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Feedback negativity and feedback-related P3 in individuals at risk for depression: Comparing surface potentials and current source densities

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Abstract

Blunted responses to reward feedback have been linked to major depressive disorder (MDD) and depression risk. Using a monetary incentive delay task (win, loss, break-even), we investigated the impact of family risk for depression and lifetime history of MDD and anxiety disorder with 72-channel EEGs recorded from 29 high and 32 low risk individuals (15-58 years, 30 male). Linked-mastoids surface potentials (ERPs) and their corresponding reference-free current source densities (CSDs) were quantified by temporal principal components analysis (PCA). Each PCA solution revealed a midfrontal feedback negativity (FN; peak around 310 ms) and a posterior feedback-P3 (fb-P3; 380 ms) as two distinct reward processing stages. Unbiased permutation tests and multilevel modeling of component scores revealed greater FN to loss than win and neutral for all stratification groups, confirming FN sensitivity to valence. Likewise, all groups had greater fb-P3 to win and loss than neutral, confirming that fb-P3 indexes motivational salience and allocation of attention. By contrast, group effects were subtle, dependent on data transformation (ERP, CSD), and did not confirm reduced FN or fb-P3 for at-risk individuals. Instead, CSD-based fb-P3 was overall reduced in individuals with than without MDD history, whereas ERP-based fb-P3 was greater for high than low risk individuals for monetary, but not neutral outcomes. While the present findings do not support blunted reward processing in depression and depression risk, our side-by-side comparison underscores how the EEG reference choice affects the characterization of subtle group differences, strongly advocating the use of reference-free techniques.

Our findings add nuanced ERP evidence of blunted versus preserved reward processing in depression and depression risk. Whereas robust condition effects (win, loss, neutral feedback) were congruent with prior research, our comparative side-by-side analysis of ERPs and their current source density transformations using permutation tests and multilevel modeling revealed that the characterization of subtle group differences is impeded and potentially altered by the EEG reference choice, underscoring the importance of using reference-free techniques for analyzing ERPs.

Keywords: depression risk, EEG reference, current source density (CSD), principal components analysis (PCA), reward processing, feedback negativity (FN)

1. Introduction

Positive valence systems, one of the core domains in the NIMH Research Domain Criteria (RDoC; Cuthbert, 2014; Cuthbert & Insel, 2013), primarily pertains to responses to positive motivational situations or contexts, such as reward responsiveness, consummatory behavior, and habit learning. Response to reward has been studied widely with various paradigms and psychophysiological methodologies (e.g., Kessel et al., 2017; Kujawa et al., 2020; Nusslock & Alloy, 2017). Rewards have been operationally defined via stimuli for which organisms expend effort to approach or acquire (Rolls, 2000; Schultz, 2016). These may indicate primary or secondary rewards or may form part of a more complex sequence of goal-directed behaviors (Haber & Knutson, 2010; Schultz, 2000). A wealth of research using psychophysiological measures, especially electroencephalogram (EEG), has shown that sensitivity to reward is altered in various psychiatric disorders, specifically depression (e.g., Babinski et al., 2019; Bedwell et al., 2016; Berry et al., 2019; Burkhouse et al., 2016; Hixson et al., 2019; Umemoto et al., 2014; Weinberg, 2022).

Taking advantage of the high temporal resolution of EEG measures, reward feedback processing can be teased apart by studying event-related potential (ERP) components during feedback anticipation and processing. At the stage of feedback processing, the earliest of these consummatory ERP components is elicited by the initial evaluation of reward feedback and outcome valence. In response to the presentation of feedback signaling favorable or unfavorable events, this ERP component peaks at approximately 200 to 300 ms post feedback at frontocentral sites when using a linked-mastoids EEG reference (e.g., Bress et al., 2015; Burkhouse et al., 2018; Novak et al., 2016). It is characterized by a relative negativity at frontocentral scalp sites referred to as feedback negativity (FN) or feedback-related negativity (FRN), or reward positivity (RewP) depending on how this ERP component was conceptualized and computed: FN and FRN reflect loss-minus-win, whereas RewP indicates win-minus-loss (also gain-minus-loss; e.g., Bowyer et al., 2022; Bress et al., 2015; Burkhouse et al., 2018; Novak et al., 2016; Proudfit, 2015). This report will refer to this ERP component as FN. As an early neural correlate of reward response, a more negative FN amplitude reflects negative outcome valence after unfavorable compared with favorable or neutral feedback stimuli (e.g., Chang et al., 2020; Foti et al., 2014; Foti & Hajcak, 2009; Novak et al., 2016). In reward conditions, the FN was found to be more prominent (i.e., more negative) following losses than wins (Carlson et al., 2011; Foti et al., 2015; Nelson et al., 2016; Novak et al., 2016), and it has been suggested that FN is an indicator of *outcome valence* (i.e., receiving a positive or negative

feedback; Novak et al., 2016; Pegg et al., 2021).

Importantly, the FN has also been considered a putative neural biomarker for depression, and several studies found that the FN response to win and the FN difference between win and loss was significantly reduced in depression (e.g., Brush et al., 2018; Chang et al., 2020; Foti et al., 2014; Glazer et al., 2018; Keren et al., 2018; Liu et al., 2014; Thoma et al., 2015). Moreover, this effect was more pronounced in individuals with more severe depression symptoms (Nelson et al., 2016). In addition, similar findings were observed in adolescents and children with depression (e.g., Belden et al., 2016; Bress et al., 2012), and FN has been directly linked to familial transmission (Moser et al., 2018). Furthermore, the FN has predicted treatment outcome (e.g., for cognitive behavioral therapy [CBT] and selective serotonin reuptake inhibitor [SSRI] medication), with increased FN associated with improvement of depressive symptoms (Burkhouse et al., 2016, 2018). However, some studies, including two meta-analyses (Clayson et al., 2020; Moran et al., 2017), found no consistent association between FN amplitude and depression and suggested FN to be a weak biomarker for predicting reduction of depressive symptoms and treatment outcome (Ait Oumeziane & Foti, 2016; Berry et al., 2019). On the other hand, Weinberg et al. (2015) reported evidence that blunted FN to reward is inheritable, related to lower levels of Positive Affect (e.g., Watson et al., 1988), and a putative predictor of future depression. Moreover, FN was identified as a moderator for the effects of transmission of maternal depression to their offspring (Kujawa et al., 2019; see also Weinberg, 2022).

Immediately following the FN, a positive-going ERP component termed feedback P300 (fb-P3) captures the sensitivity to reward salience, that is, an increased response on monetary trials compared to neutral trials (e.g., Ait Oumeziane et al., 2019; Novak et al., 2016; Pornpattananangkul & Nusslock, 2015). The maximum of fb-P3 occurs approximately 350 to 450 ms after feedback onset over centroparietal sites (Ait Oumeziane et al., 2019; Novak & Foti, 2015). It has been suggested that fb-P3 particularly indexes the *motivational* salience of feedback instead of performance feedback, showing overall greater amplitude in monetary trials regardless of feedback outcome (i.e., win or loss; Foti & Hajcak, 2009; Hajcak et al., 2007; Novak & Foti, 2015; Tunison et al., 2019) and also for distant future reward (Glazer et al., 2018). Some reports have argued that fb-P3 reflects allocation of attention to uncertainty or violation of expectation, such that fb-P3 was larger to monetary trials with uncertain outcomes than neutral trials in which break-even is ensured (Foti & Hajcak, 2009; Novak et al., 2016; Novak & Foti, 2015; Song et al., 2020). Accordingly, blunted fb-P3 may then indicate reduced allocation of attention to uncertainty in monetary trials.

With regard to depression, studies have found fb-P3 to be inversely related to depressive symptoms in trials offering a chance of monetary gains, suggesting deficient motivational salience for these reward stimuli (e.g., Ait Oumeziane et al., 2019; Chang et al., 2020; White et al., 2021; Zhang et al., 2020). To the extent that fb-P3 is related to a common oddball P3b (e.g., Polich, 2007) or the late positive potential to arousing stimuli (e.g., Olofsson et al., 2008), both considered an index of motivated attention (e.g., Bradley, 2009; Kayser et al., 2017; Vuilleumier & Driver, 2007), its reduction in depression is consistent with prior findings (e.g., Bruder, Kayser & Tenke, 2012; Kayser et al., 2000). In a study using clinical trial data employing certain forms of behavior therapy (White et al., 2021), fb-P3 amplitude was an indicator of treatment completion outcome: individuals who completed the treatment had a reduced fb-P3 amplitude before treatment than those who did not complete treatment. Furthermore, the amplitude of fb-P3 was found to be particularly reduced in depression to like/dislike media feedback (thumbs-up or -down picture) compared to monetary win or loss (Ait Oumeziane et al., 2019; Zhang et al., 2020), suggesting group-specific differences in attention allocation between social and monetary reward. However, as with FN, several studies have failed to find reduced fb-P3 in depression (e.g., Bowyer et al., 2022; Santopetro et al., 2021; Thoma et al., 2015).

Despite numerous published ERP reports investigating reward processing in depression, considerable discrepancies among EEG methodologies hinder integration of study results. One persistent impediment in ERP research in general is that surface potentials are dependent on the EEG reference, however, this choice is arbitrary (e.g., Kayser & Tenke, 2010). Among the EEG studies on reward feedback processing in depression, the majority applied ERP analyses with a linked-mastoids (e.g., Bowyer et al., 2022; Bress et al., 2015; Burkhouse et al., 2018; Novak et al., 2016; Santopetro et al., 2021; Weinberg et al., 2015) or common average surface potential reference (Meyer et al., 2021; Thoma et al., 2015). However, the reference-dependence can be easily overcome by transforming ERPs into scalp current source density (CSD), rendering reference-free signals in the same temporal dimension (Kayser & Tenke, 2015b). A CSD transform, also referred to as the scalp surface Laplacian, provides estimates of radial current flow at scalp (sinks and sources) that have sharper topographies and improved temporal resolution compared to ERP signals smeared by volume conduction (Burle et al., 2015; Nunez & Srinivasan, 2006; Tenke & Kayser, 2012). Although direct systematic comparisons have demonstrated superior statistical properties of CSD over ERP signals (e.g., Kayser & Tenke, 2006a, 2006b, 2015a; Zhang et al., 2023), in addition to their theoretical and interpretational advantages, to the best of our knowledge, no such comparison has been performed for ERP components related to reward feedback, even though a few studies have

employed CSD methods (Kamarajan et al., 2015; Nelson & Jarcho, 2021). This study aimed to address this shortcoming. To be sure, we did not intend to reevaluate the known benefits of CSD over ERP methods which are generic and not specific to any particular component (e.g., see Kayser & Tenke, 2015a, 2015b, 2015c; Zhang et al., 2023). Rather, we aimed to provide a side-by-side comparison of findings stemming from reference-dependent versus reference-free data transformations to inform the field in an area of research that has yet to adopt reference-free techniques more broadly (Kayser & Tenke, 2010). Nonetheless, we also probed if any effect differences between ERP and CSD component estimates were statistically significant.

ERP research in reward processing has employed different paradigms to elicit FN and fb-P3 components but little effort has been made to understand the impact of task-specific feedback. Whereas many studies have employed a variation of a gambling or guessing paradigm, such as the doors task (e.g., Proudfit, 2015), in which the outcome (money win or loss) is predetermined and entirely out of the participant's control, other commonly-used paradigms, such as the monetary incentive delay task (e.g., Novak & Foti, 2015), link outcome to successful behavioral performance, thereby assuring task engagement. Although a meta-analysis by Moran et al. (2017) suggested that gambling/guessing tasks yielded a smaller FN amplitude in depression compared with other tasks that included reinforcement learning, Clayson et al. (2020) emphasized that effect size heterogeneity might be also due to reasons that could undermine attempts to identify effect moderators (e.g., selective reporting of analyses, omission of non-significant effects, or other questionable research practices). An NIMH advisory council evaluating positive valence system tasks for RDoC noted that reward value and reward expectancy are possibly conflated in the monetary incentive delay task (National Advisory Mental Health Council Workgroup on Tasks and Measures for Research Domain Criteria, 2016). However, in the absence of strong empirical grounds to favor one specific reward processing ERP paradigm over another, we opted to employ the monetary incentive delay task that demonstrably tracks consummatory processing stages by distinctly eliciting FN and fb-P3 (e.g., Ait Oumeziane et al., 2019; Novak & Foti, 2015; Novak et al., 2016).

Accordingly, the present study had two primary objectives. The first goal was to examine whether monetary reward feedback processing is blunted in individuals with high family risk for depression and those with a lifetime history of major depressive disorder (MDD) or anxiety disorder (Kayser et al., 2017). Thus, we expected 1) reduced loss-minus-win FN and 2) reduced fb-P3 (win/loss versus neutral) in high compared to low risk individuals and in those with compared to those without an MDD history. Given mixed prior evidence that suggested reduced

FN in high compared with low trait anxiety individuals (Gu, Huang & Luo, 2010), no relation between FN and fear-based anxiety symptoms (Burkhouse et al., 2017) or, compared to depressive symptoms, an inverse relationship between FN and social anxiety symptoms (Nelson & Jarcho, 2021), we had no specific hypotheses regarding anxiety disorder.

The second goal was to compare the psychophysiological findings using surface potentials alongside their reference-free CSD counterparts. Given its frequent use in this line of ERP research, we opted to use a linked-mastoids reference scheme for the CSD comparison, noting that the use of other EEG reference schemes (e.g., common average, nose) will yield divergent ERP but identical CSD findings (Kayser & Tenke, 2015b).

Finally, we used a multivariate, data-driven approach to identify and quantify ERP and CSD reward processing components of interest (Kayser & Tenke, 2003, 2006a). Compared to more traditional approaches, such as baseline-to-peak or integrated time window amplitudes, principal components analysis (PCA) provides an unbiased means to yield orthogonal component estimates, meaning that PCA-based FN and fb-P3 measures will be uncorrelated and will not share variance. Moreover, there is ample evidence demonstrating that PCA-based measures have more favorable statistical properties compared to conventional component measures, including larger effect size and reliability (e.g., Beauducel et al., 2000; Beauducel & Debener, 2003; Kayser & Tenke, 2015a; Kayser, Tenke, & Bruder, 1998). Varimax-rotated temporal PCA components can be conceived as weighted time window amplitudes that summarize ERP and CSD data better than conventional a priori or post hoc measures (Kayser & Tenke, 2003).

2. Method

2.1 Participants

The sample consisted of 61 working- or middle-class individuals (Hollingshead, 1975) who were between 15 and 58 years old (*Median* = 25; *Mean* ±*SD* = 30 ±13; age < 18, *n* = 8). Most participants were non-Hispanic (*n* = 56) and Caucasian (*n* = 58). Participants were enrolled in a multi-generational, longitudinal study of families at high and low risk for depression that employed stringent selection and assessment procedures as detailed in prior reports (Talati et al., 2013; Weissman et al., 2016a, 2016b). Probands (i.e., the first generation) were initially selected for the presence or absence of a lifetime history of major depressive episode (MDD)

from outpatient psychiatric clinics and their urban community in New Haven, CT (Weissman et al., 1992, 1997). The current participants were a representative subsample (~26%) of all biological descendants (i.e., offspring in the second and third generation) of the original probands after excluding spouses of the second generation. Although data were collected from 62 participants, one person had to be excluded due to an unacceptably low signal-to-noise ratio in ERPs consisting of too few valid and artifact-free trials (6 for win, 3 for loss).

To the extent possible, all clinical assessments were repeated, or employed highly comparable instruments, across up to seven longitudinal waves spanning more than 30 years. All probands, their spouses, children, and grandchildren (born after Wave 2) received a semi-structured interview by mental health professionals who had demonstrated high inter-rater reliability and who were blind to the clinical status of participants in previous generations. Diagnoses were based on age-appropriate versions of the Schedule for Affective Disorders and Schizophrenia-Lifetime Version (Kaufman et al., 1997; Mannuzza et al., 1986; Orvaschel et al., 1982) using the best estimate procedure (Leckman et al., 1982) which involved an independent review of all assessment materials by experienced clinicians. Second and third generation participants were assessed if older than 5 years, with both parents and children reporting separately on symptoms for participants under 18. Assessments at each wave covered the time period since the previous interview. Accordingly, the overall assessment included birth to most recent interview, resulting in lifetime diagnosis. Participants who are descendants from the depressed probands were classified as high family risk for depression, whereas descendants from the healthy control participants of Wave 1 were classified as low family risk for depression. To be eligible for the monetary incentive delay task and an emotional hemifield task (e.g., Kayser et al., 2017), for which data were collected in the same session but which are not the focus of the present report, participants had to be older than twelve years and without a history of seizures, head trauma or psychosis.

As summarized in Table 1, the 61 participants were stratified by family risk, lifetime history of MDD, and lifetime history of anxiety disorder (ANX), which yielded approximately even cell sizes with regard to sex for each stratification. There were no significant differences in age or education between stratification groups. All 61 participants completed the Edinburgh Handedness Inventory (EHI; Oldfield, 1971): 48 were right-handed (EHI laterality quotient [LQ] > 40), 3 were left-handed (LQ < −40), and 10 were ambidextrous (−40 ≤ LQ ≤ 40). There were no significant differences in handedness categorization for any stratification group (all $\chi^2_{[1]} \leq 4.80$, all $p \geq 0.09$), nor did groups differ in the degree of handedness (Tab. 1).

<insert Table 1 about here>

Of the 61 participants, 60 completed the Patient Health Questionnaire-9 (PHQ-9; Kroenke et al., 2001), yielding a mean total score of 4.9 ±4.8 and a range from 0 to 18. PHQ-9 scores were significantly higher in participants with a lifetime diagnosis of MDD or anxiety disorder compared to those without, but there were no significant differences between participants at high versus low risk (Tab. 1). The same 60 participants also completed the Inventory of Depression and Anxiety Symptoms-II (IDAS-II; Watson et al., 2012). The IDAS-II subscales of General Depression (GD) and Social Anxiety (SA) revealed mean total scores of 40.6 ±12.8 (range 22 to 76) and 9.4 ±4.4 (range 6 to 27). IDAS-II GD scores were greater for participants at high than low risk and for those with than without a lifetime diagnosis of MDD; IDAS-II SA scores were also greater for participants with than without a lifetime diagnosis of MDD (Tab. 1). Two-way analyses of variance (ANOVAs) also showed no significant effects of sex and sex × stratification group for all symptom severity scores (all $F_{[1,56]} \leq 3.66$, all $p \geq 0.06$).

Past use of psychotropic medication (i.e., sedatives, tranquilizers, stimulants, antidepressants, anticonvulsants, and lithium, but excluding over-the-counter drugs) was reported by 19 participants (31%); however, there were no subgroup differences between risk and lifetime diagnosis of MDD or ANX (all $\chi^2_{[1]} \leq 0.90$, all $p \geq 0.34$). Although uncommon, past use of stimulants differed between low and high risk ($n = 5$ vs. $n = 0$; $\chi^2_{[1]} = 4.77$, $p = 0.029$). As expected, there were more participants with a lifetime diagnosis of MDD than those without who had used antidepressants in the past ($n = 12$ vs. $n = 3$; $\chi^2_{[1]} = 5.71$, $p = 0.017$).

All participants had normal or corrected-to-normal visual acuity using a Snellen chart. EEG testing was performed at the Psychophysiology Laboratory at New York State Psychiatric Institute (NYSPI) during Wave 7. All procedures were approved by the institutional review boards at Yale University and NYSPI/Columbia University. All participants gave written informed consent (≥ 18 years) or provided written assent (< 18 years; written informed consent from parents).

2.2 Paradigm and procedure

The monetary incentive delay (MID) paradigm was originally developed for fMRI studies (Knutson et al., 2000, 2001) and later adapted for EEG studies (Novak & Foti, 2015). Figure 1 schematically depicts the paradigm structure with sequence and timing of the stimuli and

response. Participants were seated in a sound-attenuated booth in front of a 20-inch monitor. On each trial, participants were first presented with a cue indicating whether they would win or lose money (dollar sign in a blue circle) or would break even regardless of their response (white circle). The cue was then followed by a variable anticipation interval (2000 to 2500 ms) of the target, a white square. Participants were instructed to press a button on a response pad with their preferred hand as quickly as possible after target onset. To win or avoid losing money the response had to occur while the target was visible. The target was presented initially for 200 ms, however, the exposure duration was continuously adjusted by ±10 ms depending on whether their response time exceeded or deceeded the target duration. The maximum target exposure time was set to 400 ms with an observed minimum of 140 ms. After the target offset, participants anticipated the feedback for 500 ms. Responses occurring after the end of the anticipation period were classified as invalid and hence recoded as missing. On trials that started with a neutral cue, the feedback was always “$0”, indicating no change in the amount of money they would receive. On trials that started with a monetary cue, a green up arrow was presented if participants responded within the target latency, indicating that they won 20 cents; a red down arrow was presented if participants responded after target offset, indicating that they lost 10 cents. Depending on the participants’ performance on the preceding trial, the target latency was shortened by 10 ms if they succeeded (i.e., won 20 cents) on the previous trial, or the target latency was extended by 10 ms if they failed (i.e., lost 10 cents). The success rate on monetary trials was thereby approximately balanced at 50%. A task practice consisted of 10 trials shuffled with 8 monetary and 2 neutral trials. Participants then completed 70 test trials shuffled with 50 monetary and 20 neutral trials which were divided into two testing blocks. Participants were informed about the total amount of money they won at the end of all trials.

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2.3 Data acquisition, recording, and artifact procedures

All critical EEG recording and data processing procedures have been detailed previously (Kayser et al., 2016). Briefly, 72-channel continuous EEGs were acquired at 1024 samples/s with a 24-bit recording system (BioSemi, Inc., 2001), followed by offline re-referencing to Nose, down-sampling to 256 samples/s and removal of volume-conducted blink artifacts (Neuroscan Inc., 2003), screening for electrolyte bridges (Alschuler et al., 2014; Tenke & Kayser, 2001), and identification and interpolation of residual EEG artifacts on a channel-by-channel and trial-by-trial basis (Kayser & Tenke, 2006a, 2006b). Cardiac artifacts were removed via EEGLAB

(Delorme & Makeig, 2004) by removing heartbeat-related ICA components. ERP waveforms were high-pass filtered at 0.1 Hz (zero-phase shift 24 dB/octave). Finally, all ERPs were re-referenced to linked mastoids (TP9/10) for direct comparison of findings with prior research (e.g., Foti et al., 2014).

Across all participants, 115 trials with a reaction time of less than 100 ms were classified as a premature response (about 2.7%) and hence excluded from the electrophysiological data analysis. Monetary trials with an invalid response time (response after feedback anticipation) were also excluded, however, neutral trials with invalid response times were included because the response time for these trials might have resulted from lacking an incentive to respond at all. Given a maximum of 70 total trials (50 win/loss combined, 20 neutral), the final means (±SD) of valid win, loss and neutral trials used for ERP averaging were 21.9 ±3.3, 19.2 ±4.2, and 17.4 ±2.2, respectively. Although all conditions differed significantly from each other (all $p \leq .002$, Bonferroni-adjusted pairwise comparisons), there were no significant interaction effects involving condition and stratification group or sex (all $F_{[2,114]} \leq 1.11$, all $p \geq 0.33$).

2.4 Current source density (CSD) and principal components analysis (PCA)

Full details regarding surface Laplacian transformation of ERPs using spherical splines (Perrin et al., 1989) and their multivariate data-reduction using temporal PCA (Kayser & Tenke, 2003) have been described previously (Kayser et al., 2016). Briefly, ERP waveforms were transformed into CSD estimates (Kayser & Tenke, 2006a, 2006b, 2015b). In contrast to conventional surface potential (ERP) measures, CSD measures are not biased by the imposition of a recording reference, nor by smearing due to volume-conduction, allowing localized activity to be placed in the context of full topography (Kayser & Tenke, 2015b; Tenke & Kayser, 2012).

Both reference-dependent ERPs (linked-mastoids reference) and reference-free CSDs were separately submitted to a covariance-based temporal PCA, followed by unrestricted Varimax rotation (Kayser & Tenke, 2003, 2005) to determine their common sources of variance. The extracted factor loadings reflect the strength of activation over time, and the corresponding factor scores (i.e., their amplitude and sign) reflect the weight and polarity of a given factor (with respect to the grand mean) for each observation (Kayser & Tenke, 2003). Because reference-free CSDs largely mitigate signal distortion due to volume conduction (Carvalhaes & de Barros, 2015; Kayser & Tenke, 2015b; Tenke & Kayser, 2012), CSD-PCA factors, in contrast to their ERP counterparts, directly reflect neuronal generator patterns at scalp (i.e., summary estimates of radial current flow).

2.5 Reliability of component estimates

Given recent discussions and concerns about scientific rigor and data replicability (e.g., Kappenman & Keil, 2017; Larson & Moser, 2017), we quantified the robustness of these PCA-based measures in terms of their reliability. Following recommendations by Thigpen, Kappenman, and Keil (2017), we obtained internal consistency estimates for each component via standardized Cronbach's alpha (Cronbach, 1951), using the three MID conditions as “test” items. Construct validity of FN and fb-P3 was evaluated via convergent and discriminant validity estimates, which we based on Pearson correlations between the ERP- and CSD-based component scores (see Campbell & Fiske, 1959; Smith et al., 2020).

2.6 Statistical analyses

Statistical analyses were conducted in parallel fashion for the temporal PCA (tPCA) solutions using either ERP waveforms with linked-mastoids (LM) reference (ERP-tPCA) or CSD waveforms (CSD-tPCA). To evaluate the main hypotheses concerning the effects of family risk for depression and lifetime diagnosis of MDD or ANX on ERP/CSD effects in the MID paradigm, a two-pronged approach was employed. First, the experimental effects of reward (i.e., win, loss, or neutral) were evaluated for the factor scores of relevant PCA components via unbiased permutation tests, as detailed previously (Kayser et al., 2007, 2016). These randomization tests do not require any assumption about the data distribution (Huo et al., 2014; Maris, 2004) and simultaneously probe the entire topography. This procedure thereby guided the selection of electrode sites for parametric statistical analysis, as detailed previously (Kayser et al., 2016). In cases where multiple clusters of sites were significant in randomization tests, the selection was based on prior literature. Because the identified electrode subsets were deemed a “representative” measure for each component, the corresponding scores were pooled across the selected sites (regions; e.g., Kayser & Tenke, 2006a) for each component (FN, fb-P3) and transformation (ERP, CSD).

Second, we used multilevel modeling (MLM) with age and sex as covariates to test main and interaction effects of trial condition (i.e., win, loss, and neutral) and group stratification, consisting of family risk (i.e., low and high family risk), lifetime diagnosis of MDD (i.e., with [+] or without [-] MDD diagnosis), or lifetime diagnosis of ANX (i.e., with [+] or without [-] ANX diagnosis). Separate analyses were run for each ERP and CSD PCA factor and each stratification. Sex was included as a covariate but not as a fixed effect because previous analyses on depressive symptoms revealed no group differences between sex and all

subgroups were approximately balanced with regard to sex. Given the small cell sizes, all group stratifications were used in separate models.

The multilevel modeling analysis was conducted in R with the *nlme* package (Pinheiro et al., 2022) using the following R syntax: *lme(PC ~ Group * Condition + age + sex, random = ~1|id)*, where *PC* (principal component) is the dependent variable, *Group* (low/high risk, MDD+/-, or ANX+/-) and *Condition* (win/loss/neutral) are fixed effects using categorical predictors with *age* as a continuous covariate and *sex* as a categorical covariate, and *id* represents participants modeled as a random effect. As per *lme* default, restricted maximum likelihood (REML) was used with the between-within method for estimating degrees of freedom (*df*). All ANOVA *F* statistics reflected marginal estimates (Type III) using R syntax: *anova(model, type = "marginal")*, where *model* corresponds to the linear mixed-effects model fit by REML via the R function *lme*. Effect sizes are reported as semi-partial R^2 (R^2_β; Edwards et al., 2008). The R package *emmeans* (Lenth et al., 2022) was used to estimate marginal means, corresponding standard errors, and pairwise comparisons.

To empirically evaluate if any differences in effects between the parallel analyses of ERP and CSD component estimates are genuine, we also conducted several joint analyses that included both estimates for a given component (FN or fb-P3). For this purpose, the pooled factor scores stemming from each transformation were first z-transformed and then jointly analyzed in analogous MLMs with transformation (ERP vs CSD) as a categorical fixed effect. The sole objective for these supplemental analyses was to detect significant interactions involving transformation.

To determine if the severity of current depressive and anxiety symptoms covaried with ERP and CSD estimates, we repeated the original MLM analyses with the scores of PHQ-9 and IDAS-II (subscales for general depression and social anxiety) as added covariates. For the one individual with missing PHQ-9 and IDAS-II data, the sample's grand mean was used as a substitution to avoid the exclusion of this participant.[1]

[1] Because all scores for PHQ-9, IDAS-II GD and IDAS-II SA were entered as simultaneous covariates, the inclusion of PHQ-9 and IDAS-II GD in the same model may partial out their common variance and impact the ability to identify a relationship between the study subgroups and the electrophysiological measures. Although all symptoms scales were positively correlated (PHQ9 with IDAS-II GD, r = .806; PHQ9 with IDAS-II SA, r = .488; IDAS-II GD with IDAS-II SA, r = .730), their pairwise common variance was 65% or less. Furthermore, additional MLM analyses that included only the two IDAS-II scales, or only one of the three symptom scores at a time, were fully consistent with the findings reported below using all three symptom scores as simultaneous covariates.

For the behavioral data, only the monetary conditions (i.e., win vs. loss trials) were compared because participants did not necessarily respond to neutral trials without monetary incentives despite instruction. For accuracy, the total number of win and loss trials were compared as the success rate of wins (in percentage) to validate the adjustment of task difficulty based on performance. Because reaction times were not normally distributed and were not directly comparable across participants given the changing target criterion, the percentage of reaction time regarding target latency was calculated. Both behavioral measures were analyzed via MLM as described above: Group (high risk vs low risk, with vs without MDD or ANX history) and, for reaction times only, Condition (win, loss) were fixed effects using categorical predictors with age and sex as covariates, and participants were modeled as a random effect.

2.7 Power analysis

To approximate a priori effect size estimates, we used repeated measures ANOVA as the closest available model implemented in G*Power software (version 3.1.9; Faul et al., 2007). For within-subjects main effects and within-between interactions having a small-to-medium effect size (f = 0.15; Cohen, 1988) and setting α error probability = 0.05, power = 0.8, two groups (high vs low risk, with vs without MDD or ANX history, respectively) and three repeated measurements (win, loss, neutral), correlation among repeated measures = 0.7, nonsphericity correction ε = 0.7, the recommended sample size is 58 with a power of 0.81. For between-subject main effects with a medium-to-large effect size (f = 0.33), the recommended sample size is 60 with a power of 0.80. Therefore, our actual sample size of 61 provided adequate power for the targeted between-within effects.

3. Results

3.1 Behavioral data

Across all monetary trials, the mean success rate (i.e., number of valid win trials divided by the total of valid win and loss trials) was 51.7% ±4.4%, thereby validating the adjustment of target latency based on prior performance. On average, participants won $5.10 ±1.10 after task completion. There was no significant difference in success rate for high vs. low risk (*Mean* ±SEM, 51.8% ±0.86 vs 51.5% ±0.82), with vs. without MDD history (51.6% ±0.82 vs 51.7% ±0.86), and with vs. without anxiety disorder history (51.6% ±0.75 vs 51.8% ±0.96; all $F_{[1,57]} \leq$

1.0, all *n.s.*).

On average (*Mean* ±SD), there were 23.7 ±2.4 and 22.3 ±3.1 valid trials in each monetary condition (win, loss) that entered into further statistical analyses. The number of valid win and loss trials did not differ significantly between stratification groups (all $F_{[1,59]} \leq 0.05$, *n.s.*). Participants responded at 89.5% ±3.0% (*Median* = 89.8%) of target latency (215 ±29 ms) in win trials, and at 116.8% ± 8.9% (*Median* = 114.8%) of target latency (204 ±28 ms) in loss trials. Results from the MLM analyses for each group stratification with age and sex as covariates confirmed the within-subjects condition main effect (win < loss; all $F_{[1,59]} \geq 513.3$, all $p \leq 0.0001$); however, there were no other significant effects involving group (family risk, lifetime diagnosis of MDD or anxiety disorder) or a covariate.

3.2 Grand mean ERP and CSD waveforms

Figure 2 shows the grand average ERP waveforms for low and high risk groups, comparing responses to three feedback types (win, loss, neutral). ERPs were characterized by a positivity peaking at about 200 ms (P2), followed by a negative deflection at 280 ms (FN), and a prominent midline positivity around 380 ms (fb-P3; see enlargements for selected midline sites in Fig. 2CD). A visual inspection of the ERP waveforms revealed that when compared to favorable events (win), the FN was greater after unfavorable events (neutral and loss) in both risk groups. Still, this relative negativity in response to loss feedback appeared to be reduced in high- compared with low-risk participants, especially at frontocentral sites (see Fz and FCz in Fig. 2CD). As expected, the fb-P3 was more prominent in monetary compared to neutral trials. Of note, the amplitude differences between monetary and neutral trials were more prominent for high-risk than for low-risk individuals, particularly at mid-parietal sites (see Pz and POz in Fig. 2CD).

<insert Figure 2 about here>

Although CSD waveforms (Fig. 3) were largely consistent with the ERPs, they also revealed distinct early visual components over lateral parietooccipital sites (P9/10, PO7/8, P7/8) that were not as prominent for the ERPs. This, of course, is a logical consequence of using adjacent sites as the EEG reference (i.e., TP9/10; Kayser & Tenke, 2015b). The FN was most prominent (i.e., greatest relative negativity) at frontocentral sites in both risk groups. Compared to ERPs, the CSD-based FN was more distinct with less preceding positivity. Consistent with the ERP component structure, the CSD-based fb-P3 was most prominent at parietal sites (e.g., P5/6,

PO3/4, POz), particularly in monetary than neutral trials.

<insert Figure 3 about here>

3.3 Component waveforms and topographies of PCA solutions

A total of 52 and 55 principal components were sufficient to completely explain the variance in the ERP and CSD data sets, respectively. In each solution, the first 3 ERP and CSD factors explained over 74% variance. Factors were selected for further analysis if they could unambiguously be related to ERP or CSD components through their temporal characteristics (i.e., the peak latencies of factor loadings) and their spatial configurations (i.e., the factor score topographies for each experimental manipulation). Figure 4 compares the time courses of the factor loadings for first 8 ERP (93.0% total variance) and CSD (91.7%) factors. It is apparent that both PCA solutions yielded analogous factors with similar levels of explained variance. The ERP factors are labeled with the polarity (N/P) of the associated ERP component and the loadings' peak latency (in ms), whereas the CSD factors are labeled with peak latency only because they tend to reflect a dipolar topography consisting of sinks *and* sources. Of all the factors, the temporal and topographical characteristics of *N309* and *309*, and *P379* and *383*, closely corresponded to known FN and fb-P3 features, respectively. N309 and 309 peaked in close temporal proximity to a post-feedback time window (200 to 300 ms) during which FN was maximal in amplitude, and its frontocentral negativity also included the sites most frequently chosen for analyzing FN (Ait Oumeziane & Foti, 2016; Cavanagh et al., 2022; Klawohn, Brush, & Hajcak, 2021a). Likewise, P379 and 383 peaked in the time window of 350 to 450 ms after feedback onset, consistent with the characteristics of fb-P3 (Donaldson et al., 2016; Novak & Foti, 2015). Both the broad centroparietal positivity in P379 and the parietooccipital positivity in 383 corresponded to the established characteristics of fb-P3 (Ait Oumeziane & Foti, 2016; White et al., 2021). Consequently, these two sets of factors, which are highlighted in the same colors in Figure 4, were considered for further statistical analyses.

<insert Figure 4 about here>

3.4 Nonparametric and multilevel modeling statistical analyses

Figure 5 shows the FN and fb-P3 factor score topographies stemming from ERP and CSD solutions and the corresponding randomization tests evaluating topographic differences between conditions. Because topographies were highly comparable for all group

stratifications, for the sake of simplicity, only those for low and high risk individuals are included in Figure 5.

<insert Figure 5 about here>

3.4.1 Findings for FN: Factors N309 and 309

It is evident that both ERP (N309: 1.5% explained variance; Fig. 5A) and CSD (309: 1.1%; Fig. 5B) factors had a frontocentral negativity in loss but not win trials. For the resulting FN (i.e., loss-minus-win), a significant midfrontal negativity was observed across all groups and both data transformations. Given the larger sample size, these effects were strongest for the full sample (Fig. 5AB, row 1); nonetheless, the effects were clearly present for low and high risk (Fig. 5AB, rows 2-3) and the other two subgroups. The corresponding more prominent change in amplitude for loss versus win trials can be seen as the negative deflection at around 290 ms in the ERP (Fig. 2) and CSD waveforms (Fig. 3). CSD-based FN effects were more focal compared to ERP-based FN effects, and they appeared to also include a relative larger source for loss than win over right occipital sites in addition to the midfrontal sink; however, this was not consistently observed across subgroups (a comparable N309 effect was not significant).

The midfrontal sites at which the win-minus-loss difference reached a $p = 0.05$ significance threshold for the entire sample ($N = 61$) were selected for multilevel modeling analyses, resulting in 19 frontocentral sites (AF3/4, F1/2, F3/4, F5/6, FC3/4, FC1/2, FPz, FP2, AF8, AFz, Fz, F8, FCz) for ERP factor N309 and 6 frontocentral sites (F1/2, FC1/2, Fz, FCz) for CSD factor 309 (marked as filled circles in Fig. 5AB, column 4).

Table 2 summarizes the statistical effects for each multilevel model. A highly significant condition main effect was seen for both ERP and CSD estimates and each stratification group. These condition effects stemmed from greater FN amplitudes to loss compared with win and neutral (pairwise contrasts, all $t_{[118]} \geq 4.16$, all $p \leq 0.0002$), whereas win and neutral did not differ from each other (all $t_{[118]} \leq 0.96$, all $p \geq 0.60$; Figure 6AB). Across all group stratifications, marginal means and standard error estimates for condition were highly comparable (for family risk: ERP, win = 0.13 ±0.14, loss = −0.56 ±0.14, neutral 0.02 ±0.14; CSD, win = −0.02 ±0.12, loss = −0.94 ±0.12, neutral −0.12 ±0.12). Of note, effect sizes for condition were small to medium for ERP FN estimates ($0.111 \leq R^2_\beta \leq 0.152$) but medium for CSD estimates ($0.149 \leq R^2_\beta \leq 0.197$; Tab. 2). There were no significant main or interaction effects involving group, nor were there any significant covariates.

<insert Table 2 and Figure 6 about here>

These effects were fully preserved in the joint analyses of ERP and CSD z-transformed FN estimates, and there were no significant effects involving transformation (all $F_{[1,295] \text{ or } [2,295]} \leq 1.77$, all $p \geq 0.18$).

The supplemental MLM analyses with added covariates of symptom severity closely replicated these effects. While there were no significant covariate effects for the current severity of depression (PHQ-9, IDAS-II) across all MLM analyses or for the current severity of social anxiety (IDAS-II) for ERP FN estimates (all $F_{[1,54]} \leq 0.80$, all $p \geq 0.37$), the MLM analyses for CSD FN estimates revealed small covariate effects of social anxiety for each stratification (all $F_{[1,54]} \geq 5.03$, all $p \leq 0.03$; $0.085 \leq R^2_{\beta} \leq 0.087$). Greater severity of social anxiety was linked to greater CSD FN (all beta estimates, $-0.0696 \leq \beta \leq -0.0704$).

3.4.2 Findings for fb-P3: Factors P379 and 383

ERP factor P379 (45.0% explained variance) showed a broad positivity at centroparietal sites (Fig. 5C), with greater amplitude in monetary (win/loss) than neutral trials, corresponding to the prominent peak around 380 ms at numerous centroparietal sites seen in the ERP waveforms (Fig. 2). In contrast, CSD factor 383 (26.4%) showed a prominent posterior source and a distinct mid-anterior source, with both sources accompanied by inferior lateral sinks (Fig. 5D). The posterior source was evident in the CSD waveforms with the long-lasting positivity peaking around 380 ms at posterior sites (e.g., P5/6 and POz in Fig. 3).

As can be seen from the factor score topographies (Fig. 5CD), the broad centroparietal (P379) or occipitoparietal (383) positivity was seen for low and high risk individuals, and this was also the case for the other two group stratifications. Notably, the parietal positivity of P379 encompassed a broad uninterrupted region spanning frontal and occipital sites, whereas the positivity associated with CSD factor 383 was separated into a mid-occipitoparietal and an off-midline frontocentral region, suggesting multiple generator sources that peaked around 383 ms after feedback. For the CSD-based fb-P3 (383), 20 prominent posterior sites (CP3/4, CP1/2, P1/2, P3/4, P5/6, PO7/8, PO3/4, O1/2, Oz, POz, Pz, CPz) that reached a significance level of $p = 0.05$ were selected for multilevel modeling analyses (selected sites are marked as filled circles in Fig. 5D, column 4). For the ERP-based fb-P3 (P379), 50 sites (AF3/4, F1/2, F3/4, F5/6, FC5/6, FC3/4, FC1/2, C1/2, C3/4, C5/6, TP7/8, CP5/6, CP3/4, CP1/2, P1/2, P3/4, P5/6, P7/8, PO7/8, PO3/4, O1/2, Oz, POz, Pz, CPz, Afz, Fz, FCz, Cz) were selected for further

analysis (Fig. 5C, column 4).

The MLM analyses of fb-P3 revealed a robust condition main effect across ERP and CSD estimates and stratification groups (all $F_{[2,118]} \geq 26.4$, all $p < 0.0001$; $0.301 \leq R^2_\beta \leq 0.374$; Tab. 2), with monetary trials (win and loss) being more positive than neutral (pairwise contrasts, all $t_{[118]} \geq 9.71$, all $p \leq 0.0001$) and no difference between win and loss (all $t_{[118]} \leq 2.03$, all $p \geq 0.11$; Figure 6CD). Across all group stratifications, marginal means and standard error estimates for condition were highly comparable (for family risk: ERP, win = 0.62 ±0.08, loss = 0.62 ±0.08, neutral −0.26 ±0.08; CSD, win = 0.64 ±0.06, loss = 0.55 ±0.06, neutral 0.04 ±0.06).

There was a small main effect of risk for ERP estimates (Tab. 2) stemming from greater fb-P3 for high versus low risk individuals (Mean ±SEM, 0.48 ±0.11 vs. 0.17 ±0.10) that was not observed for CSD fb-P3 estimates (0.44 ±0.08 vs. 0.38 ±0.08). The ERP risk main effect was qualified by a risk × condition interaction (Tab. 2) stemming from significant group differences for monetary (both $t_{[57]} \geq 2.50$, both $p \leq 0.015$) but not neutral trials ($t_{[57]} = 0.04$, *n.s.*; left subpanel in Fig. 6C). There was also a risk × condition interaction for CSD fb-P3 (Tab. 2), however, there were no significant contrasts of risk for any condition (all $t_{[57]} \leq 1.39$, all $p \geq 0.17$; left subpanel in Fig. 6D). Instead, while win and loss trials were greater than neutral trials for both risk groups (all $t_{[118]} \geq 5.64$, all $p \leq 0.0001$), individuals at low risk also had a marginally greater CSD fb-P3 for win than loss ($t_{[57]} = 2.13$, $p = 0.09$) whereas those at high risk did not ($t_{[57]} = 0.50$, *n.s.*).

By contrast, there was a small main effect of MDD history for CSD estimates (Tab. 2) stemming from reduced fb-P3 for individuals with a lifetime history of MDD compared to those without (0.28 ±0.07 vs. 0.57 ±0.08; middle subpanel in Fig. 6D) that was not observed for ERP fb-P3 estimates (0.29 ±0.11 vs. 0.35 ±0.11; middle subpanel in Fig. 6C). There were no other significant effects for fb-P3, including covariates.

Importantly, the joint analyses of ERP and CSD z-transformed fb-P3 estimates revealed significant interactions of risk × transformation ($F_{[1,295]} = 6.02$, $p = 0.01$, $R^2_\beta = 0.020$) and MDD history × transformation ($F_{[1,295]} = 5.70$, $p = 0.02$, $R^2_\beta = 0.019$). These effects stemmed from significant differences between low and high risk for ERP ($t_{[57]} = 2.24$, $p = 0.03$) but not CSD fb-P3 ($t_{[57]} = 0.40$, *n.s.*), and between individuals with and without MDD history for CSD ($t_{[57]} = 2.69$, $p = 0.009$) but not ERP fb-P3 ($t_{[57]} = 0.35$, *n.s.*). There were no other significant interaction effects involving transformation (all $F_{[1,295] \text{ or } [2,295]} \leq 2.13$, all $p \geq 0.15$). Across

transformation, a marginally significant risk × condition interaction ($F_{[1,295]}$ = 2.39, p = 0.09, R^2_β = 0.020) confirmed what had already been separately observed for each transformation (Tab. 2), with marginal means (±SEM) revealing greater fb-P3 for high than low risk for loss (0.54 ±0.14 vs. 0.10 ±0.13; $t_{[57]}$ = 2.22, p = 0.03) and win (0.59 ±0.14 vs. 0.22 ±0.13; $t_{[57]}$ = 1.89, p = 0.06) but not neutral trials (−0.74 ±0.14 vs. −0.67 ±0.13; $t_{[57]}$ = 0.37, *n.s.*).

The supplemental MLM analyses with added symptom severity covariates closely replicated these effects. There were no significant covariate effects (all $F_{[1,54]}$ < 1.0, all *n.s.*).

3.5 Component reliability

To obtain internal consistency estimates for the two identified components of interests, each component was pooled across their target sites, as indicated above (see also Fig. 5, column 4 in each panel). Using the MID conditions as items, Cronbach's alpha was .76 (N309, ERP) and .61 (309, CSD) for FN, and .81 (P379, ERP) and .87 (383, CSD) for fb-P3. These values for PCA-based components correspond to moderate (.5 < α ≤ .7) and high (.7 < α ≤ .9) internal consistency (see Thigpen et al., 2017).

Construct validity estimates for these two components are given in Table 3. Pearson correlations (183 value pairs stemming from N = 61 participants and 3 conditions) were computed between all four components measures (FN and fb-P3 for ERP and CSD PCA solutions) pooled across their target sites. Correlations between corresponding component estimates (i.e., ERP FN with CSD FN, ERP fb-P3 with CSD fb-P3) were reasonably high (r > .64, bold in Tab. 3), suggesting good convergent validity, whereas all other correlations were reasonably low (|r| < .22), suggesting good discriminant validity. Of course, it should be noted that correlations between Varimax-rotated components (i.e., within any given PCA solution) are by definition zero if all component scores are included due to their orthogonality constraint; this PCA characteristic was effectively maintained despite using pooled component estimates from different site subsets (|r| ≤ .05, italics in Tab. 3).

<insert Table 3 about here>

4. Discussion

4.1 ERP- and CSD-based reward processing components

In agreement with several prior ERP studies employing the MID task (Ait Oumeziane et al., 2019; Landes et al., 2018; Novak et al., 2016; Novak & Foti, 2015), we identified distinct tPCA-based components that were unambiguously related to feedback-related FN (factor N309) and P3 (factor P379). FN peaked around 300 ms after feedback onset with a mid-frontocentral maximum, whereas the ensuing fb-P3 peaked around 380 ms having a broad centroparietal maximum. Importantly, these ERP components revealed the commonly-reported condition effects, that is, a relatively increased FN (i.e., more negative) to loss compared with win trials (Chang et al., 2020; Foti et al., 2014; Foti & Hajcak, 2009; Novak et al., 2016), and a greater fb-P3 (i.e., more positive) for monetary (win or loss) compared to neutral trials (Ait Oumeziane et al., 2019; Novak et al., 2016; Song et al., 2020).

Notably, the reward feedback ERP component structure was closely matched by two distinct tPCA-based CSD components related to FN (309) and fb-P3 (383), both in terms of peak latency and topography; as expected, however, the CSD topographies were more focal than their ERP counterparts. The CSD-based FN with its mid-frontal maximum was highly comparable to a response-locked mid-frontal negativity (Kayser et al., 2007) and therefore appears to be part of a family of mid-frontal negativities associated with performance monitoring, which include the error-related negativity (ERN; e.g., Gehring & Knight, 2000), correct-response negativity (CRN; e.g., Vidal et al., 2000), and also the feedback-related negativity (e.g., Krigolson, 2018; Proudfit, 2015). In contrast to its ERP counterpart, the CSD-based fb-P3 had a mid-parietooccipital maximum that was separated from a mid-frontal positivity, similar to CSD-based P3 topographies to targets during an oddball task (e.g., Tenke et al., 2010).

In addition, the condition-related effects of FN and fb-P3 were not only highly robust across all statistical analysis models, but the associated findings were also highly consistent across ERP- and CSD-based measures. Collectively, our findings confirmed that FN is sensitive to valence: it was greater after unfavorable than favorable outcome (e.g., Chang et al., 2020; Foti et al., 2014; Foti & Hajcak, 2009; Novak et al., 2016); on the other hand, fb-P3 is sensitive to motivational salience: it was greater to monetary (win or loss) than neutral outcome (e.g., Foti & Hajcak, 2009; Hajcak et al., 2007; Novak & Foti, 2015; Tunison et al., 2019). Both ERP- and CSD-based fb-P3 were greater (i.e., more positive) after win and loss than neutral feedback, suggesting greater motivated attention on monetary than neutral trials.

Importantly, PCA-based ERP and CSD component estimates showed at least moderate internal

consistency.[2] Moreover, good convergent and good discriminant construct validity was observed for ERP and CSD estimates, both for FN and fb-P3 components, which further bolsters the adopted analytic approach for our clinical objective (e.g., Larson & Moser, 2017).

4.2 Outcome valence: Feedback negativity related to risk for and lifetime history of depression

After stratifying the current sample of 61 individuals at family risk for depression (Weissman et al., 2016a, 2016b) into groups of high and low risk and those with and without a lifetime history of MDD or anxiety disorder, condition-related FN differences between loss and win trials (i.e., more negative for loss) were not altered as a function of stratification group. Contrary to our hypothesis, risk for or history of MDD was not associated with reduced FN, which is in agreement with some prior reports (e.g., Foti et al., 2015; Nelson et al., 2016; Novak et al., 2016) but in conflict with a considerable number of other studies showing reduced FN in depression (e.g., Brush et al., 2018; Chang et al., 2020; Foti et al., 2014; Glazer et al., 2018; Liu et al., 2014; Thoma et al., 2015). There was no indication that individuals at high risk for or with a history of depression differed from their respective control groups in their responsivity to negative or positive feedback (i.e., loss of pleasure; e.g., Pizzagalli, 2022; Weinberg, 2022). There was also no evidence that current symptoms of depression, as measured by PHQ-9 and IDAS-II, co-varied with FN; however, greater severity of social anxiety was linked to greater CSD-based FN amplitude which is in agreement with the findings of Nelson & Jarcho (2021) who notably also applied a CSD-PCA approach to their data analysis.

It has been argued that the current MID task is problematic with regard to feedback processing because participants are, or could be, aware if their responses are fast enough or not (National Advisory Mental Health Council Workgroup on Tasks and Measures for Research Domain Criteria, 2016), manifesting in altered stimulus-preceding negativity (SPN) during feedback anticipation (Clayson et al., 2019). In these instances, feedback may be predicted. By contrast, feedback cannot be predicted during a guessing task, and perhaps the unpredictability feature is what causes a reduced FN in depression. While indeed several of the studies that reported reduced FN in depression or depressing risk used a guessing task (e.g., Brush et al., 2018; Foti et al., 2014; Liu et al., 2014), not all did (Glazer et al., 2018; Thoma et al., 2015). Moreover, two recent studies in young adolescents add to this mixed evidence: one found reduced FN during the MID task to be associated with increased depressive symptoms (Thompson et al., 2023),

[2] Given the comparatively low number of task trials, internal consistency may likely be improved by increasing the total number of MID trials and thus the ERP signal-to-noise ratio.

whereas the other used the doors task and found no FN differences between individuals with and without a history of preschool-onset MDD task (Santopetro et al., 2023).

For the present data, it is important to note that there was no evidence that the behavioral performance differed between the stratification groups. Furthermore, given that the average response latency was about 192 ms for wins and 238 ms for losses, it seems unlikely that performance awareness and feedback predictability would be a major contributing factor. In addition, we excluded those trials that would predict feedback, that is, those having a decidedly premature response time or no response at all. In any case, lacking a guessing task as a control condition, our study was not designed to answer this important question. Although a recent study employed both MID and doors tasks, the two tasks were used to target different ERP components, and therefore, FN and fb-P3 were not directly compared between tasks (Tsypes, Owens, & Gibb, 2021). However, an empirical comparison of different reward processing paradigms is required for a better understanding of what different aspects of reward processing, if any, are targeted with these tasks, and whether any differences are critical to demonstrate reward processing impairments in depression.

4.3 Reward salience: Feedback P3 related to risk for and lifetime history of depression

Unlike FN amplitude, condition effects of fb-P3 differed between individuals at low and high family risk for depression. Across data transformation, and contrary to our hypothesis, individuals at high compared with those at low risk showed a greater fb-P3 for monetary trials (win or loss) but fb-P3 did not differ between risk groups in response to neutral trials. For ERP-based fb-P3 only, these salience-related group effects were sufficient to sustain an overall risk main effect. Condition effects did not differ between individuals with and without a history of MDD or anxiety disorder, which is in agreement with some prior studies (e.g., Bowyer et al., 2022; Santopetro et al., 2021; Thoma et al., 2015; Thompson et al., 2023) but contrary to others (e.g., Ait Oumeziane et al., 2019; Chang et al., 2020; White et al., 2021; Zhang et al., 2020). Accordingly, the present findings do not support hypothesized impairments in processing reward salience in individuals at high risk or without a lifetime history of MDD.

Still, CSD- but not ERP-based estimates revealed overall reduced fb-P3 in individuals with compared to those without a lifetime history of MDD, which is in agreement with prior findings of reduced P3 in depression across a variety of paradigms (e.g., Bruder et al., 2012). For this multigenerational sample of individuals at high family risk for depression, we have previously reported P3 source reductions in response to aversive pictures, particularly for those having

experienced prior depressive episodes (Kayser et al., 2017), suggesting a blunted responsivity to motivational salience. However, we note the considerable difference between passively viewing salient stimuli and receiving monetary reward after effortful responses which likely elicit a fundamentally different affective response. It should also be recognized that the current study employed monetary but not social reward (Ait Oumeziane et al., 2019; Zhang et al., 2020), and only the latter was found to predict increased symptoms of depression (see Weinberg, 2022).

These considerations cycle back to the above discussion regarding the undetermined significance of different reward processing paradigms, a question of critical importance that needs to be addressed by future research.

4.4 Findings affected by data transformation

Contrary to the robust condition effects, more subtle fb-P3 effects having smaller effect sizes involving group differed significantly between ERP- and CSD-based measures. Although a main effect of MDD history was observed for CSD-based but not ERP-based fb-P3, a risk main effect was found for ERP- but not CSD-based fb-P3. At the very least, these subtle effects should be interpreted with great caution because they may be especially prone to false positives. This is a particular concern for ERP (i.e., surface potential) measures that are dependent on an arbitrary reference choice, in this case, linked mastoids. The even bigger concern is that different data transformations will lead to different conclusions.

One may argue that without knowing the true effect, as is the case here, it is impossible to determine what analysis is correct. However, we have demonstrated that for established (i.e., known or simulated) effects CSD measures provide a more accurate, valid and reliable characterization compared to surface potentials (e.g., Kayser & Tenke, 2015a; Tenke & Kayser, 2015). It can therefore be argued that CSD measures are more trustworthy than ERP measures. Even more importantly, ERP measures are arbitrary in the sense that each reference choice (e.g., linked mastoids, vertex, nose, common average, etc.) will likely yield different results; by contrast, reference-free CSD measures are unique (all reference choices yield the same CSD transformation), which provides a strong theoretical and parsimonious rationale for relying on CSD than on ERP findings if they diverge.

4.5 Strengths and limitations

This report presents a systematic, data-driven analysis of *reference-free* ERP measures to

study feedback-related reward processing. To this end, we directly compared findings using a linked-mastoids EEG reference with those stemming from CSD-transformed ERP data. While both sets of results were largely consistent with those of prior reports, theoretical (e.g., no ambiguity due to reference choice) and practical advantages (no smearing due to volume conduction yielding a sharper time course and more focal topographies, larger effect sizes) advocate the use of reference-free CSD over reference-dependent ERP measures (e.g., Kayser & Tenke, 2015a, 2015b). Moreover, the use of CSD has the promise to unify findings obtained with disparate EEG reference schemes (Kayser & Tenke, 2015b).

Another strength of the current study is an explicit task manipulation check and evaluation of possible differences in behavioral performance between stratification groups. The individual adjustment of response time targets did indeed yield a balance between win and loss trials, and there were no differences in performance between subgroups.

In our sample, findings tended to be similar between stratifications based on risk and history of MDD or anxiety disorder. This observation is possibly, or at least in part, due to the inclusion of a large proportion of individuals assigned to the same stratification group (e.g., low risk with no MDD history, n = 22; high risk with MDD, n = 22). As a direct consequence, most of the group differences were a result of re-assigning a rather small number of people for the other stratification (e.g., low risk with MDD, n = 10 vs high risk without MDD, n = 7). Thus, this sample characteristic limits generalizability of the findings and conclusions that can be drawn.

While the large three-generation longitudinal, cohort study (Weissman et al., 2016a, 2016b) including participants at high and low family risk for depression provides an excellent opportunity for examining this population across time (e.g., has a complete history of MDD and anxiety disorder episodes), it is not diverse in terms of ethnicity and background. The majority of the sample is Caucasian from a New Haven neighborhood with similar socioeconomic status, which further reduces the generalizability of the findings. At the same time, however, the sample's homogeneity in this regard is also a strength because it will likely reduce the influence of uncontrolled variables and thereby increase internal validity.

On the other hand, the sample is heterogeneous with regard to age. Using the doors guessing task, Klawohn et al. (2021b) reported that FN only predicted impaired mood reactivity in younger (≤ 26 years of age) but not older adults. Then again, Moser et al. (2018) who also used the doors task found that FN increased with age from preschool to adolescence and adults. For the current MID task, age was included as a covariate in our multilevel models but did not reveal significant

age effects. More research will be required to clarify the meaning of task-dependent and/or developmental reward sensitivities of FN.

Furthermore, parental depression was shown to predict reduced FN in children (Kujawa et al., 2019), but the rather small sample size in our study did not allow for taking the effect of lifetime MDD history in the second generation into consideration for the symptomology in the third generation. Collectively, we can therefore not draw conclusions on whether age or generation had an effect on the relationship between depression and FN/fb-P3.

Finally, we did not account for the generational difference of risk and onset of MDD due to the small sample size. People who had MDD before the age of 18 were not separated in our analyses from those who developed depression later in life (Freeman et al., 2020; Galván, 2013; Smith et al., 2012). Also, individuals with a depressed grandparent were not compared with people with a depressed parent (Warner et al., 1999), across generations, or both parents affected, which has been shown to affect EEG measures (i.e., alpha power during rest; Bruder et al., 2005).

Although we note that there were more individuals at high risk having a lifetime diagnosis of MDD, this did not enter into our analyses due to the limited sample size. As for depression severity, we found symptom score differences between subgroups of risk and MDD history in the expected direction, but depressive symptom scores did not covary with FN or fb-P3 measures. The inclusion of onset of depression, as well as other putative moderator and mediator variables (e.g., Positive Affect; Weinberg et al., 2015), should be considered in future studies with an adequate sample size.

5. Conclusions

The predicted overall reduced FN (difference between loss and win outcomes) and fb-P3 (difference between monetary and neutral outcomes) in individuals at high family risk for depression and those with a lifetime history of MDD were not supported by our findings. As predicted, subtle group differences were subject to data transformation, that is, the EEG reference choice. Given the robustness of CSD-based reward processing components in the context of the present MID paradigm, the use of reference-free techniques is strongly recommended as a sound methodological choice.

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Author Contributions

Yifan Gao: Data curation; Formal analysis; Investigation; Methodology; Validation; Visualization; Writing – original draft; Writing – reviewing & editing. **Lidia Y.X. Panier**: Investigation; Data curation; Writing – reviewing & editing. **Marc J. Gameroff**: Data curation; Writing – reviewing & editing. **Randy Auerbach**: Conceptualization; Writing – reviewing & editing. **Jonathan Posner**: Writing – reviewing & editing. **Myrna Weissman**: Funding acquisition; Project administration; Writing – reviewing & editing. **Jürgen Kayser**: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Resources; Software; Supervision; Validation; Visualization; Writing – original draft; Writing – reviewing & editing.

Data Availability Statement

Data were obtained as part of an ongoing, multi-generational study of families at risk for depression that started in 1982 (before data sharing existed); therefore, data sharing consent was not obtained. According to the Institutional Review Board at Columbia University and New York State Psychiatric Institute, public data sharing, even anonymously, is restricted by participants' informed consent. For further information on data access requests, interested readers may contact IRBMail@nyspi.columbia.edu.

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Table 1. Crosstabulation of demographic information for all group stratifications.

	Risk				MDD				ANX			
	Low (n = 32)	High (n = 29)	$\chi^2_{[1]}$ or $t_{[59]}$	p	Without (n = 29)	With (n = 32)	$\chi^2_{[1]}$ or $t_{[59]}$	p	Without (n = 23)	With (n = 38)	$\chi^2_{[1]}$ or $t_{[59]}$	p
Sex [m/f] female [%]	15/17 53.1%	15/14 48.3%	0.01	0.91	15/14 48.3%	15/17 53.1%	0.14	0.71	14/9 39.1%	16/22 57.9%	2.02	0.16
Age	27.7 ±12.2	33.7 ±14.0	−1.81	0.08	27.1 ±12.6	33.6 ±13.5	-1.94	0.06	31.6 ±14.6	29.9 ±12.7	0.48	0.63
Education [years]	13.9 ±2.4	15.0 ±2.2	−1.87	0.07	14.5 ±2.1	14.4 ±2.7	0.23	0.82	14.4 ±2.4	14.4 ±2.4	-0.02	0.98
EHI	53.2 ±56.4	76.6 ±35.0	−1.93	0.06	59.9 ±55.8	68.4 ±41.3	-0.68	0.50	61.9 ±51.5	65.8 ±47.3	-0.30	0.76
PHQ-9 [a]	3.94 ±4.87	5.90 ±4.48	−1.62 [b]	0.11	**3.41 ±3.70**	**6.26 ±5.25**	**−2.41** [b]	**0.02**	**3.26 ±2.30**	**5.89 ±5.57**	**−2.15** [b]	**0.04**
IDAS-II GD [a]	**37.3 ±11.6**	**44.2 ±13.3**	**−2.17** [b]	**0.03**	**36.3 ±10.3**	**44.7 ±13.8**	**−2.65** [b]	**0.01**	37.1 ±10.3	42.8 ±13.9	−1.71 [b]	0.09
IDAS-II SA [a]	8.68 ±3.77	10.28 ±4.91	−1.42 [b]	0.16	**8.28 ±2.70**	**10.55 ±5.35**	**−2.05** [b]	**0.04**	8.78 ±2.83	9.86 ±5.13	−0.93 [b]	0.36

Note. All values reflect *Mean* ±*SD* unless indicated otherwise. Risk: low or high family risk for depression; MDD, ANX: with or without lifetime history of major depressive disorder or anxiety disorder. m/f: male/female; EHI: Edinburgh Handedness Inventory laterality quotient (range −100.0 [extremely left-handed] to +100.0 [extremely right-handed]; Oldfield, 1971); PHQ-9: Patient Health Questionnaire-9 total score (Kroenke et al., 2001); IDAS-II GD/SA: Inventory of Depression and Anxiety Symptoms-II (Watson et al., 2012) General Depression and Social Anxiety subscales; [a] n = 60 (missing data for one male participant of low risk, with lifetime history of MDD and anxiety disorder); [b] df = 58. Significant group differences are listed in bold.

Table 2. Summary of ANOVA results (main effects, covariates, interactions) in three multilevel models.

		FN						fb-P3					
		ERP			CSD			ERP			CSD		
	Variable	*F*	*p*	R^2_β	*F*	*p*	R^2_β	*F*	*p*	R^2_β	*F*	*p*	R^2_β
Risk	Group							**7.93**	**0.007**	**0.12**			
	Cond	**10.56**	**0.0001**	**0.15**	**14.47**	**<0.0001**	**0.20**	**26.36**	**<0.0001**	**0.31**	**32.27**	**<0.0001**	**0.35**
	Age										1.10	0.30	0.02
	Sex	1.82	0.18	0.03									
	Group × Cond							**6.03**	**0.003**	**0.09**	**3.63**	**0.03**	**0.06**
MDD	Group										**5.87**	**0.02**	**0.09**
	Condition	**7.79**	**0.0007**	**0.12**	**10.35**	**0.0001**	**0.15**	**30.29**	**<0.0001**	**0.34**	**35.27**	**<0.0001**	**0.37**
	Age												
	Sex	2.00	0.16	0.03									
	Group × Cond							1.23	0.29	0.02	1.31	0.27	0.02
ANX	Group										2.43	0.12	0.04
	Condition	**7.39**	**0.0009**	**0.11**	**13.65**	**<0.0001**	**0.19**	**27.92**	**<0.0001**	**0.32**	**34.11**	**<0.0001**	**0.37**
	Age										1.07	0.30	0.02
	Sex	2.23	0.14	0.04									
	Group × Cond												

Note. FN: feedback negativity; fb-P3: feedback P3; ERP: event-related potential (linked-mastoids reference); CSD: current source density (reference-free); Risk: low or high family risk for depression; MDD, ANX: lifetime history of major depressive disorder or anxiety disorder; Cond: Condition (win, loss, neutral). The *R* syntax for each model was: lme(PC ~ Group * Cond + age + sex, random = ~1|id), where PC reflects the selected principal component (FN: N309 or 309; fb-P3: P379 or 383) as the model's dependent variable, Group reflects the model-specific stratification (Risk, MDD, or ANX), and id reflects a unique participant identifier. For all effects including condition, *df* = 2, 118; for all other effects, *df* = 1, 57. Only *F* values ≥ 1.0 are listed. Significant effects ($p \leq 0.05$) are bolded. Effect sizes are indicated as semi-partial R^2 (R^2_β).

Table 3. Convergent and discriminant validity of component measures.

PC	ERP fb-P3	CSD FN	CSD fb-P3
ERP FN	*0.029*	**0.645**	-0.217
ERP fb-P3		0.013	**0.675**
CSD FN			*-0.054*

Note: Values reflect Pearson correlations *r* between principal component (PC) scores derived from ERP- and CSD-based PCA solutions for components corresponding to feedback negativity (FN) and feedback P3 (fb-P3). Corresponding component measures shown in bold reveal good convergent validity ($r \geq .645$), all other values indicate good discriminant validity ($|r| \leq .217$). Correlations within a given PCA solution are given in italics.

Figures

Figure 1. Trial structure for the monetary incentive delay (MID) task. On each trial, either a monetary or a neutral cue was presented for 500 ms. After an interval of 2000 to 2500 ms, the target was presented for 200 ms, however, this exposure time was continuously adjusted based on the performance on the preceding trial (i.e., increased or decreased by 10 ms). After an interval of 500 ms, a feedback was presented for 2000 ms to indicate a monetary win, loss, or break-even trial outcome.

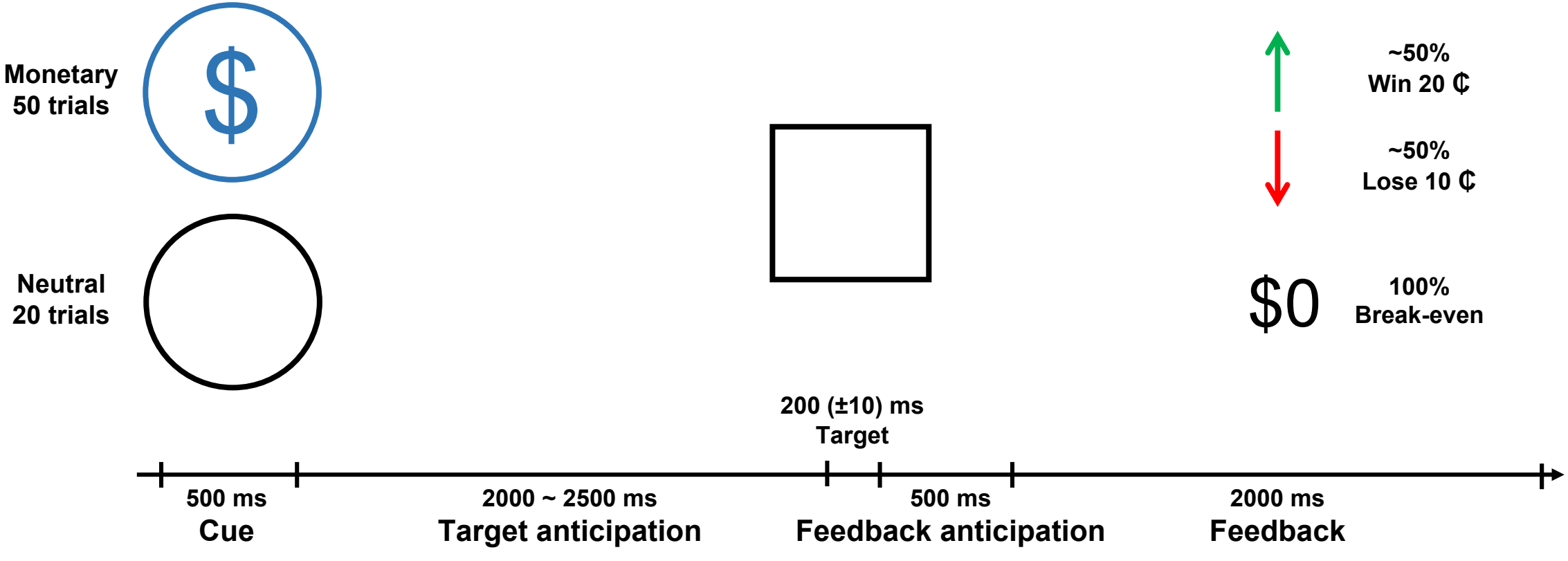

Monetary
50 trials
$
Neutral
20 trials
~50%
Win 20 ¢
~50%
Lose 10 ¢
$0
100%
Break-even
200 (±10) ms
Target
500 ms
Cue
2000 ~ 2500 ms
Target anticipation
500 ms
Feedback anticipation
2000 ms
Feedback

Figure 2. Linked-mastoids referenced grand mean surface potential (ERP [μV]) waveforms (−100 to 800 ms, 100 ms pre-stimulus baseline) comparing win, neutral, and loss stimuli in individuals at low (**A**; *n* = 32) and high risk (**B**; *n* = 29) at all 72 scalp sites. **C**, **D**: Corresponding enlargements (±SEM in shaded color) at selected midline sites, with FN and fb-P3 marked at FCz and Pz, respectively.

A

B

C

D

Win
Loss
Neutral

Low Risk

High Risk

NOSE NZ FPZ FP1 FP2 AF7 AF3 AFZ AF4 AF8 F9 F7 F5 F3 F1 FZ F2 F4 F6 F8 F10 FT9 FT7 FC5 FC3 FC1 FCZ FC2 FC4 FC6 FT8 FT10 T7 C5 C3 C1 CZ C2 C4 C6 T8 TP9 TP7 CP5 CP3 CP1 CPZ CP2 CP4 CP6 TP8 TP10 P7 P5 P3 P1 PZ P2 P4 P6 P8 PO7 PO3 O1 POZ PO4 O2 PO8 PO9 OZ PO10 IZ

-5 0 5 10 15
ERP [µV]
0 300 600
Latency [ms]

FZ
FCZ
PZ
POZ
FN
fb-P3
ERP [µV]
0 300 600
Latency [ms]

Figure 3. Grand mean current source density (CSD [μV/cm^2]) waveforms for low and high risk individuals (A-D as shown in Fig. 2). FN and fb-P3 are marked at FCz and POz, respectively.

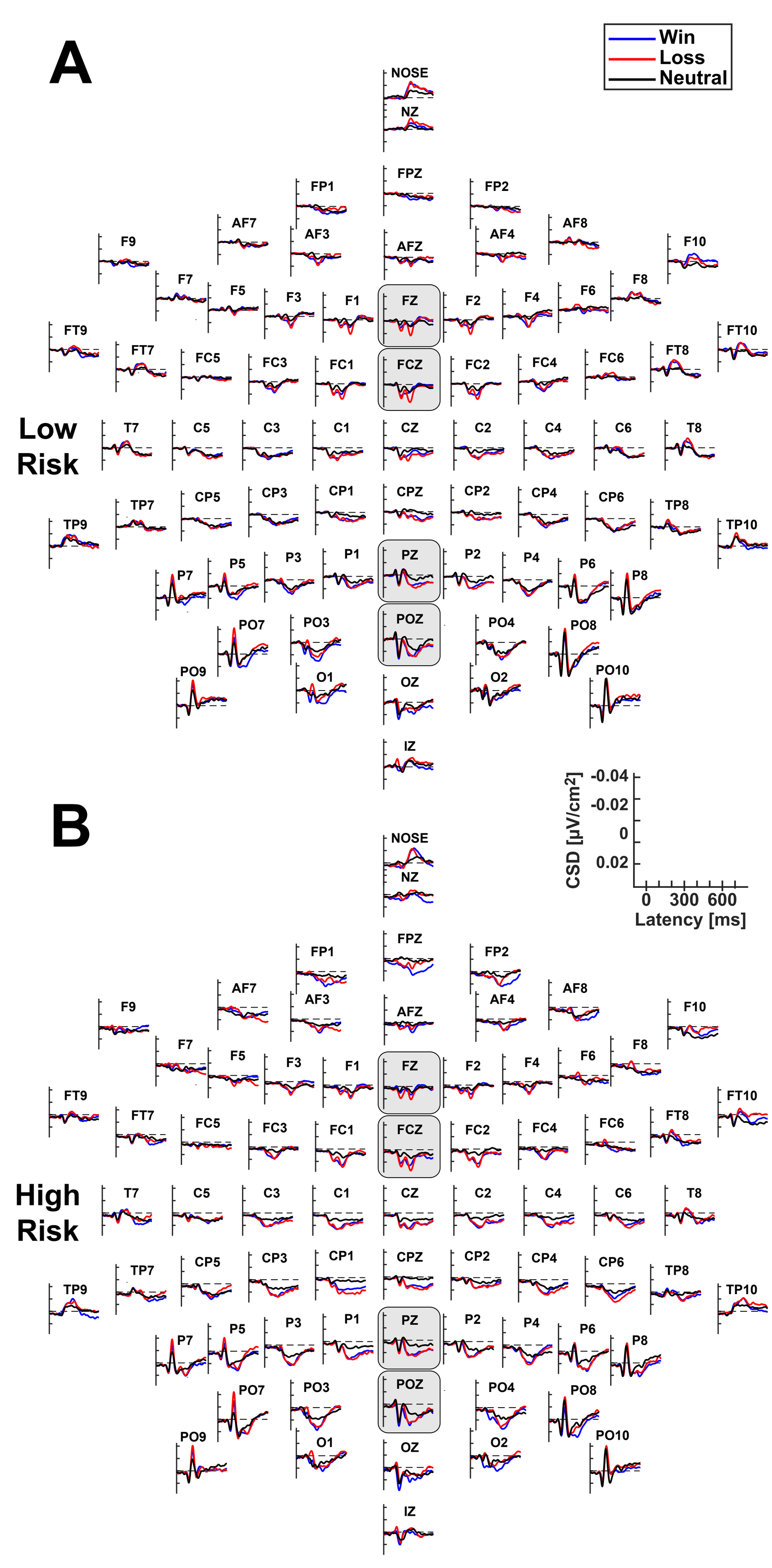

A
Win
Loss
Neutral
NOSE
NZ
FP1
FPZ
FP2
AF7
AF3
AFZ
AF4
AF8
F9
F7
F5
F3
F1
FZ
F2
F4
F6
F8
F10
FT9
FT7
FC5
FC3
FC1
FCZ
FC2
FC4
FC6
FT8
FT10
Low Risk
T7
C5
C3
C1
CZ
C2
C4
C6
T8
TP9
TP7
CP5
CP3
CP1
CPZ
CP2
CP4
CP6
TP8
TP10
P7
P5
P3
P1
PZ
P2
P4
P6
P8
PO7
PO3
POZ
PO4
PO8
PO9
O1
OZ
O2
PO10
IZ
B
CSD [µV/cm²]
-0.04
-0.02
0
0.02
0
300
600
Latency [ms]
High Risk
C
D
FZ
FCZ
PZ
POZ
-0.04
-0.03
-0.02
-0.01
0
0.01
0.02
FN
fb-P3
CSD [µV/cm²]
0
300
600
Latency [ms]

Figure 4. Time courses of factor loadings for first eight temporal PCA (tPCA) loadings extracted from ERP (surface potentials; **A**) and CSD (current source density; **B**) waveforms (*N* = 61). ERP and CSD factor labels indicate the peak latency [ms] of the factor loadings relative to feedback onset. Legends include the percentage of explained variance (all > 1%) after Varimax rotation for each factor. Corresponding grand mean factor score topographies are shown next to the factors of interest. Note that the CSD solution yielded factor loadings with less temporal overlap (i.e., “sharper” time courses) compared with the ERP solution.

A

ERP

μV

2

4

6

121

160

309

266

199

535

719

379

-0.3

0.0

+0.5

-1.6

0.0

+0.8

379 45.0%
719 27.3%
199 8.1%
266 4.1%
535 3.8%
160 1.7%
121 1.6%
309 1.5%

0 100 200 300 400 500 600 700

Latency [ms]

B

CSD

μV/cm2

0.05

0.10

0.15

117

156

309

254

191

500

383

707

-0.5

0.0

+0.7

-1.5

0.0

+0.8

707 40.5%
383 26.4%
191 7.3%
254 7.1%
500 4.7%
117 2.1%
156 2.1%
309 1.4%

Figure 5. Statistical evaluation of topographic FN (**A**, **B**) and fb-P3 (**C**, **D**) differences between conditions for ERP (**A**: N309; **C**: P379) and CSD (**B**: 309; **D**: 383) factors using randomization tests for paired samples (10,000 repetitions). In each panel, the top row represents the full sample (N = 61), and the following two rows represent the subsamples for low (n = 32) and high (n = 29) risk. For each panel and row, the columns show the two to-be-compared mean factor score topographies of two conditions (win vs loss for FN, win/loss vs neutral for fb-P3), their topographic difference, and squared univariate (channel-specific) paired samples T statistics threshold at the 95th quantile (p = 0.05) of the corresponding randomization distribution (maximum of all 72-channel squared univariate paired samples T statistics). To facilitate comparisons of the max(T^2) topographies with the underlying difference topographies, the sign of the difference at each site was applied to the respective T^2 value, which is otherwise always positive. For each row, asymmetric scales were applied for the score ranges of the two conditions, and symmetric scales were optimized for the score ranges of the differences between conditions and the max(T^2) topographies for better visualization and comparison of statistical effects across data transformations. All topographies are two-dimensional representations of spherical spline interpolations (m = 2; λ = 0) derived from the mean factor scores of T^2 statistics available for each recording site. Sites selected for further statistical analysis are marked by filled circles in the last column of each panel.

ERP

CSD

A

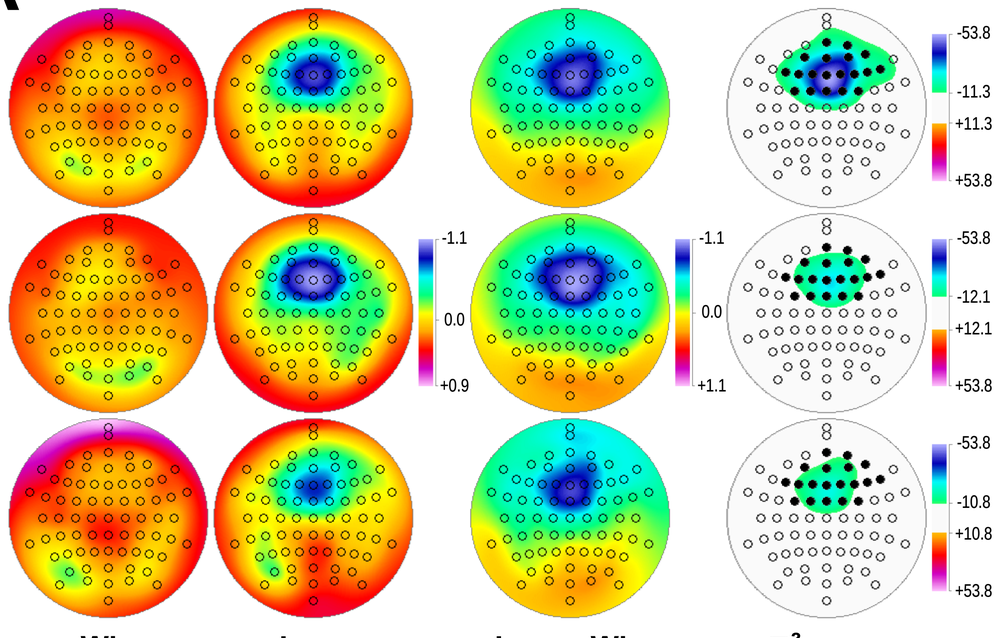
B

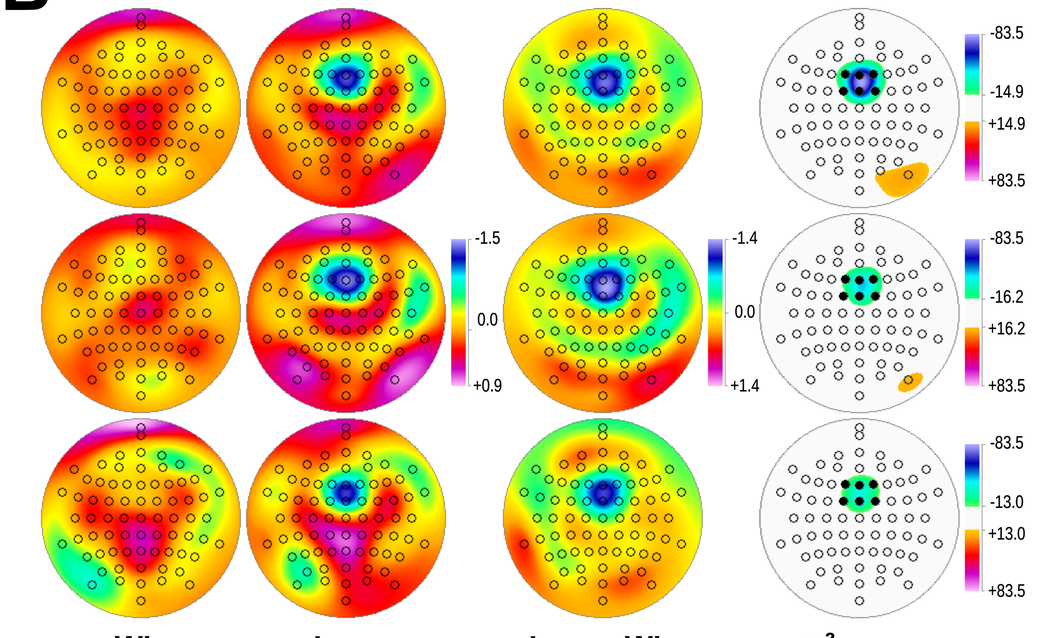
FN

Full Sample (*N* = 61)

Low Risk (*n* = 32)

High Risk (*n* = 29)





C

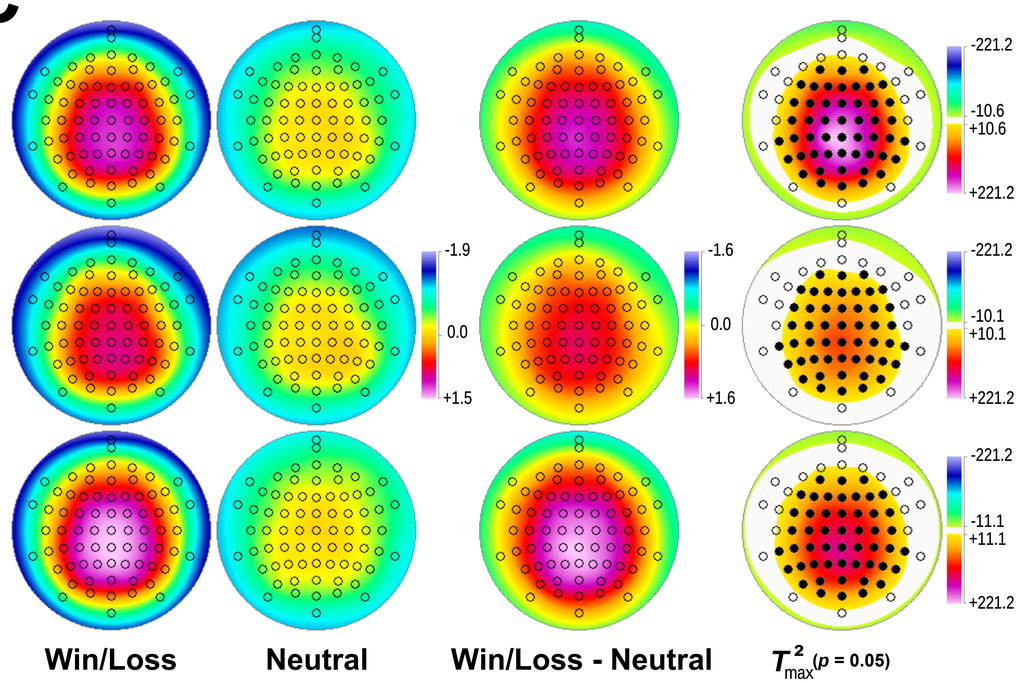
D

fb-P3

Full Sample (*N* = 61)

Low Risk (*n* = 32)

High Risk (*n* = 29)



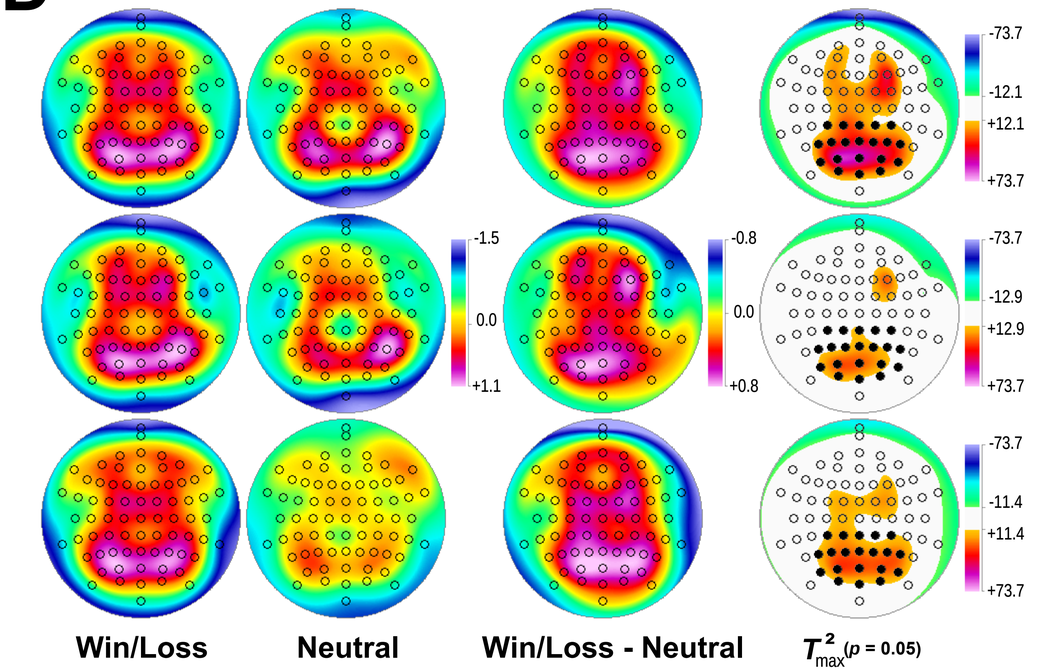


Figure 6. Predicted means (±SEM) of factor scores (pooled across representative sites) for ERP (**A**, **C**) and CSD (**B**, **D**) solutions corresponding to estimates of FN (**A**, **B**) and fb-P3 (**C**, **D**). Each panel shows the means for each stratification group (low vs high risk, with vs without a lifetime history of major depressive disorder [MDD+/-] or anxiety disorder [ANX+/-]) as a function of condition (win, loss, neutral). Note that the ordinate axis direction reflects component amplitude (i.e., negative for FN, positive for fb-P3), with its scaling optimized for the data range.

Low Risk | MDD- | ANX-
High Risk | MDD+ | ANX+

A

ERP FN

Risk

MDD

ANX

Factor Score

-0.8
-0.6
-0.4
-0.2
0.0
0.2
0.4

Win Loss Neutral

B

CSD FN

Risk

MDD

ANX

-1.2
-1.0
-0.8
-0.6
-0.4
-0.2
0.0
0.2

Win Loss Neutral

C

ERP fb-P3

Risk

MDD

ANX

Factor Score

0.8
0.6
0.4
0.2
0.0
-0.2
-0.4

Win Loss Neutral

D

CSD fb-P3

Risk

MDD

ANX

0.8
0.6
0.4
0.2
0.0

Win Loss Neutral