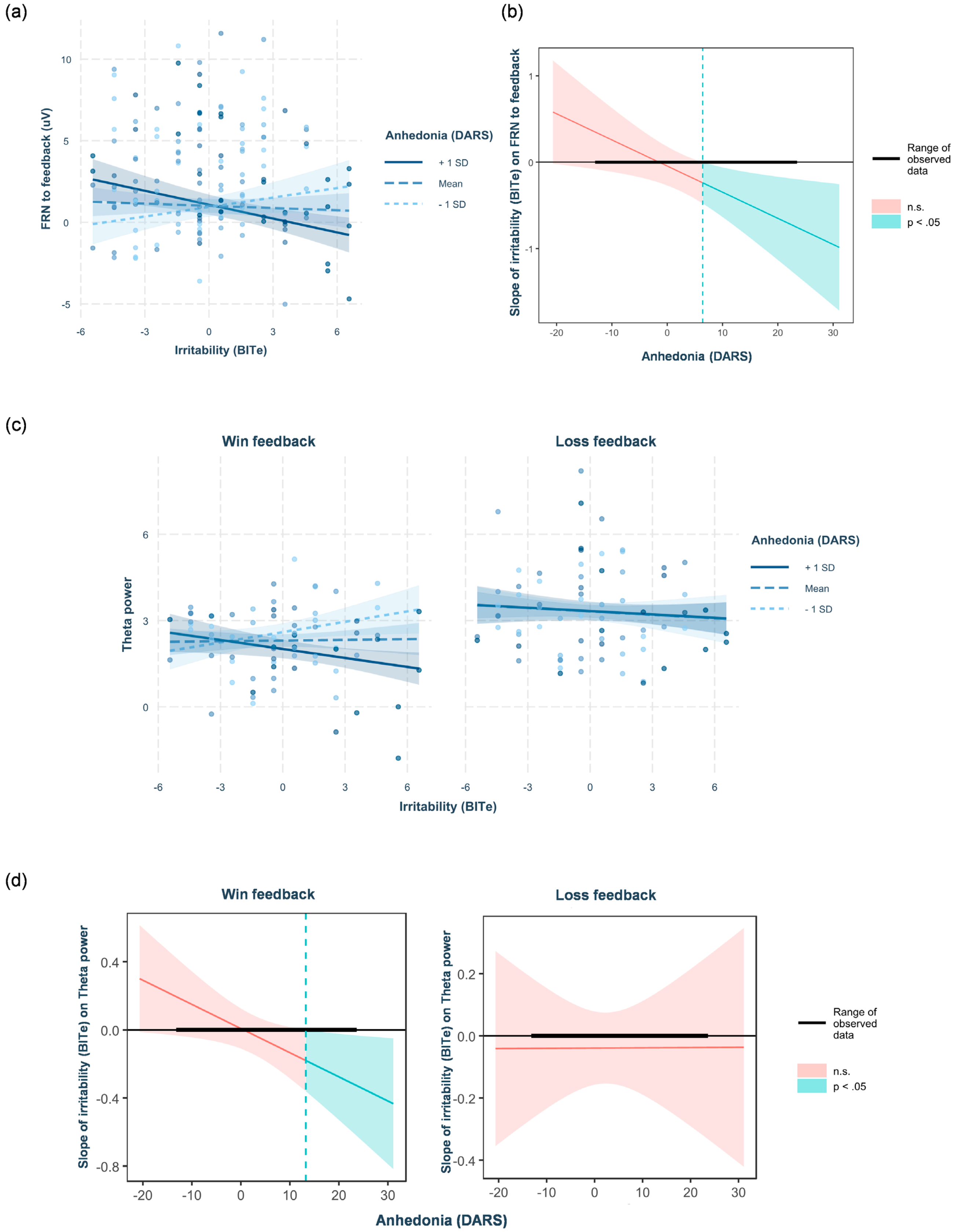

FIGURE 4 | Legend on next page.

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FIGURE 4 | (a) Interaction plot of the regression model predicting FRN/N2 from irritability and anhedonia. (b) Johnson–Neyman plot for the simple slope of irritability predicting FRN/N2 across the range of anhedonia scores. The Johnson–Neyman technique showed that irritability was significantly associated with FRN/N2 amplitude only when anhedonia scores were above 6.42, with higher irritability predicting larger (more negative) FRN/N2. At anhedonia scores below 6.42, irritability showed no significant association with FRN/N2. Note that FRN/N2 amplitude is plotted with more negative values corresponding to larger amplitudes, unlike theta power. (c) Interaction plot of the regression model predicting theta power from irritability and anhedonia, split by feedback valence. (d) Johnson–Neyman plots for the simple slope of irritability predicting theta power, split by feedback valence. The Johnson–Neyman technique showed that the association between irritability and theta power to win was significant only when anhedonia scores were above 13.24, with higher irritability predicting reduced theta power. The association between irritability and theta power to loss was not significant at any levels of anhedonia. DARS scores were reverse-coded so that higher scores correspond to higher anhedonia.

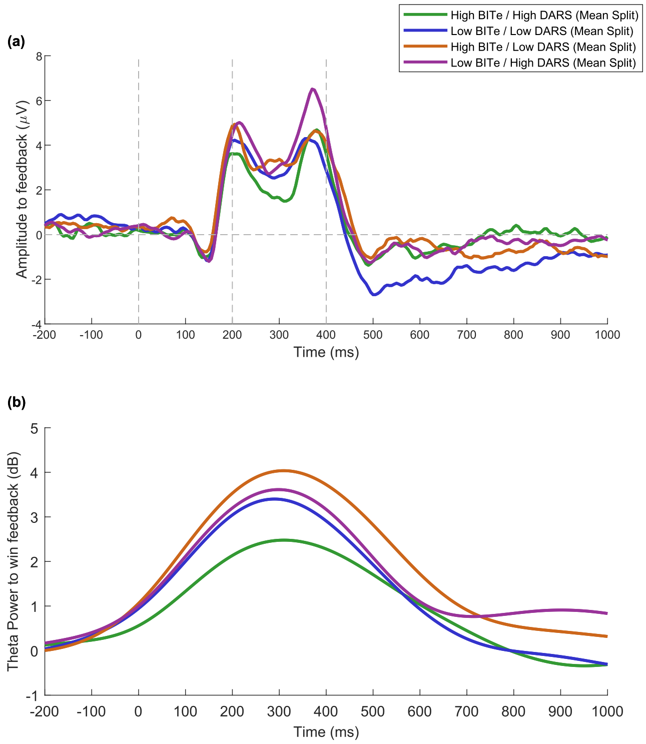

FIGURE 5 | Effects of anhedonia and irritability on reward-related EEG measures. FRN/N2 waveforms averaged across loss and feedback (a) and the time course of theta power to win feedback (b) for four groups defined by a mean split of irritability (BITe) and anhedonia (DARS): High BITe/High DARS (green), Low BITe/Low DARS (blue), High BITe/Low DARS (orange), and Low BITe/High DARS (purple). FRN waveforms represent the average amplitude across eight frontocentral channels surrounding FCz across feedback valence. Theta power in response to win feedback was extracted using GED and averaged over the 4–7 Hz frequency band and the 0–700 ms postfeedback interval.

We also found that individuals presenting with both irritability and anhedonia exhibited an overall increase in FRN/N2 amplitude to feedback, independent of feedback valence. This pattern suggests that the obligatory N2 component, which reflects a general neural response to infrequent or salient task-relevant events, is heightened in these individuals. Importantly, because no effects were observed on the RewP, which specifically indexes sensitivity to positive feedback, the observed FRN/N2 increase does not appear to reflect altered processing of rewards per se. Instead, it likely reflects a general enhancement of feedback monitoring or salience detection, independent of valence. While much of the anhedonia literature reports reduced neural responses to feedback (Borsini et al. 2020; Keren et al. 2018), the present finding of increased FRN/N2 amplitude arises in the context of co-occurring irritability. This suggests that irritability may modulate neural responses underlying feedback monitoring and salience detection. This interpretation aligns with accounts linking pediatric irritability to heightened reactivity to both reward and frustrative nonreward (Brotman et al. 2017). Individuals endorsing both symptoms may perceive feedback as particularly salient, potentially reflecting difficulties in accurately predicting outcomes and adjusting their behavior accordingly. Anhedonia has been shown to correlate with blunted RPE signaling during reward learning tasks, leading to fewer optimal choices and diminished learning from rewards (Geugies et al. 2019; Rothkirch et al. 2017). This process may be modulated by irritability, which has been theoretically linked to reinforcement learning difficulties (Brotman et al. 2017), although empirical work is limited (Adleman et al. 2011; Dickstein et al. 2010). Overall, these findings suggest that further research is needed to better understand

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how anhedonia and irritability may jointly influence feedback monitoring and reinforcement learning.

When examining the bivariate correlations between EEG measures of reward sensitivity with anhedonia symptoms, we did not find significant associations with time-domain measures. This is consistent with past studies showing that RewP or FRN/N2 in response to monetary rewards were not correlated with various measures of anhedonia in community samples of students (Banica et al. 2022; Hager et al. 2022; Irvin et al. 2020; Umemoto et al. 2019). In patients with MDD, some studies have similarly found no associations between FRN/N2 and anhedonia (Foti et al. 2014; Klawohn et al. 2021; Sun et al. 2023) while another study (Liu et al. 2014) found that reduced FRN/N2 to gain was related to anhedonia as measured by the Temporal Experience of Pleasure Scale (Gard et al. 2006) but not as measured by the Snaith-Hamilton Pleasure Scale (Snaith et al. 1995). However, these null findings contrast with previous research indicating a blunted RewP during reward receipt in depression (Keren et al. 2018). Recent evidence suggests a potential distinction between MDD and anhedonia in reward processing (Pirrung et al. 2025); in a reward learning paradigm, activity in brain areas producing the RewP (i.e., ventromedial and right ventral temporal areas) in response to reward was diminished in MDD patients compared to controls. However, activity in the same region was positively correlated with anhedonia in the MDD group. It is possible that these alterations are specific to reinforcement learning paradigms and do not apply to the simple guessing task used in this study and studies where feedback is not used in a learning context to inform subsequent behaviors. It is also possible that associations between FRN/RewP and anhedonia might be contingent on reward types, as anhedonia was associated with reduced FRN to social (Banica et al. 2022; Zhang et al. 2024) but not monetary (Banica et al. 2022) rewards. Regarding the null associations between anhedonia and TF measures of reward sensitivity, Hager et al. (2022) have similarly reported no effect of anhedonia severity on delta or theta power during reward receipt in an undergraduate sample. In a small group of MDD patients ($n = 14$), Mueller et al. (2015) also reported no association between anhedonia and theta power for negative minus positive feedback.

Similarly, irritability was not independently associated with any time-domain or TF measures of reward sensitivity. This contrasts with the one prior study of irritability and FRN/N2 and RewP amplitudes in young adults (Deveney 2019). In that study, higher irritability was associated with reduced FRN/N2 in response to loss, a finding that was not replicated in the present study. The failure to replicate may be due to the different tasks used in the two studies. Deveney (2019) used a frustration paradigm where task feedback was rigged, so that participants did not receive an expected reward. This violation of reward expectation likely influenced the FRN/N2, as this component is modulated by reward expectation (Hauser et al. 2014; Walentowska et al. 2019). In contrast, the Doors task used in our study does not explicitly build or violate reward expectations since the reward probability is consistently fixed at 50% and does not induce a feeling of agency (i.e., the perception that one's own action impacts the outcome). This may explain the discrepancies between our findings and those of Deveney (2019).

It is important to note that outliers ($n = 4$ on the BITe and $n = 1$ on the DARS) were identified and excluded from the regression analyses of the study using a very conservative outlier detection method (Leys et al. 2013). This was done because linear regression coefficients are highly sensitive to outliers (Montgomery et al. 2021). When these outliers were retained, the LMMs predicting neural measures from irritability and anhedonia were not significant (see Table S1). Consequently, the observed linear associations may only apply to individuals from the general population who do not present with clinical levels of irritability and anhedonia.[1] Future studies incorporating samples enriched for irritability and anhedonia, spanning the full dimensional spectra of these symptoms, are needed to determine how these associations manifest at the higher end of the continuum. Preliminary findings suggest the potential presence of nonlinear associations between irritability, anhedonia, and EEG measures of reward sensitivity. For anhedonia, Mueller et al. (2015) identified an inverted U-shaped relationship between anhedonia severity and the FRN/N2 amplitude in response to negative feedback, indicating that moderate depression without severe anhedonia is linked to increased sensitivity to negative feedback. For irritability, Grabell et al. (2018) observed nonlinear associations between irritability and brain activity in young children during a frustration task where a desired/expected reward was taken away. Specifically, results showed that irritability was positively associated with lateral prefrontal cortex (LPFC) activation during frustration within the normative ranges of irritability, but negatively associated with LPFC activation at the severe end of the irritability spectrum. These findings highlight the need for further exploration of potential nonlinear dynamics in the relationships between reward sensitivity and irritability/anhedonia.

Our results should be considered in light of the following limitations. First, our sample was community-based. Therefore, the generalizability of our results is limited to this specific population and, as discussed earlier, the full irritability and anhedonia spectra may not be represented. Accordingly, our findings should be understood as reflecting associations in a nonclinical context and cannot be generalized to clinical phenotypes in which anhedonia is typically more severe and heterogeneous (Cooper et al. 2018). Rather, the present study provides insight into subclinical manifestations of these symptoms, while future work in clinical groups sampling for high irritability and anhedonia levels will be necessary before any potential clinical implications can be drawn. Second, an important proportion of participants (14.29%) were excluded because our EEG hardware was incompatible with certain hair types (e.g., coarse hair) or hairstyles (e.g., braids, cornrows). As these participants were predominantly Black American, our results may not generalize to this population. Future work should attend to the considerations to increase racial and ethnic representativeness in EEG research recently proposed in the literature (Choy et al. 2021; Penner et al. 2023). Third, our sample was from a high socioeconomic status. Although our results remained significant when controlling for individual and family annual income, this study should be reproduced in more representative samples. Fourth, the version of the Doors task we used has a relatively small number of trials (i.e., 60), which may have reduced the reliability of the FRN/N2 (Marco-Pallares et al. 2011). Finally, the study had only moderate power to detect the observed interaction effects, and future studies with larger samples should aim

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to replicate these findings and further explore smaller effects that may not have been detected.

5 | Conclusions

We found interacting effects of irritability and anhedonia on ERP and TF measures of reward sensitivity in a nonclinical sample, via univariate and novel multivariate approaches. Overall, our findings suggest that individuals with co-occurring irritability and anhedonia may perceive feedback (both reward and loss) as more salient, while interpreting *rewards* as less motivationally relevant, highlighting the importance of examining symptom interactions. Using this approach to investigate other aspects of reward processing (effort expenditure and reward learning) could be an important next step for understanding how alterations in reward sensitivity specific to anhedonia and irritability influence subsequent behavior and motivation. This may ultimately inform our comprehension of the reward-related mechanisms underlying clinical conditions in which both symptoms are prominent (e.g., MDD, schizophrenia, bipolar disorder). While our focus was on anhedonia and irritability, other depressive symptoms (e.g., fatigue, sad mood) may also modulate reward-related neural responses and should be considered in future research.

Author Contributions

Nellia Bellaert: conceptualization, investigation, funding acquisition, writing – original draft, methodology, visualization, software, formal analysis. **Federico Cassioli:** formal analysis, writing – review and editing. **Michael J. Crowley:** writing – review and editing, supervision, resources. **Yi Voon Lim:** writing – review and editing, investigation. **Mandy Rossignol:** conceptualization, supervision, writing – review and editing, funding acquisition. **Christen M. Deveney:** conceptualization, supervision, writing – review and editing. **Wan-Ling Tseng:** conceptualization, funding acquisition, resources, supervision, writing – review and editing.

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Ethics Statement

The study was approved by the Institutional Review Board of Yale University (HIC#: 2000031088).

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data and code that support the findings of this study are openly available on the Open Science Framework: https://osf.io/97wks/.

Endnotes

[1] The raw score (not reverse-coded) of the DARS outlier was below the recommended clinical cutoff value of 28.5 (Lin et al. 2022), which suggests that this participant had clinical levels of anhedonia. No clinical cutoff is defined for the BITe (Holtzman et al. 2015).

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