PARSING OUT ANTICIPATORY AND CONSUMMATORY REWARD UNDERLYING ANHEDONIA IN MOOD DISORDERS

by

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Abstract

Anhedonia presents in various psychiatric disorders and is a core symptom of depression. It involves disruptions in temporally and anatomically distinct subcomponents of reward processing, including reward anticipation and consumption. Studies using electroencephalography (EEG) to examine both anticipatory and consummatory reward processing components are limited, and study paradigms that incorporate psychological constructs like decision-making and reinforcement learning often overlap and confound the electrophysiological markers that are unique to anticipatory and consummatory reward processing. This study aims to validate an EEG-based Monetary Incentive Delay (MID) paradigm in a college population to examine relevant anticipatory and consummatory reward-related event-related potentials (ERPs), P3 and stimulus-preceding negativity (SPN), during different stages of reward processing. We found that the paradigm successfully elicited reward-related ERPs. Cue-P3 amplitudes and latency were modulated by reward magnitudes, however, no significant effect of reward magnitudes and valence was found on SPN and Feedback-P3, respectively. The paradigm was adjusted following the initial study to eliminate potential interfering visual effects from the cue and feedback stimuli. Additional data were collected in a new group of participants, and we found similar results, but without the confounding potentials. The paradigm also incorporated behavioural measurements of reward anticipation and consumption, and higher anticipatory and consummatory ratings and shortened response time towards the target stimuli were elicited as reward magnitudes increased. We concluded that the validated MID paradigm allows for a precise examination of reward-related ERPs, especially at early anticipation stage, and offers a valuable tool for investigating reward processing and related symptoms in clinical populations. Future studies should consider recruiting larger and more

diverse samples besides college populations to investigate symptoms of anhedonia and reward processing in clinical populations.

Lay Summary

Anhedonia, the inability to experience pleasure, is a defining symptom of major depressive disorder and can hinder treatment response. In this study, we investigate how the brain responds to wanting a reward and liking a reward by using electroencephalography (EEG) recordings to see how the brain responds to a specific event. Participants were instructed to press the spacebar after a cue was shown to win lottery tickets (the reward) towards a prize. We found higher reward correlated with enhanced P3, an ERP reflecting early reward anticipation. Reward magnitudes and outcomes did not affect late reward liking and wanting in this paradigm. This task was able to elicit greater experience of excitement in specific phases of reward processing, reflected in changes in brain activity recorded via EEG, and could serve as a valuable tool to examine anhedonia in individuals with depression and other psychiatric disorders characterized by anhedonia.

Preface

I completed this thesis under the supervision of Dr. Trisha Chakrabarty and Dr. Rebecca Todd. The leading concept of the experiment was designed by Dr. Trisha Chakrabarty, Dr. Rebecca Todd, Alexander Terpstra, and me. I conducted all data collection, analysis, and interpretation. All research in this thesis was approved by the University of British Columbia Behavioural Research Ethics Board (BREB), certificate ID H20-01388.

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List of Abbreviations

BDI	Beck's Depression Inventory
BIS/BAS	Behavioral Inhibition/Activation Systems
CMS	Common mode sense
COI	Channels of interest
DARS	Dimensional Anhedonia Rating Scale
DRL	Driven right leg
EEG	Electroencephalography
EOG	Electrooculogram
ERP	Event-related potential
fMRI	Functional magnetic resonance imaging
GAD	Generalized Anxiety Disorder
HCL	Hypomania Check List
MDD	Major depressive disorder
MID	Monetary Incentive Delay
RewP	Reward positivity
SPN	Stimulus-preceding negativity
SSRI	Serotonin reuptake inhibitor
STAI	State-Trait Anxiety Inventory

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Thank you to all my friends, for the moments of shared laughter and tears.

A very special thank you to my mom, for your unconditional supports and always being my rock and backbone.

Dedication

To my family and my grandfather (1942.02.06 – 2022.11.26)

愿姥爷天堂安好

Introduction

Anhedonia

Anhedonia is broadly defined as a "markedly diminished interest or pleasure in all, or almost all activities most of the day, nearly every day" (DSM-V; American Psychiatric Association, 2013). It is observed in many psychiatric disorders such as schizophrenia (Meehl, 1962), substance use disorder (Markou et al., 1998), eating disorders (Davis and Woodside, 2002), and post-traumatic stress disorder (PTSD) (Nawijn et al., 2015). Anhedonia is an important defining symptom of depressive episodes in mood disorders such as major depressive disorder (MDD) and bipolar disorder. It is a predictor of poor response to antidepressant medication (Uher et al., 2012, Vinckier et al., 2017), and is a key feature of treatment-resistant depression (Kelly et al., 2022). Primary pharmacological treatment for MDD, such as selective serotonin reuptake inhibitors (SSRIs) fail to effectively alleviate symptoms of anhedonia which might be accountable for the lack of efficacy and resistance of the treatment (McCabe et al., 2010, Treadway & Zald, 2013). A fulsome understanding of the neurobiological correlates of anhedonia is thus critical to advancing effective therapies for mood and other psychiatric disorders characterized by anhedonia.

Components of Reward Processing and Anhedonia

Anhedonia is believed to be correlated and manifested from dysfunction in the reward processing system. This dysfunction involves disruption and alteration in the mesolimbic reward circuitry, including key areas that mediate reward-related information processing like the prefrontal cortex (PFC), ventral tegmental area (VTA), and nucleus accumbens (NAcc) that mediate reward processing. Both adults and youth with MDD report reduced reward sensitivity

towards appetitive stimuli and increased anhedonia (Alloy et al., 2016; Luby et al., 2004), and individuals with blunted reward sensitivity are at risk to develop MDD (Alloy et al., 2016).

Disruption in distinct subcomponents of reward processing may underlie anhedonia. While reward processing is complex and involves several distinct subcomponents, two of particular relevance to anhedonia are reward anticipation (motivation to pursue rewards) and reward consumption (ability to enjoy the reward). Depending on the stages of reward processing that are affected, the clinical presentation of anhedonia can vary (Borsini et al., 2020; Treadway & Zald, 2011); for example, impairments in reward anticipation could result in diminished interest and motivation to pursue rewarding stimuli, while impairments in reward consumption could lead to the inability to derive enjoyment from a pleasurable event (Treadway & Zald, 2013, Winer et al., 2019). That temporally distinct stages of reward processing relate to clinical symptoms of anhedonia was first proposed by Dr. Donald Klein in 1984 in the book *Anhedonia and Affect Deficit States*. He noted in clinical practice that successful pharmacologic treatment for depression would restore the consummatory phase of reward first, followed by motivation to pursue reward (Klein, 1984).

On top of the multifaceted clinical presentation of reward processing deficits, extensive research also shows that anticipatory and consummatory reward processing involves temporally and spatially distinct functional networks (Berridge et al., 2009). Anticipatory reward processing is thought to be controlled by the dopaminergic pathway that activates the ventral striatal regions, particularly at the nucleus accumbens (NAcc) (Knutson et al., 2001a); whereas consummatory reward processing is controlled by opioid and serotonergic mechanisms that activate the ventromedial frontal cortex (Der-Avakian et al., 2016). The explicit neural circuits involved in each processing stage were first discovered in rodent studies, where researchers

found that dopamine release only mediated the anticipatory stage of reward but not the hedonic pleasure of reward consumption, and the opioid, endocannabinoid, and GABA-ergic pathways were shown to contribute to generating pleasure to appetitive stimuli (Berridge et al., 2009; Robinson & Berridge, 1993). Similar results were seen in human neuroimaging studies, with the ventral striatum, and specifically the NAcc, active during anticipation of expected positive reward but not reward consumption, while the orbitofrontal (OFC) and ventromedial prefrontal (vmPFC) cortical regions and subcallosal cortex were recruited in reward consumption but not anticipation (Dillon et al., 2007; Knutson et al., 2001b; Oldham et al., 2018). Despite these distinct neural circuits and functions, anticipatory and consummatory reward processing are still highly coupled; for instance, increased reward consumption is often correlated with increased reward anticipation in healthy populations. However, how those connections may be disrupted in anhedonia, and how to disentangle them into substages to facilitate diagnosis and treatment, remains unclear (Admon & Pizzagalli, 2015; Eshel & Roiser, 2010).

Distinguishing between anticipatory and consummatory reward processing can further our understanding of psychiatric diseases since not all disorders present anhedonia similarly. For example, in schizophrenia patients often present with impaired reward anticipation but relatively intact consummatory reward (Gard et al., 2007; Wang et al., 2015). MDD patients often report clinical impairment in both reward anticipation and consumption, however, there also have been cases where patients present without impairment in reward consumption, suggesting a subtype of MDD that does not exhibits dysfunction in reward consumption, which might have an impact on their clinical presentation and treatment response (Rizvi et al., 2016; Sherdell et al., 2012).

Assessing Anticipatory and Consummatory Reward in Anhedonia

Current clinical tools for assessing anhedonia generally conflate consummatory and anticipatory reward processing. Commonly used self-report questionnaires such as the Snaith-Hamilton Pleasure Scale (SHAPS) often fail to disentangle anticipatory and consummatory phases of reward processing and exclusively focus on the consummatory phase (Rizvi et al., 2016); although, the more recently developed, newer generations of scales have started to focus on distinguishing between anticipatory and consummatory pleasures (i.e., Temporal Experience of Pleasure Scale (TEPS); Gard et al., 2006). That being said, current practices may overgeneralize anhedonia, and therefore, may miss specific therapeutic targets. It's been suggested that instead of a global target of anhedonia symptoms, tailored interventions toward individuals' experience in anhedonia would be more effective in both evaluating and addressing treatment (Treadway & Zald, 2013; Winer et al., 2019) and future research should conceptualize anhedonia as a multifaceted deficit with distinct clinical phenotype of reward processing alteration in order to better characterize anhedonia and dissociate its subcomponents in different disorders (Admon & Pizzagalli, 2015; Borsini et al., 2020). To achieve that, researchers have been using behavioral measures like self-reported scales (e.g., The Dimensional Anhedonia Rating Scale (DARS), Behavioral Inhibition/Activation Systems (BIS/BAS), as well as computer-based behavioral tasks (e.g., probabilistic reinforcement learning paradigms and gambling paradigms; Berridge et al., 2009; Burkhouse et al., 2018) to study subcomponents of reward processing.

Electrophysiological studies in reward processing

Because reward processing involves a large brain network and could be modulated by different stimuli and behaviour, researchers have become more reliant on neuroimaging techniques to gain

a better understanding of the mechanisms involved in reward processing in the human brain. Among those, functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) scans have been the most popular methods for investigating the underlying networks and neuromodulators that are involved during reward processing (Calabro et al., 2023; Knutson et al., 2001b). Knutson and colleagues have developed the monetary incentive delay (MID) task to separate activity elicited by reward anticipation and outcome consumption to better dissociate different stages of reward processing (Knutson et al., 2000). The MID has been widely used to study the effects of reward and punishments in different conditions in both healthy and clinical populations, and the development of the MID paradigm for fMRI has enabled investigation of the heterogeneities and subcomponents of reward processing. However, signals associated with different stages are often problematically con-founded due to poor temporal resolution in fMRI.

Electroencephalography (EEG), with its fine temporal resolution, has the potential to parse out the anticipation and consumption of reward in a single task. EEG studies to date have largely focused on decision-making and reinforcement learning tasks; in a review by Glazer et al (2018), it was noted that the majority of studies had neglected the "rich temporal heterogeneity of reward processing" and focused solely on the consumption phase of reward processing. Distinct temporal patterns of brain activity are represented in EEG via event-related potentials (ERPs). ERPs can be modulated by experimental manipulations, are time-locked to the onset of an event and usually elicited by sensory stimuli or motor action (Rugg, 2001). Distinct ERPs can potentially unpack stages of reward anticipation and consumption processing and those components down to milliseconds. Historically, the field had extensively investigated feedback-related and error-learning ERPs seen during reward consumption, such as the feedback-related negativity (FRN), or reward positivity (RewP) (Sambrook & Goslin, 2015). More recently, the

field has shifted toward exploring other stages of reward processing such as anticipatory reward. MID tasks may be particularly useful in this regard, as they have demonstrated the ability to separate stages of reward processing (Meyer et al., 2021).

Different ERPs are identified during three different phases of reward anticipation: cue evaluation, motor preparation, and feedback anticipation. Similarly, subcomponents of feedback-related processing during reward consumption include early reward reactivity, updating working memory, and affective processing of information (Glazer et al., 2018). Processing of reward informational unfolds sequentially in a few seconds, and multiple cognitive processes, such as reinforcement learning and decision making, can take place within this brief time. These processes, although closely related to and potentially involved in reward processing, could yield inconsistent and complex results due to components overlapping and interference (Glazer et al., 2018). Therefore, the aim of the task paradigm and the selection of ERPs is to better isolate unique reward processing components that are independent of decision-making and learning but include investigation in both reward anticipation and consumption phases.

Reward-related event-related potentials (ERPs)

We selected three relevant ERPs to examine in our study: Cue-P3 which occurs during early reward anticipation, stimulus-preceding negativity (SPN) which occurs during late reward anticipation, and lastly, Feedback-P3 which occurs during reward consumption. P3 or P300 is an ERP component that is visualized as the centroparietal positivity that peaks at around 300-600ms after cue onset. It was shown to be elicited by salient stimuli resulting in an update in working memory and especially enhanced by incentive information on rewards (Hughes et al., 2013). Previous studies also found that reward-specific P3 that was elicited by incentive cues covaried with activation in the ventral striatum (Pfabigan et al., 2014), which is a region implicated in

motivational reward processing. During feedback processing, P3 had shown to be sensitive to the motivational salience of the stimuli and updating outcome-related information to optimize future actions to maximize rewards (Glazer et al., 2018; San Martín, 2012). Another ERP component during anticipation that has also been overlooked by most outcome-focused studies is SPN which occurs at a late stage of reward anticipation preceding outcome onset and is presented as a passive and sustained negativity that grows with anticipation of information (Ohgami et al., 2006). It serves as a promising index of anticipatory reward processing since it's shown to be elevated by highly informative stimuli, especially the anticipation of feedback associated with monetary rewards, (Glazer et al., 2018; Mattox et al., 2006 Walentowska et al., 2018). Enhanced SPN is also observed with increased reward magnitudes, greater efforts, and greater uncertainties, which is not surprising since those components are usually associated together in most paradigms, with higher reward representing greater uncertainties to getting the reward (Hackley et al., 2013; Teti Mayer et al., 2021; see Zhao et al., 2017 for alternative).

These ERPs allow for the examination of the neurophysiological features of reward processing in both anticipatory and consummatory phases, while parsing out the effects of reinforcement learning and decision-making. This enables a more precise understanding of the neurophysiological features pertaining to reward processing, which exhibits a more direct association with reward-related symptoms such as anhedonia (Treadway & Zald, 2013).

In the current study, our goal is to validate a MID EEG paradigm as a tool to investigate reward magnitudes and valence effects of reward-related ERP (i.e., Cue-P3, Feedback-P3, and SPN). To ensure the elicited ERPs are relatively pure to reward anticipation and reward consumption, we specifically eliminated reward learning, punishment, or neutral conditions in the task. We hypothesized that as reward magnitudes increase, we will see an increase in both

behavioural and physiological responses (i.e., higher ratings in excitement and higher P3 and SPN amplitudes). The effect of reward valence is difficult to predict since some studies showed enhanced Feedback-P3 amplitudes in reward gain versus reward loss (Mei et al., 2018; Sato et al., 2005; Wu & Zhou, 2009), and some studies showed the opposite or insensitive to reward valence and performance evaluation (Foti et al., 2011; Hajcak et al., 2005; Yeung & Sanfey, 2004). The involvement of reinforcement learning and error updates might contribute to the mixed results in Feedback-P3, however as our paradigm does not contain those components, we might be able to observe a more precise relationship between reward valence and Feedback-P3. We developed two study paradigms: an EEG-adapted MID paradigm followed by a revised paradigm with stimuli modifications to better capture the ERP components based on the results observed from the initial experiment. This study will explore whether the current MID paradigm would serve as a reliable tool to investigate electrophysiological changes associated with the anticipatory and consummatory stages of reward processing and serve as an exploratory study on how anhedonia-related symptoms would correlate with ERP changes.

Experiment 1

Methods

Participants

A total of 33 participants (12 males, 1 non-binary, mean age = 22.55 years, SD = 5.88) were recruited through the Human Subject Pool system from the University of British Columbia and received course credit for their participation. 1 participant was excluded due to technical failure, 2 were excluded from EEG analysis due to medication history, leaving N = 32 in the behavioral analysis (11 males, 1 non-binary, mean age = 22.63 years, SD = 6.00), and N = 30 in the EEG analysis (11 males, 1 non-binary, mean age = 22.90 years, SD = 6.04). The sample size was predetermined by power analysis based on an observed effect size in a similar reward processing task in previous studies (Meadows et al., 2016) (n=20). With a partial $\eta 2$ of 0.256, significance criterion of $\alpha = .05$, and power = .80, the minimum sample size needed with this effect size is N = 30 for a within-effect repeated measure ANOVA test.

Table 1. Participant demographics in Experiment 1

Variables	Mean ± SD
Age	22.55 ± 5.88
% Female	21 (63.36%)
BDI	10.44 ± 8.98
GAD	4.75 ± 4.63
HCL	14.13 ± 6.38
STAI-S	39.91 ± 11.16
STAI-T	47.38 ± 10.43
DARS	51.72 ± 11.53
BIS	12.38 ± 3.52
BAS	9.13 ± 2.46

Procedure

Each participant was contacted approximately 24 hours before the study via email for instructions to prepare for the EEG recording, including cleaning their hair and avoiding spray, lotion, or oil products, avoiding wearing make-up and contact lenses during testing, avoiding caffeine intake before testing, avoiding sleep-deprivation, and assessing possible COVID-19 related exposure. Consent to participate in the study and reward lottery draw was obtained upon participants' arrival in the lab through a Qualtrics survey form. Demographic and Daily Evaluation questionnaires, which record information on self-report food intake, sleeping hours and quality, caffeine, tobacco, alcohol, and medication intake, were given after consent was obtained. When participants arrived at the lab, EEG data were collected while they completed a Monetary Incentive Delay task. After EEG recording, participants were asked to complete additional validated self-reported scales including the Beck's Depression Inventory (BDI) (Beck et al, 1960), Generalized Anxiety Disorder 7-item (GAD-7) (Spitzer et al., 2006), Hypomania Check List (HCL-32) (Angst et al., 2005), BIS/BAS (Carver & White, 1994), State-Trait Anxiety Inventory (STAI) (Spielberger et al., 1983), and the Dimensional Anhedonia Rating Scale (DARS) (Rizvi et al., 2015) to assess their clinical symptoms of depression, anxiety, hypomania/mania, and anhedonia. A lottery draw was conducted at the end of the study in August 2023 to reward one participant with $100.

Monetary Incentive Delay Task

The Monetary Incentive Delay Task was first developed by Knutson and colleagues in 2000 (Knutson et al., 2000), and the current incentive delay paradigm was an adaptation of the task. In the present study, participants were instructed that they are playing for tickets that will be entered into a lottery draw of $100 at the end of the study. During the task, participants were first

presented with an incentive cue for 1000ms. The incentive cue was an image of one, three, or five lottery tickets, indicating the magnitude of the reward they were playing for in the current trial. Following the cue participants were asked to rate their anticipatory excitement in response to the question “How excited do you feel?” on a number scale from 1 to 7, where 1 indicated “neutral” or minimal excitement, and 7 indicated “very excited”. The rating was presented for 2000ms, followed by a fixation cross that was jittered between 500 and 750ms. Then a smiley face icon was presented in the middle of the screen for 100ms. When the face appeared, the participants were instructed to press the spacebar as quickly as they could to win the tickets. If the participant failed to make a response within one second, the trial was automatically looped back to restart. After participants made a response, there was a delay of 1250, 1500, or 1750ms before feedback. Feedback indicating either a win, shown as a green thumb up, or a non-win, shown as a red thumb down, for 1000ms. Finally, another rating scale assessing consummatory reward rating appeared for 2000ms asking “How excited do you feel” if the feedback was positive, and “How disappointment do you feel” if the feedback was negative. Unbeknown to participants, each result (win vs. non-win) was pre-set and appeared in a pseudo-randomized order such that in each round, there were equal numbers of reward magnitudes in the win and non-win conditions.

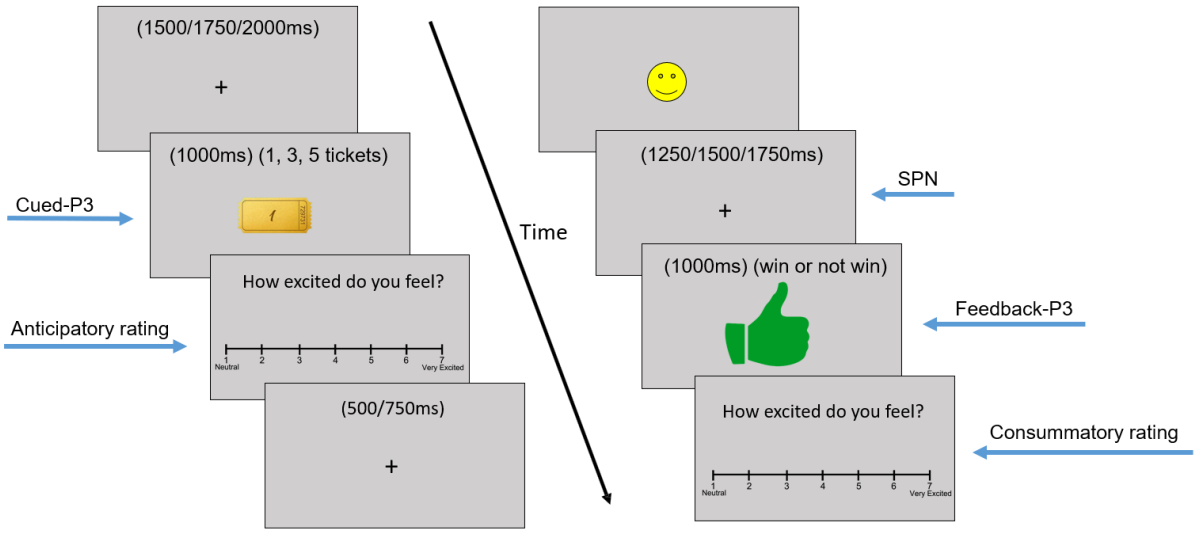

Figure 1. Schematic representation of the monetary incentive delay task and related ERP and beahviroual measurements captured.

Self-Report Questionnaires

Beck's Depression Inventory (BDI)

BDI is a 21-item, self-report inventory that measures depressive symptoms (Beck et al., 1960). It's widely used in clinical and research practice as a well-established scale with high internal consistency in both psychiatric and nonpsychiatric samples, high content, and convergent validity with depression rating scales (Richter et al., 1998).

Generalized Anxiety Disorder 7-item (GAD-7)

GAD-7 is a 7-item, self-report scale assessing anxiety symptoms over the past two weeks on a 4-point scale. The scores can range from 0 to 21, with higher scores indicating severe generalized anxiety disorder (GAD) symptoms. The scale has shown good criterion, construct, factorial, and procedural validity, and is an efficient tool to assess GAD in clinical and research settings (Spitzer et al., 2006).

State-Trait Anxiety Inventory (STAI)

The State-Trait Anxiety Inventory (STAI) form Y consists of two parts: Y-1 and Y-2, each has 20 statements on a 4-point scale to measure a person's state anxiety (i.e., how anxious they are feeling right now) and trait anxiety (i.e., how anxious they feel in general) (Spielberger et al., 1983). Each part's scoring varies from 20 to 80 points, where a higher score indicating higher anxiety with a suggested clinical cut of 39-40 detecting clinically significant symptoms. The scale shows high internal consistency, and STAI S-Anxiety (Y1) showed a high correlation with depression since it was derived from testing situations with highly stressed states (Julian, 2011).

Hypomania Check List (HCL-32)

The Hypomania Checklist-32 (HCL-32) is a 32-item measure of hypomanic thoughts and behaviors that is sensitive to sub-clinical traits (Angst et al., 2005). The scale includes preliminary items regarding current feelings compared to respondents' usual state and how respondents are usually compared to other people, followed by 32 yes-or-no hypomania-related statements. For the current study, only respondents' responses to the 32 yes-or-no questions were used.

Behavioral Inhibition/Activation Systems (BIS/BAS)

The BIS/BAS Scale is a 24-item, self-report inventory, to measure the behavioral inhibition system (BIS), which activates inhibition and avoidance responses, and the behavioral activation system (BAS), which facilitates goal-motivated approaching behavior on a 4-point scale (Carver & White, 1994). Epidemiological study has shown that BIS/BAS is a reliable factor for detecting vulnerability to psychiatric disorders such as depression, anxiety, and drug and non-comorbid alcohol abuse (Johnson et al., 2003).

Dimensional Anhedonia Rating Scale (DARS)

The DARS is a self-report inventory that measures anhedonia levels across four domains: hobbies, food and drinks, social activities, and sensory experiences. Within each domain, participants are required to provide two or three of their own examples that they enjoy doing. The scale was developed specifically to target four different facets of anhedonia: interest, motivation, effort, and pleasure, and had shown high reliability and convergent and divergent validity (Rizvi et al., 2015).

Analysis

EEG Recording and Analysis

EEG recording was obtained from a 64-channel Biosemi Actiview system. Common mode sense (CMS) and driven right leg (DRL) ground electrodes and Electrooculogram (EOG) electrodes were placed over medial-parietal cortex. Continuous EEG data were recorded at a sample rate of 256 Hz. All EEG data were processed offline using MATLAB R2022a (The MathWorks Inc., 2022), EEGLAB v2022.1 (Delorme & Makeig, 2004), and ERPLAB 9.0 (Lopez-Calderon & Luck, 2014). Offline data preprocessing was completed using the PREP pipeline 0.55.4 (Bigdely-Shamlo et al., 2015) to high-pass-flite with 0.01 Hz, remove line noise, and re-reference all EEG channels to an average reference. An additional low-pass-filter of 30 Hz was added after PREP pipeline. P3 ERPs were epoched from -200ms to +800ms, time-locked to stimulus onset, and baseline corrected to 200 ms prestimulus. SPN ERPs were epoched from -1200ms to +200ms, time-locked to feedback stimulus onset, and baseline corrected to a window -1200ms to -1000ms from the stimulus. Epoched data was processed automatically for artifact rejection using a peak-to-peak detection with a 100ms window, then data were visually inspected for additional eye movement and blinks. 5 participants had less than 20 available trials in a

condition where their data were marked too noisy and not included in further processing, leaving 25 participants each had more than 20 trials available in each condition in further ERP analysis. Finally, each participant's data were averaged for each condition (cue-P3 and SPN: reward magnitudes, feedback-P3: reward valance).

Table 2. *Mean and standard deviation of the number of trials averaged in each condition in Experiment 1.*

Condition	Mean	SD
Cue one ticket	66.52	17.63
Cue three tickets	62.72	18.94
Cue five tickets	63.84	16.61
Feedback win	79.84	29.56
Feedback non-win	76.92	29.86
SPN one ticket	58.80	15.47
SPN three tickets	58.48	17.39
SPN five tickets	59.28	15.84

Upon visually examining the averaged ERPs, we observed differences between conditions for a negative peak at around 150ms, which could be caused by visual inconsistency in our stimulus sets that could potentially carry over into and confound P3 amplitude differences. To control for potential carryover effects, we performed a peak-to-peak analysis from the

negative peak prior to P3 to the positive peak at P3, so that the preceding negativities caused by visual effects are not contributing to the amplitudes. The Cue-P3 and Feedback-P3 and the preceding negativities were measured at Oz where the largest amplitudes were observed in the grand mean data. To measure the Cue-P3, the negative peak prior to the P3 was extracted between 150-250ms and the positive peak and peak latency for the cue-P3 was extracted between 230-400ms at midline central and parietal sites: Cz, CPz, Pz, POz, CP1, and CP2 (Mathalon, 2009; Salisbury et al., 2001). The Feedback-P3 was measured between 150-300ms (negative peak) and between 250-400ms (positive peak and peak latency) (Hajcak et al., 2007) at the same sites. SPN amplitudes were examined from 200-0ms preceding feedback stimuli at centroparietal sites: CPz, Pz, CP1, and CP2 (Guyer et al., 2019).

Task Behavioural Analysis

We performed a repeated measure analysis of variance (ANOVA) to assess participants' excitement ratings during reward anticipation and reward consumption, using reward magnitudes (1, 3 and 5 tickets) and rating type (anticipatory ratings and consummatory ratings) as factors. The test analyzed whether ratings increased on average as the number of tickets played for and won increased, as well as whether anticipatory and consummatory ratings differed when played and won the same number of tickets. Repeated measure ANOVA was also used to compare mean response times to target stimuli as the number of tickets increased. We did not conduct any analysis on the non-win condition since the reward consumption implies the acquisition of a reward, and in the non-win scenario, rewards were omitted.

Exploratory Analysis

Participants' responses to the questionnaires were collected and scored based on the survey instructions. We do not have enough data to have enough power in correlational analysis

or control for multiple comparisons, therefore the regressions that were performed on the correlation between each questionnaire score and ERPs amplitudes, and score and reward sensitivity ratings were exploratory on the current dataset, and no conclusion was drawn from the results.

Results

Task behaviour

We conducted a repeated-measure analysis of variance (ANOVA) on excitement ratings using levels of reward magnitudes (1, 3, and 5) and incentive type (anticipatory and consummatory). We found main effects of reward magnitudes, [$F(1.10, 34.15) = 59.47$, $p < .001$, partial $\eta 2 = .66$], incentive type, [$F(1.00, 31.00) = 38.42$, $p < .001$, partial $\eta 2 = .55$] and an interaction effect of reward magnitudes and incentive type, [$F(5.64, 8.06) = 21.70$, $p < .001$, partial $\eta 2 = .41$]. Separate repeated-measure ANOVAs and post-hoc pairwise t-tests showed that both anticipatory and consummatory ratings increased with the number of tickets (1 vs. 3 vs. 5), with all $p < .001$.Post-hoct-test revealed that, for 1 and 3 tickets, anticipatory ratings were significantly lower than consummatory ratings, [$t(31)=-7.36$, $p < .001$], and [$t(31) = -6.38$, $p < .001$]. For 5 tickets, the disparities between anticipatory ratings ($M = 4.15$, $SD = 0.28$) and consummatory ratings ($M = 4.48$, $SD = 0.28$) progressively decreased, $t(31) = -2.30$, $p = 0.01$. There was also a significant decrease in response times as the number of tickets played for increased, [$F(2, 40) = 28.93$, $p < .001$, partial $\eta 2 = .59$], and pairwise t-test showed a significant decrease in reaction time when playing for 3 tickets ($M = .37$, $SD = .02$) compared to 1 ticket ($M = .38$, $SD = .02$), [$t(20) = 3.07$, $p = 0.01$], and when playing for 5 tickets ($M = .35$, $SD = .02$), [$t(20) = 4.44$, $p < .001$].

Table 3. *Repeated-measure ANOVA results of the behvaioural ratings to assess the effects of reward magnitudes and types of ratings in Experiment 1.*

Effects	Sum of Square	*df*	Mean square	*F*	*p*	Partial η2
Magnitudes	80.887	1.10	73.41	59.47	< .001	.66
Error(Magnitudes)	42.15	34.15	1.23			
IncentiveType	28.57	1.00	28.57	38.42	< .001	.55
Error(IncentiveType)	23.05	31.00	0.74			
Magnitudes * IncentiveType	5.64	1.15	4.92	21.70	< .001	.41
Error(Magnitudes* IncentiveType)	8.06	35.53	0.23			

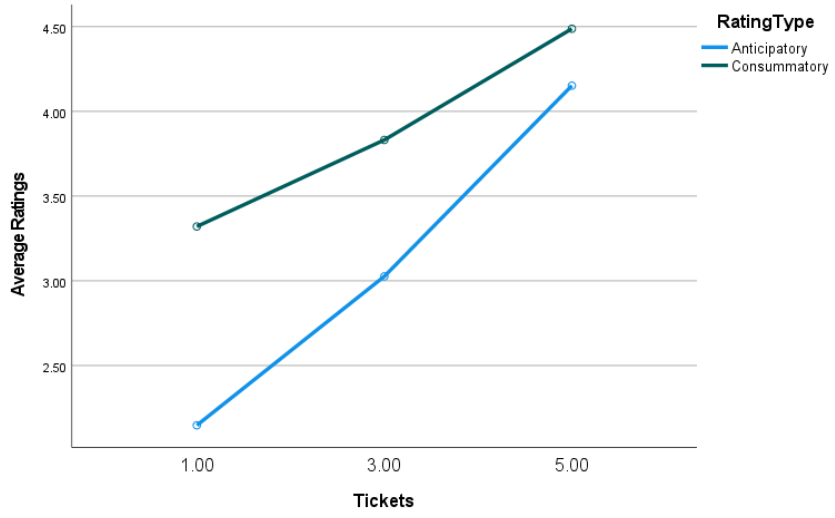

Figure 2. Averaged anticipatory and consummatory ratings in three reward magnitudes levels in Experiment 1.

Table 4. *Repeated-measure ANOVA results of reaction time to targets to assess effects of reward magnitudes in Experiment 1.*

Effects	Sum of Square	*df*	Mean square	*F*	*p*	Partial η2
Magnitudes	.01	2	.01	28.93	< .001	.59
Error(tickets)	.01	40	.00			

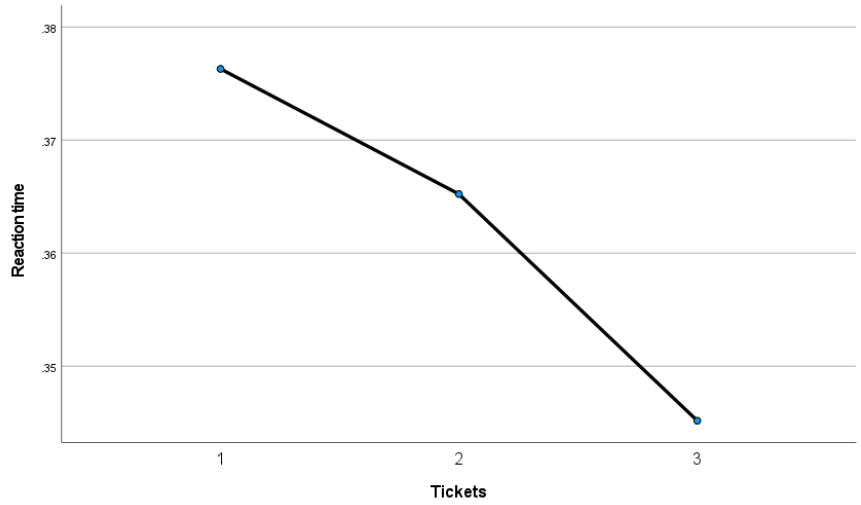

Figure 3. Averaged reaction time to targets in three reward magnitudes levels in Experiment 1.

EEG

Repeated measure ANOVA for the Cue-P3

Cue-P3 amplitudes were extracted from eight different scalp channels of interest (COI), Cz, CPz, Pz, POz, CP1, and CP2 using the peak-to-peak approach described above. A repeated measure ANOVA was calculated on Cue-P3 amplitudes using the levels reward valence (1, 3, and 5) and channel location (Oz, POz, Pz, CPz, Cz, CP1 and CP2). Main effects and interactions were corrected using the Greenhouse-Geisser correction if sphericity is violated. Results showed main effects of both reward magnitudes, [$F(1.83, 43.89) = 13.93$, $p < .001$, partial $\eta 2 = .37$], and

COI [$F(1.86, 44.75) = 42.40$, $p < .001$, partial $\eta 2 = .64$]. Furthermore, there was also a significant interaction between reward magnitudes and COI, [$F(4.21, 101.14) = 5.09$, $p < .001$, partial $\eta 2 = .18$]. Separate repeated measure ANOVAs were conducted on individual COI and reward magnitudes as factors to explore the interaction effects. As shown in Table 5, there was a highly significant difference in ERP amplitudes between reward magnitudes at channel Oz [$F(1.51, 36.21) = 13.29$, $p < .001$, partial $\eta 2 = 0.36$], and that amplitude in 1 ticket condition was significantly lower than 3 tickets and 5 tickets ($p < .001$); at channel POz [$F(2, 48) = 7.90$, $p = 0.001$, partial $\eta 2 = 0.25$], P3 amplitudes in 1 ticket condition were significantly lower than 3 tickets ($p < .001$), and there was no significant difference in 1 and 3 tickets conditions in comparison with 5 tickets conditions; and finally at channel Pz, [$F(2, 48) = 3.98$, $p < 0.05$, partial $\eta 2 = 0.14$], and at channel CP1, [$F(2, 48) = 3.67$, $p < 0.05$, partial $\eta 2 = 0.13$], P3 amplitudes in 1 ticket condition were significantly lower than 5 tickets condition ($p = 0.01$).Pairwise comparisons using LSD correction showed that overall Cue-P3 amplitudes were significantly higher for 3 tickets ($M = 4.98$, $SD = 0.44$) and 5 tickets ($M = 5.31$, $SD = 0.45$) compared to 1 ticket ($M = 3.99$, $SD = 0.35$) ($ps < .001$), and we did not find a significant difference between 3 tickets and 5 tickets ($p = .23$). Peak latency was also analyzed using repeated measure ANOVA with the same factors and levels which showed a significant main effect on magnitudes, [$F(1.93, 46.21) = 5.15$, $p = .01$, partial $\eta 2 = .18$], and COI, [$F(3.85, 92.47) = 8.25$, $p < .001$, partial $\eta 2 = .26$]. There was also an interaction effect on magnitudes and COI, [$F(6.36, 152.64) = 2.84$, $p = 0.01$, partial $\eta 2 = .11$]. Additional ANOVA revealed significant effects at channel CPz, [$F(2, 48) = 5.75$, $p = 0.01$, partial $\eta 2 = 0.19$], where peak latency in 1 ticket condition was shorter than 3 tickets condition ($p < 0.05$) and 5 tickets condition ($p < .001$); at channel Cz, [$F(2, 48) = 5.83$, $p = 0.01$, partial $\eta 2 = 0.20$], where similarly, peak latency was shorter in 1 ticket condition in

comparison with 3 tickets condition ($p < 0.05$), and 5 tickets condition ($p < 0.01$); and finally at channel CP1, [$F(2, 48) = 3.16$, $p = 0.05$, partial $\eta 2 = 0.12$], where peak latency was significantly shorter in 1 ticket condition in comparison with 5 tickets condition ($p < 0.01$). Peak latency for 1 ticket ($M = 310.07$, $SD = 6.04$) was significantly shorter than 3 tickets ($M = 326.94$, $SD = 5.89$, $p = 0.01$), and 5 tickets ($M = 325.20$, $SD = 6.27$, $p = 0.01$).

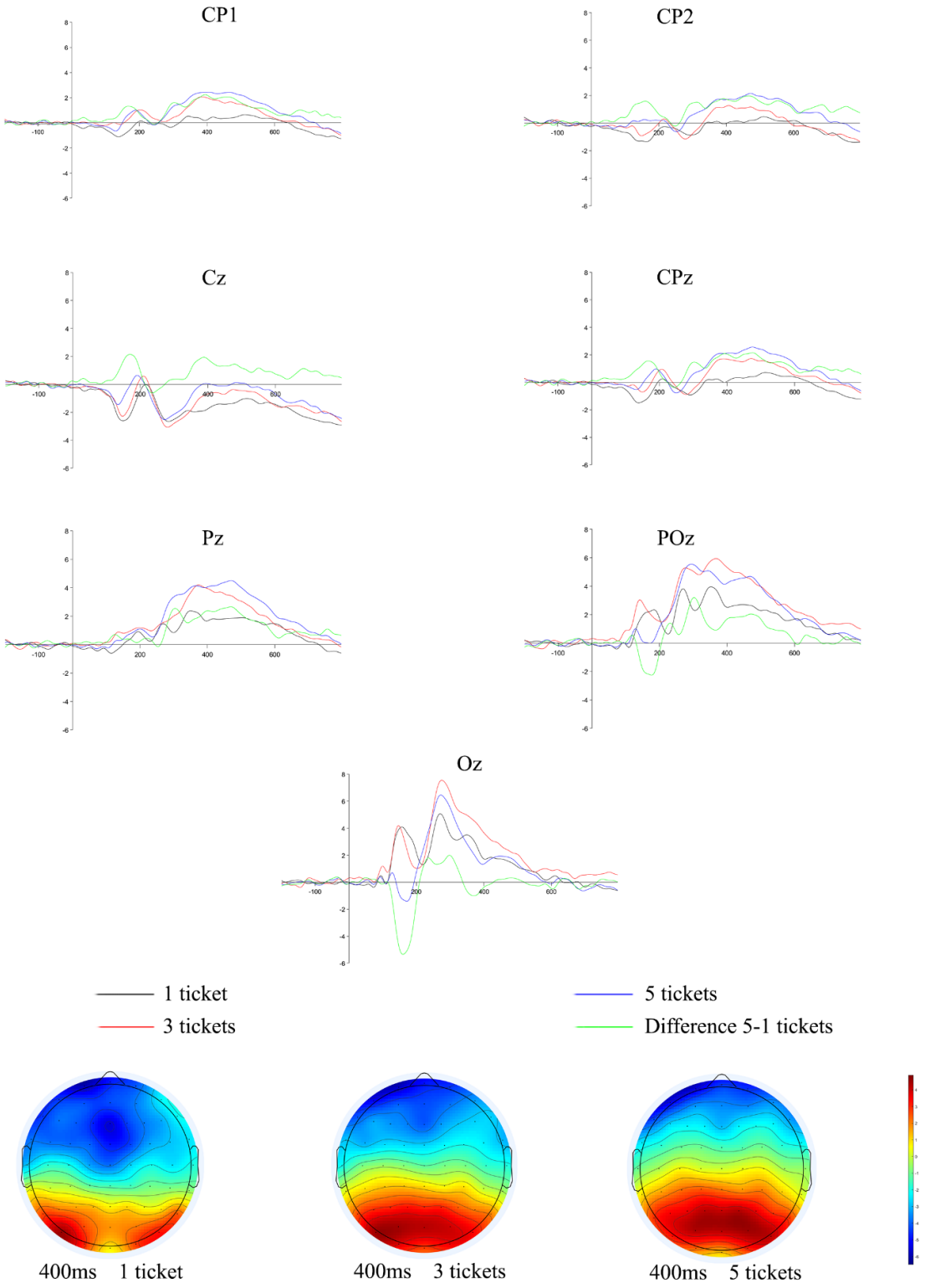

Figure 4. Cue-P3 grand average ERP waveforms from -200ms to 800ms and topography at 400ms in Experiment 1.

Repeated measure ANOVA in Feedback-P3

We also analyzed ERP amplitudes for feedback-P3 against the same COIs and between the win and not win conditions. A repeated measures ANOVA was calculated on Feedback-P3 amplitudes using the levels reward valence (1, 3 and 5) x channel location (Oz, POz, Pz, CPz, Cz, CP1 and CP2). Results showed a significant main effect for COI, [$F(1.92, 46.00) = 99.18$, $p < .001$, partial $\eta 2 = .28$]. We did not find any significant effects for reward valence, [$F(1, 24.00) = 1.05$, $p = 0.32$, partial $\eta 2 = .04$], nor reward valence and COI interaction, [$F(2.54, 148.56) = 0.41$, $p = 0.80$, partial $\eta 2 = .02$]. Latency analysis only found a main effect in COI, [$F(3.84, 92.21) = 15.30$, $p < .001$, partial $\eta 2 = .39$].

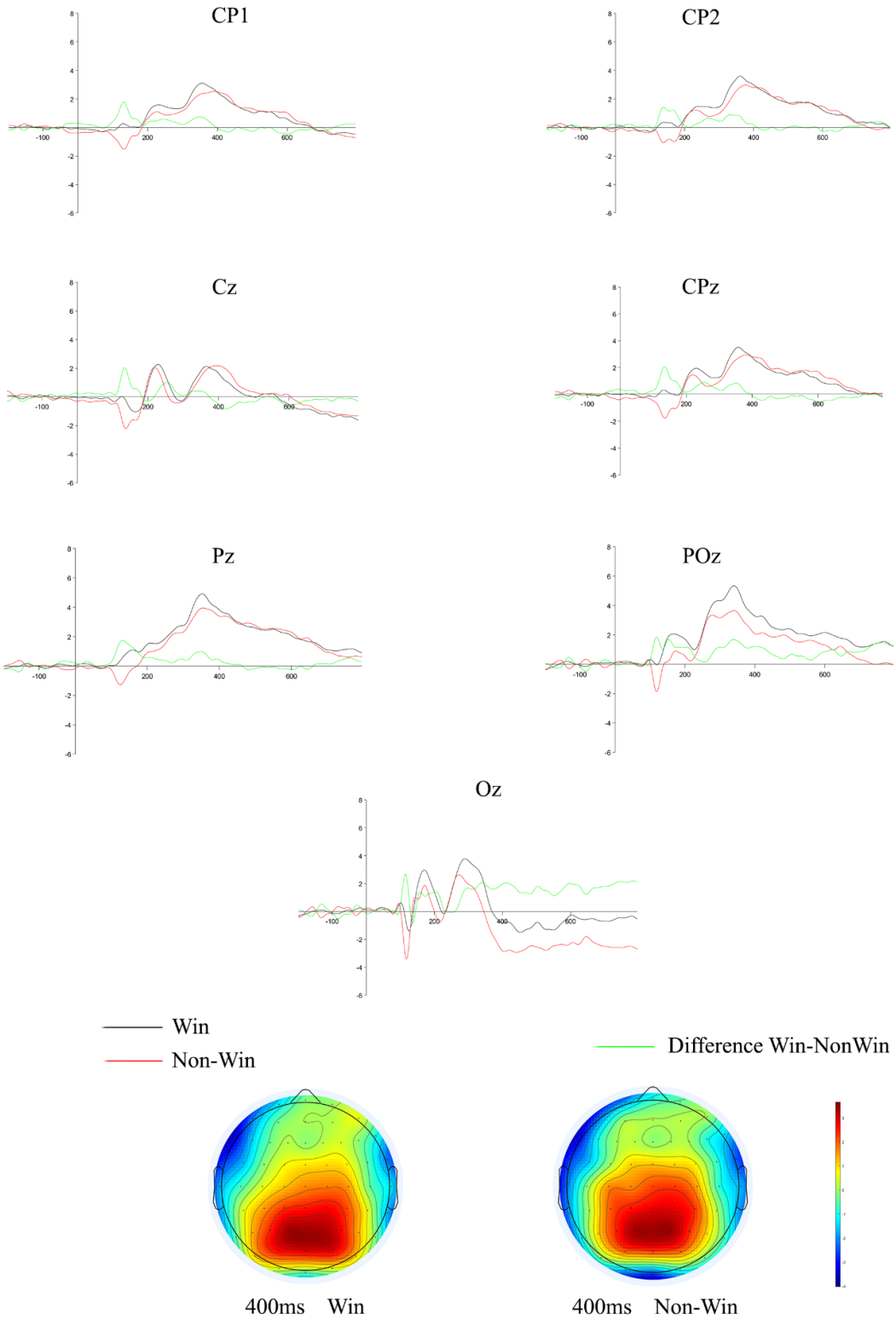

Figure 5. Feedback-P3 grand average ERP waveforms from -200ms to 800ms and topography at 400ms in Experiment 1.

Repeated measure ANOVA in SPN

In the current task, we did not find any significant effect of reward magnitudes on the SPN, [$F(1.85, 44.27) = 0.78$, $p = 0.54$, partial $\eta 2 = 0.03$], and COI, [$F(2.07, 49.68) = 1.37$, $p = 0.39$, partial $\eta 2 = 0.04$], and no significant interaction effect between reward magnitudes and COI, [$F(4.26, 102.23) = 0.12$, $p = 0.69$, partial $\eta 2 = 0.02$].

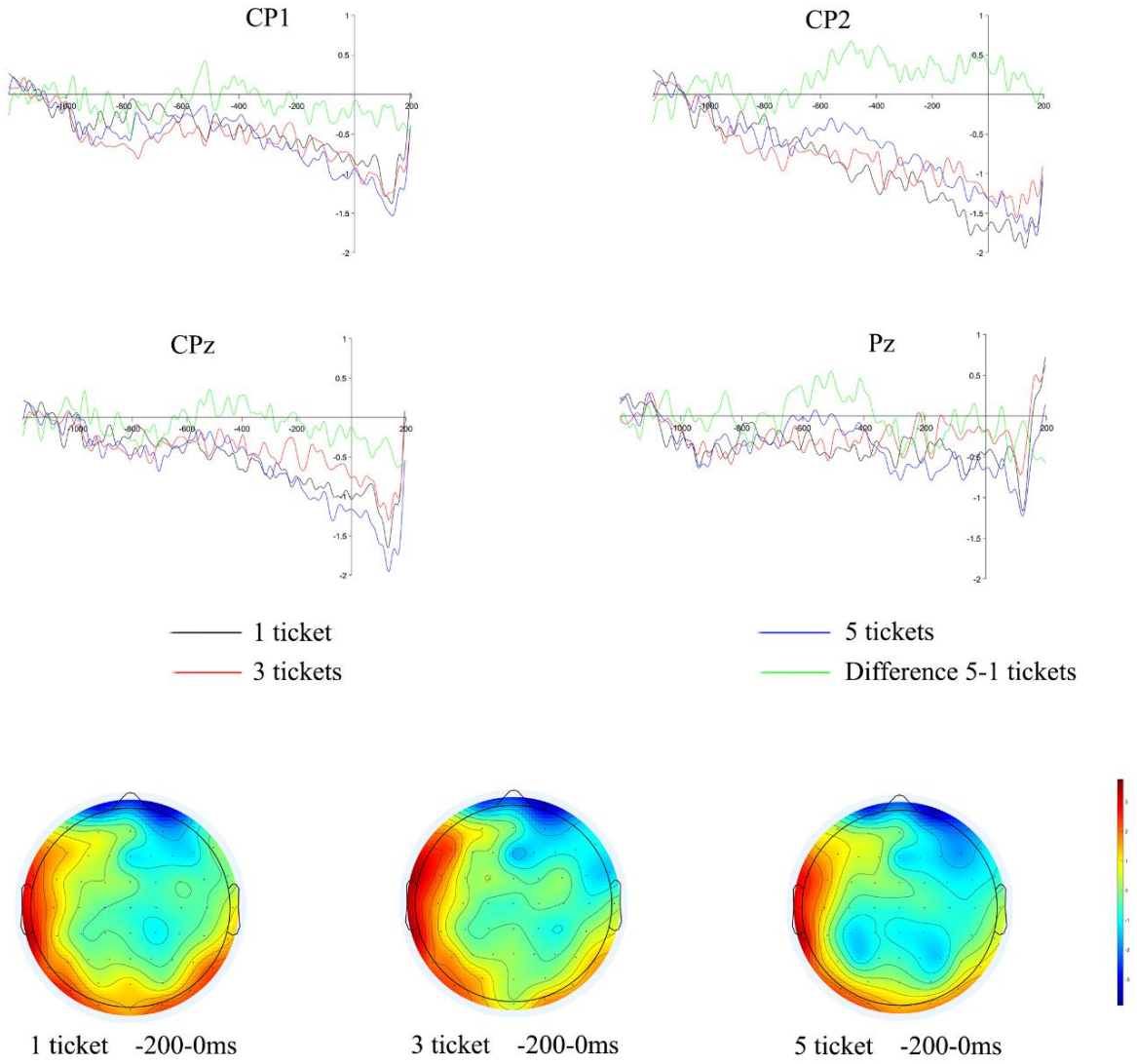

Figure 6. Stimulus-preceding negativity (SPN) grand average ERP waveforms from -1200ms to 200ms and averaged topography from -200ms to 0ms in Experiment 1.

Table 5. *Summary of repeated-measure ANOVA results for the ERPs amplitude and latency to assess effects of magnitudes in Cue-P3 and SPN and the effects of reward valence in Feedback-P3 in Experiment 1*

	Cue-P3						Feedback-P3						SPN		
	Amplitude			Latency			Amplitude			Latency			Amplitude		
	F	*p*	*η2*	*F*	*p*	*η2*	*F*	*p*	*η2*	*F*	*p*	*η2*	*F*	*p*	*η2*
Magnitudes	13.93	< .001	.37	5.15	0.01	0.18							.60	.54	.03
Valence							1.05	.32	.04	.66	.42	.03			
COI	42.40	< .001	.64	8.25	< .001	.26	9.18	< .001	.28	15.30	< .001	.39	.96	.39	.04
Magnitudes * COI	5.09	< .001	.18	2.84	.01	.11							.58	.69	.02
Valence * COI							.41	.80	.02	1.37	.25	.05			

Table 6. *Post-hoc pairwise comparison of Cue-P3 amplitudes and latency in different reward magnitudes conditions in channels with significant interaction effects observed in Experiment 1*

COIs	1 Ticket	3 Tickets	5 Tickets	Overall *p*	Pairwise *p*		
					1 vs. 3	3 vs. 5	1 vs. 5
Amplitudes							
Oz	6.66(2.84)	9.40(4.57)	10.62(6.34)	< .001	**< .001**	0.07	**< .001**
POz	6.05(2.60)	8.17(3.34)	7.92(3.19)	0.001	**< .001**	0.67	0.10
Pz	4.38(2.49)	5.13(3.10)	5.54(3.19)	0.03	0.06	0.40	**0.01**
CP1	2.97(1.72)	3.08(2.02)	3.81(2.09)	0.03	0.71	0.08	**0.01**
Latency							
CPz	303.59(48.42)	334.53(53.54)	335.47(51.33)	0.01	**0.02**	0.93	**< .001**
Cz	305.94(53.32)	337.50(60.74)	350.94(54.17)	0.01	**0.04**	0.29	**0.004**
CP1	314.22(48.86)	333.75(53.00)	337.34(47.30)	0.05	**0.07**	0.76	**0.004**

Exploratory analysis

Correlation between ERP amplitudes and anticipatory and consummatory rating

Exploratory correlation analyses were conducted on ERP amplitudes at the channels with the largest effects and anticipatory and consummatory excitement ratings. There was a small positive correlation between Cue-P3 amplitudes at channel Oz and anticipatory ratings ($r = 0.24$, $p < 0.05$).

Feedback-P3 amplitudes at channel Oz were analyzed with consummatory ratings. We found a small positive correlation in ERP amplitudes in the win condition and consummatory ratings ($r = 0.26$, $p < 0.05$). Again, there is no significant conclusion since we are underpowered, and we will need a larger sample size to determine if the correlation holds true.

No significant correlation was found between SPN amplitudes at channel CP2 and either anticipatory or consummatory ratings.

Correlation between ERP amplitudes and questionnaire scores

Correlation analyses were also conducted on ERP amplitudes at the channels mentioned above and the questionnaire scores. Trends of the correlations are shown below in Figure 7. There was a small correlation between the winning Feedback-P3 amplitudes at channel Oz and the BIS scores ($r = -.442$, $p < 0.05$). No significant correlation was found between the other ERPs and questionnaire scores.

Conclusion

The current study used an adapted MID task to measure electrophysiological responses to anticipatory and consummatory reward stimuli. The behavioural component of the task showed increased anticipatory and consummatory reward sensitivity and faster response times as reward magnitudes increased. In general, excitement ratings were higher during the consumption phase than the anticipation phase, but the difference became smaller as reward magnitudes increased. ERPs following reward cues (i.e., cue-P3), response (i.e., SPN), and feedback (i.e., feedback-P3) were evaluated. During the anticipatory phase, larger reward magnitudes increased P3 amplitudes compared with smaller reward magnitudes, but didn't show effects on the SPN. This demonstrates the motivational salience effects of rewards across the cue-evaluation stage (Sato et al., 2005; Yeung & Sanfey, 2004; Zheng et al., 2017). In the anticipatory phase, high reward magnitudes (3 and 5 lottery tickets) elicited larger P3 in comparison with low reward magnitudes (1 lottery ticket), whereas there was no significant difference between 3 and 5 tickets. Low reward magnitude also had shorter peak latency than high reward magnitudes in the centroparietal regions. In conclusion, the task's results partially confirmed the hypothesized changes, indicating that as reward magnitudes escalated during the anticipatory phase, there were corresponding increases in cue-P3 amplitudes. However, no significant changes were observed in the late anticipatory SPN and consummatory feedback-P3 components.

We observed a negative sink of amplitudes in the 5 tickets condition around 150-170ms after cue onset which was more obvious around the occipital region (channel Oz and POz). We hypothesized that the asymmetric arrangement of 5 tickets on the screen exposed the left visual field to more visual stimuli than the right visual field, whereas 1 and 3 tickets were symmetrically arranged. In feedback evaluation, we also found a similar dampening around 150-

170ms after the non-win feedback stimuli onset. The feedback was visualized using a green thumbs-up as win and a red thumbs-down as non-win. We suspected the effect was caused by an inversion effect of the thumbs-down compared with the thumbs-up which is more commonly observed and familiar in life. Accordingly, in Experiment 2, we attempted to adjust the stimulus and eliminate the early visual effect that could have contaminated the adjacent ERPs such as cue and feedback-P3, and possibly contributed to the lack of effect in Feedback-P3 in the initial paradigm.

Experiment 2

Methods

Modified MID Task Paradigm

Two stimuli were adjusted aiming to eliminate the confounding visual effects, the reward cue stimuli, and the feedback stimuli. In the original paradigm, reward cues for five tickets were asymmetrically presented in the left and right visual fields, resulted in a dampened potentiation in the five-tickets condition prior to P3 onset in comparison to other conditions. In the new paradigm, we adjusted the stimuli to be presented as a single ticket printed with numbers (1, 3, and 5) as indicators of reward magnitudes. Pilot behaviour data from 7 participants indicated good salient effects of these new cue stimuli (p = 0.03). The other stimuli we modified were the feedback stimuli, in which the thumb down had resulted in a possible inversion effect also prior to P3. To adjust, we used a checkmark "√" and a cross "×" mark as the new win and non-win feedback stimuli. The rest of the task remained unchanged (Figure. 8).

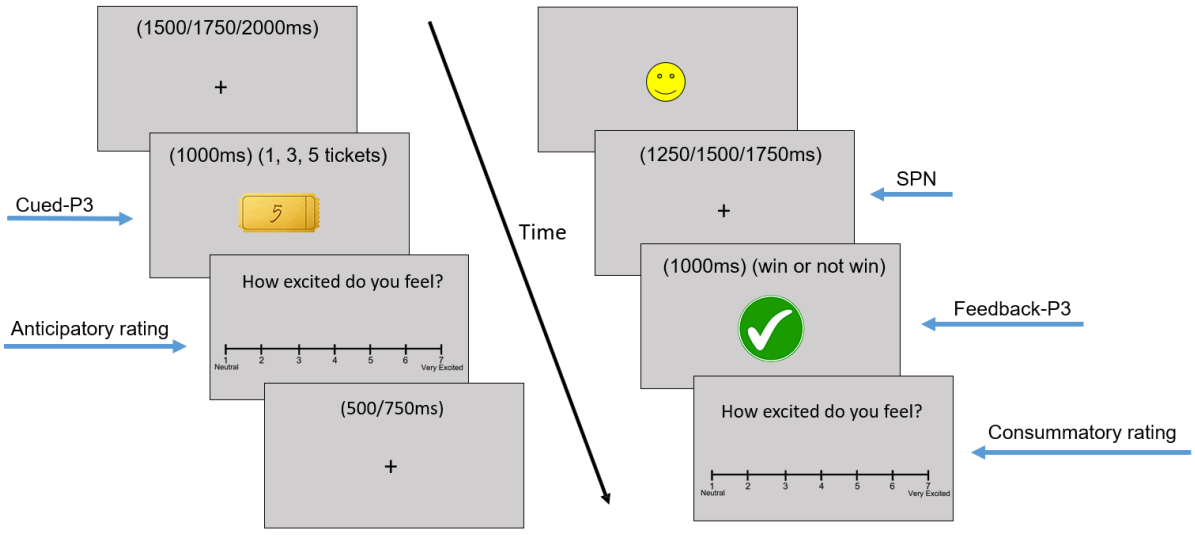

Figure 7. Schematic of the adjusted MID task. Both the cue stimuli and feedback stimuli were changed from the previous paradigm.

Participants

A total of 23 participants (6 males, 2 non-binaries, mean age = 21.30, *SD* = 5.21) were recruited through the human subject pool system from the University of British Columbia and received course credit for their participation. 2 participants were excluded from EEG analysis due to medication history and their EEG data were not analyzed. 1 participant's data was too noisy with less than 20 good trials in at least one condition, leaving 20 participants' EEG data, which had more than 20 trials in all conditions, in the ERP analysis.

Table 7. Participant demographic in Experiment 2

Variables	Mean ± SD
Age	21.30 ± 5.21
% Female	15 (73.91%)
BDI	13.61 ± 14.43
GAD	5.22 ± 5.24
HCL	17.09 ± 4.80
STAI-S	38.17 ± 8.55
STAI-T	44.48 ± 9.01
DARS	56.22 ± 13.17
BIS	12.96 ± 2.85
BAS	7.35 ± 1.61

Table 8. *Mean and standard deviation of the number of trials averaged in each condition in Experiment 2.*

Condition	Mean	SD
Cue one ticket	60.15	17.50

Cue three tickets	58.90	19.37
Cue five tickets	58.80	18.65
Feedback win	72.40	26.52
Feedback non-win	70.15	25.94
SPN one ticket	61.70	18.18
SPN three tickets	59.80	16.80
SPN five tickets	59.10	15.92

Procedure

The new task procedures remained the same as the previous task. Participants read and confirmed consent to participate in the study and the reward lottery draw followed by completing the Demographic and Daily Evaluation questionnaire mentioned above. EEG data were collected while completing the new MID paradigm. Finally, participants completed additional self-reported scales mentioned above. Additional information about participants' assumptions of the task was also collected (i.e., whether they believe the reward results were not related to their reaction time to the target cue).

Results

Behavioural Task

A 2 (Incentive type) x 3 (levels of reward magnitudes) repeated-measure ANOVA showed main effects of reward magnitudes, [$F(1.19, 26.09) = 42.63$, $p < .001$, partial $\eta 2 = .66$], incentive type, [$F(1.00, 22.00) = 46.27$, $p < .001$, partial $\eta 2 = .68$], and an interaction effect of reward magnitudes and incentive type, [$F(1.19, 26.11) = 16.57$, $p < .001$, partial $\eta 2 = .43$]. Post-hoc t-test revealed that anticipatory ratings were significantly lower than consummatory ratings

in all three magnitudes, [$t(22)$=-7.20, p < .001, $t(22)$ = -6.12, p < .001], and [$t(22)$ = -3.90, p < .001] for 1, 3 and 5 tickets respectively. Similar to the previous paradigm, anticipatory excitement ratings for 1, 3, and 5 tickets were all significantly lower than consummatory ratings (p <.001). Response time was also lower when the number of tickets played for increased, [F(2, 44) = 16.71, p < .001, partial η2 = .43]. Although the post-hoc t-test didn't show a significant change in reaction time when playing for 1 ticket (M = 0.39, SD = 0.10) compared to 3 tickets (M = 0.39, SD = 0.10), [$t(22)$ = 0.92, p = 0.18], there was a significant decrease when playing for 3 tickets and 5 tickets (M = 0.37, SD = 0.10), [$t(22)$ = 4.68, p < .001], and for 1 ticket and 5 tickets [t(22) = 4.68, p < .001].

Table 9. *Repeated-measure ANOVA results of the behvaioural ratings to assess the effects of reward magnitudes and types of ratings in Experiment 2.*

Effects	Sum of Square	*df*	Mean square	*F*	*p*	Partial η2
Magnitudes	53.41	1.19	45.04	42.63	< .001	.66
Error(Magnitudes)	27.56	26.09	1.06			
IncentiveType	40.62	1.00	40.62	46.27	< .001	.68
Error(IncentiveType)	19.31	22.00	.88			
Magnitudes * IncentiveType	5.22	1.19	4.40	16.57	< .001	.43
Error(Magnitudes* IncentiveType)	6.93	26.11	.27			

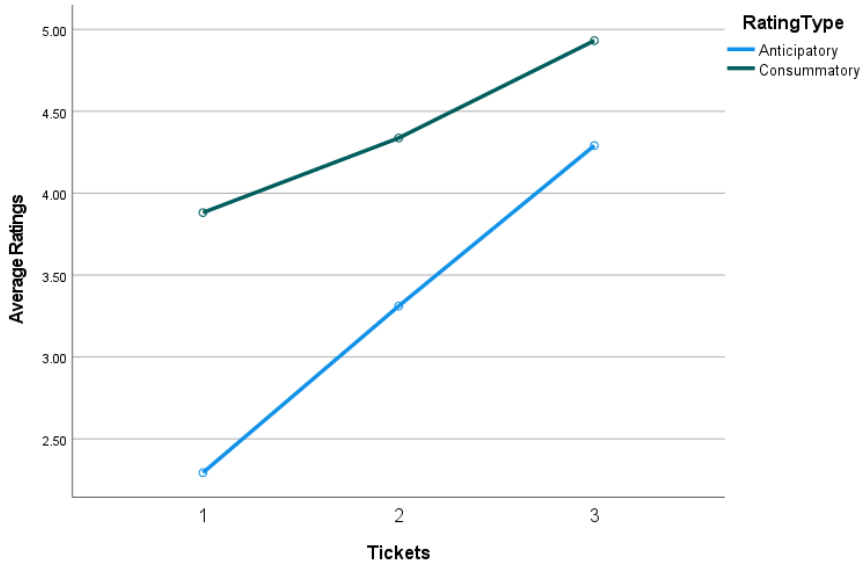

Figure 8. Averaged anticipatory and consummatory ratings in three reward magnitudes levels in Experiment 2.

Table 10. *Repeated-measure ANOVA results of reaction time to targets to assess effects of reward magnitudes in Experiment 2.*

Effects	Sum of Square	*df*	Mean square	*F*	*p*	Partial η2
Tickets	.01	2	.00	16.71	< .001	.43
Error(tickets)	.01	44	.00			

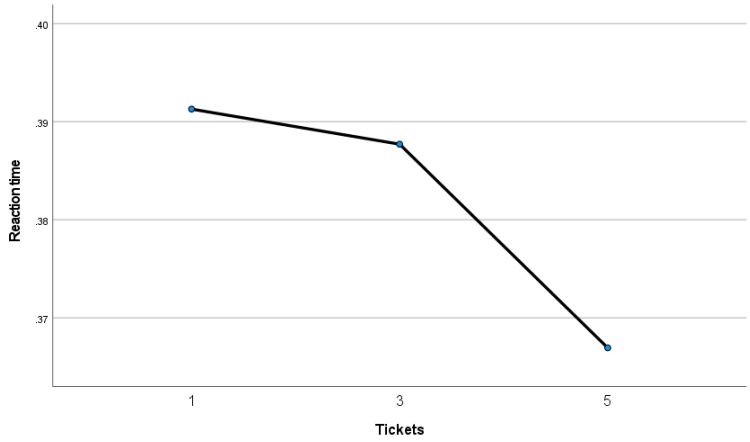

Figure 9. Averaged reaction time to targets in three reward magnitudes levels in Experiment 2.

EEG

Repeated measure ANOVA for the Cue-P3

We conducted the same test for ERP results as the previous paradigm. Repeated measure ANOVA showed main effects on COI, [$F(1.76, 33.47) = 27.46$, $p < .001$, partial $\eta 2 = .59$]**,** and a significant interaction between reward magnitudes and COI**,** [$F(4.58, 87.05) = 4.60$, $p = .001$, partial $\eta 2 = .19$]. Additional ANOVA on reward magnitudes and COIs revealed a significant difference in peak amplitudes at channel Oz, [$F(2, 38) = 15.22$, $p < 0.001$, partial $\eta 2 = 0.45$], where Cue-P3 amplitudes were significantly smaller in 1 ticket condition than 3 tickets ($p < .001$) and 5 tickets ($p < .001$); and at channel POz, [$F(2, 38) = 3.70$, $p < 0.05$, partial $\eta 2 = 0.16$], where amplitudes in 1 ticket condition were significantly smaller than 5 tickets condition ($p < 0.05$). The main effect for overall reward magnitude, however, was not significant, [$F(1.76, 33.37) = 1.37$, $p = 0.27$, partial $\eta 2 = 0.07$]. Peak latency analysis only revealed a main effect in COI, [$F(3.23, 61.30) = 4.53$, $p = .01$, partial $\eta 2 = .19$].

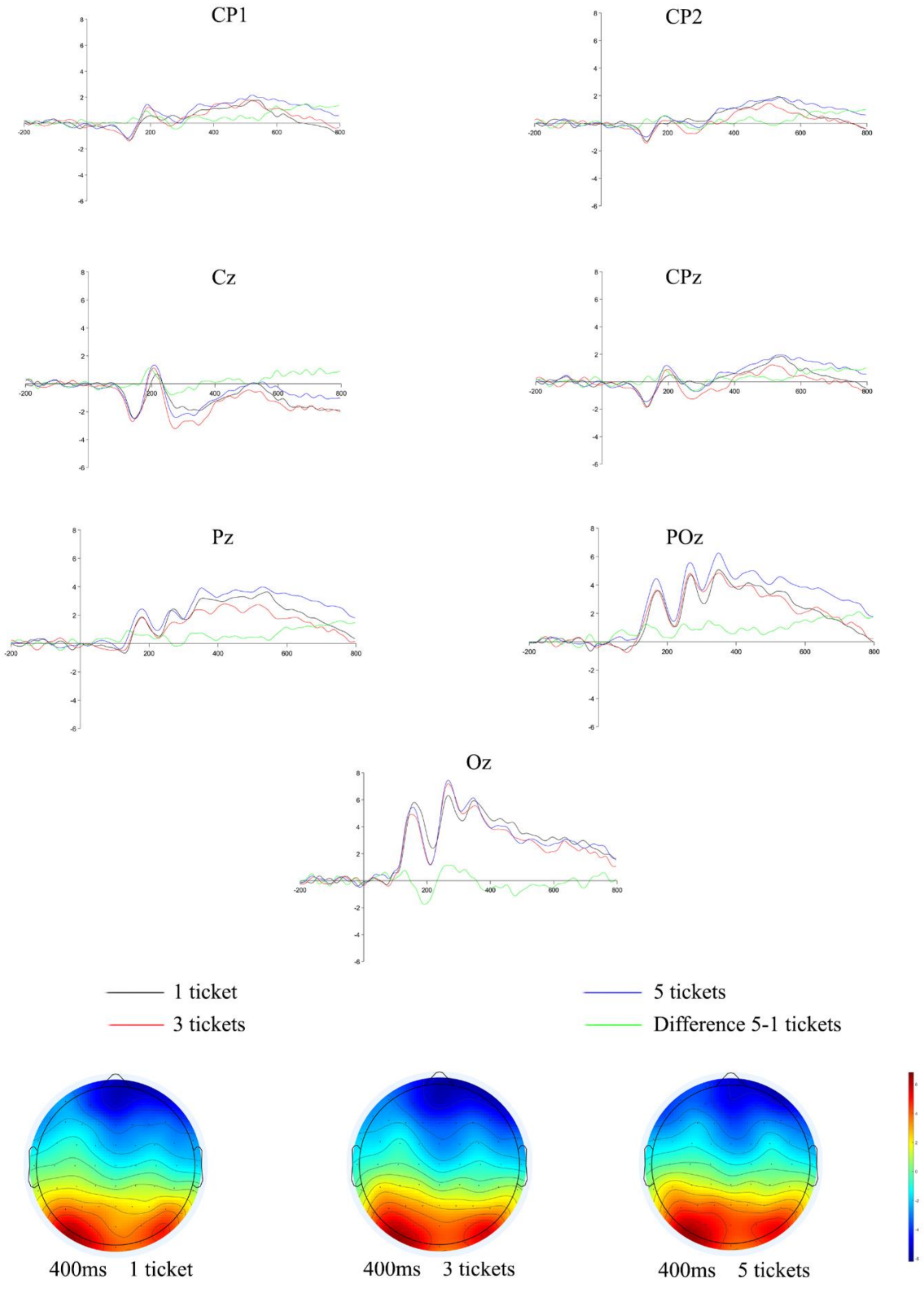

Figure 10. Cue-P3 grand average ERP waveforms from -200ms to 800ms and topography at 400ms in Experiment 2.

Repeated measure ANOVA in Feedback-P3

Results for feedback-P3 showed a significant main effect for COI, [$F(1.69, 32.11) = 12.13$, $p < .001$, partial $\eta 2 = .39$]. We did not find any significant effects for reward valence, [$F(1, 19.00) = 0.00$, $p = 0.99$, partial $\eta 2 = .00$], nor reward valence and COI interaction, [$F(2.81, 53.40) = 1.24$, $p = 0.32$, partial $\eta 2 = .06$]. Latency analysis only showed a main effect for COI as well, [$F(3.30, 62.72) = 11.45$, $p < .001$, partial $\eta 2 = .38$].

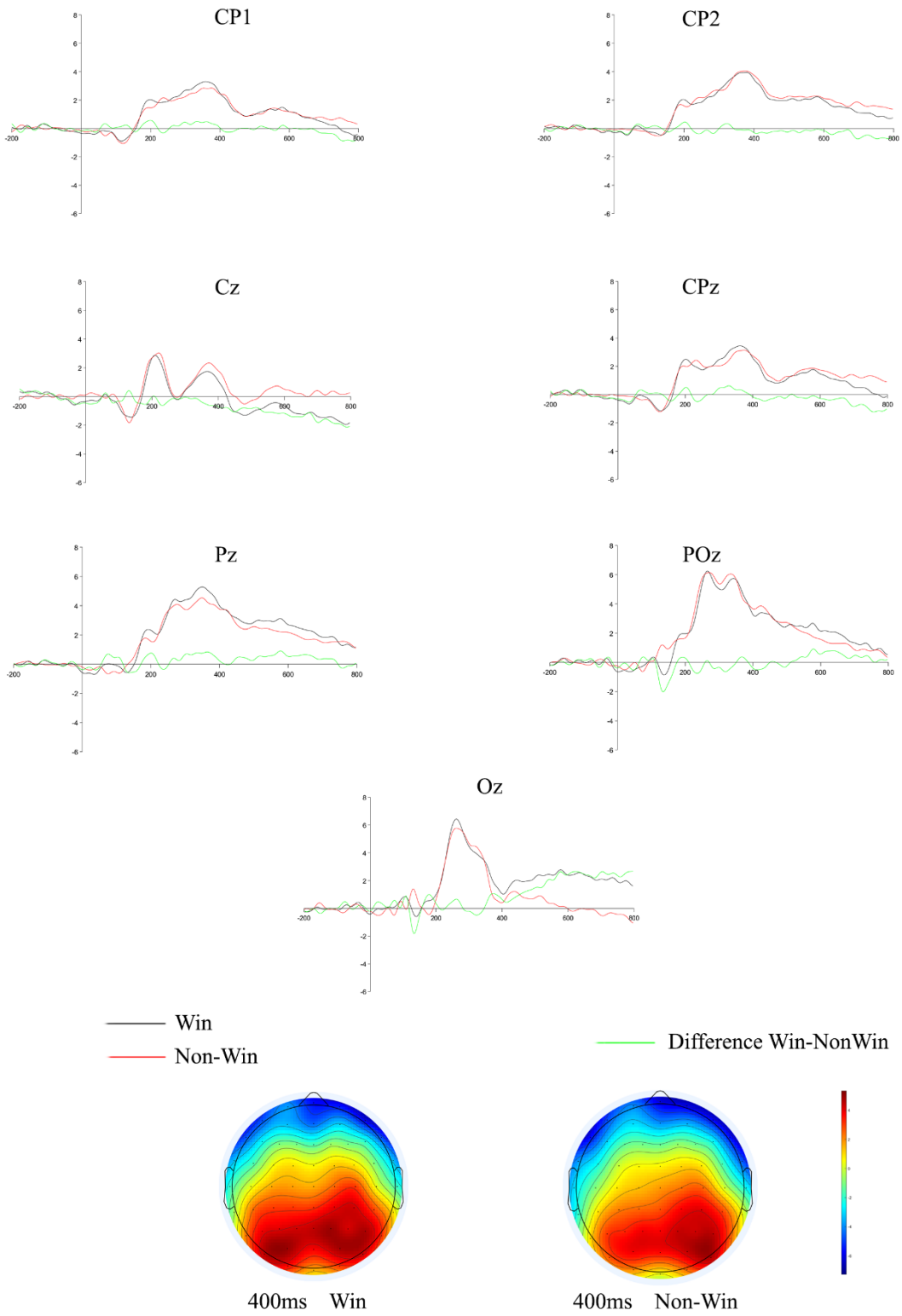

Figure 11. Feedback-P3 grand average ERP waveforms from -200ms to 800ms and topography at 400ms in Experiment 2.

Repeated measure ANOVA in SPN

In the current task, we did not find any significant effect of reward magnitudes on the SPN, [$F(1.67, 31.69) = 0.25$, $p = 0.78$, partial $\eta 2 = 0.01$], and COI, [$F(2.90, 55.01) = 2.22$, $p = 0.10$, partial $\eta 2 = 0.10$], and no significant interaction effect between reward magnitudes and COI, [$F(4.19, 79.55) = 2.14$, $p = 0.08$, partial $\eta 2 = 0.10$].

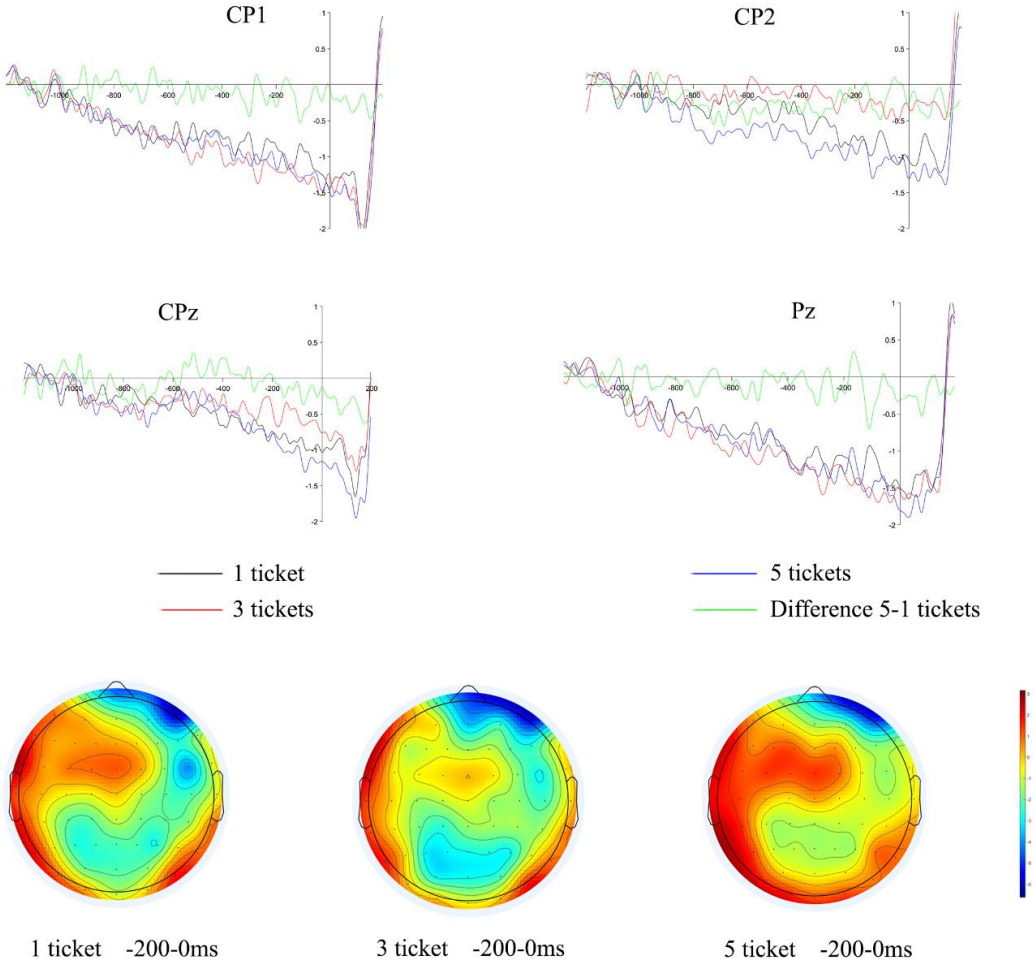

Figure 12. Stimulus-preceding negativity (SPN) grand average ERP waveforms from -1200ms to 200ms and averaged topography from -200ms to 0ms in Experiment 2.

Table 11. *Summary of repeated-measure ANOVA results for the ERPs amplitude and latency to assess effects of magnitudes in Cue-P3 and SPN and the effects of reward valence in Feedback-P3 in Experiment 2.*

	Cue-P3						Feedback-P3						SPN		
	Amplitude			Latency			Amplitude			Latency			Amplitude		
	F	*p*	*η2*	*F*	*p*	*η2*	*F*	*p*	*η2*	*F*	*p*	*η2*	*F*	*p*	*η2*
Magnitudes	1.37	0.27	0.07	1.55	0.22	0.08							.25	.78	.01
IncentiveType							0.00	0.99	.00	0.78	.39	.04			
COI	27.46	<.001	0.59	4.53	< .001	.19	12.13	< .001	.39	11.45	< .001	.38	2.22	.10	.10
Magnitudes * COI	4.60	0.001	0.19	1.75	.12	.08							2.14	.08	.10
IncentiveType * COI							1.24	.32	.06	0.83	.55	.04			

Table 12. *Post-hoc pairwise comparison of Cue-P3 amplitudes in different reward magnitudes conditions in channels with significant interaction effects observed in Experiment 2*

COIs	**1 Ticket**	**3 Tickets**	**5 Tickets**	**Overall *p***	**Pairwise *p***		
					1 vs. 3	**3 vs. 5**	**1 vs. 5**
Oz	6.81(4.39)	8.96(4.84)	8.82(4.50)	< .001	**< .001**	0.75	**< .001**
POz	6.37(4.16)	7.30(3.95)	7.49(4.10)	0.03	0.06	0.60	**0.03**

In the second paradigm, we also wanted to explore whether participants were aware, or became aware of the experimental manipulation, that is the outcomes of task were independent of their actions and efforts, therefore, we included the following question at the end of the experiment questionnaire asking, “How likely did you think you would win the tickets if you pressed the spacebar fast enough?”. Among 23 participants, 6 (26.09%) responded “Likely”, 6 (26.09%) responded “Not likely” and 11 (47.83%) responded “Maybe”.

Conclusion

From the grand average ERPs waveforms, we did not observe any significant differences between conditions at around 150-170ms at the occipital regions, therefore, we concluded that the revised paradigm had showed cleaner ERPs compared with the previous paradigm after we had removed the asymmetric and inverted stimuli. We still saw behavioural differences in anticipatory and consummatory excitement ratings, in that ratings increased with reward magnitudes, and similarly to the previous paradigm, differences between anticipatory and consummatory ratings became smaller as reward magnitudes went up. Higher reward magnitudes also caused quicker motor response to the targets which showed that our new paradigm still created different salient effects even though the stimuli were not dramatically different from each other. We observed a significant enhancement in Cue-P3 amplitudes at the occipital regions, Oz and POz, as reward magnitudes increase. However, we did not see a significant difference in the ERP amplitudes and latencies in the new paradigm which could also be because we had less power compared to the previous paradigm.

Discussion

In this study, we aimed to validate an adapted MID paradigm that included monetary gain and no gain in different reward magnitudes to study the temporal dynamics of anticipatory and consummatory reward processing by extracting reward-related ERPs, including Cue-P3 and SPN at the early and late anticipatory stage, and Feedback-P3 at the reward consumption stage. This paradigm was designed to specifically eliminate decision-making and reinforcement learning which are two common psychological constructs that are included in other tasks that are used to investigate reward processing, and the adapted MID paradigm is able to more precisely capture ERP responses responsible for reward anticipation and consumption and disentangle the temporal overlaps of various reward-related ERPs. In the initial experiment, we found that the task elicited enhanced Cue-P3 as reward magnitudes increased, demonstrating the motivational salience effects of rewards and reward magnitudes across the cue-evaluation stage (Sato et al., 2005; Yeung & Sanfey, 2004; Zheng et al., 2017). We did not find a significant effect of reward valences (i.e., win vs. non-win) which supports previous findings that Feedback-P3 is not modulated by reward valence. As mentioned in the introduction, the results of Feedback-P3 and its sensitivity to performance evaluation have been mixed. The high temporal overlap between P3 and another feedback-related ERP, reward positivity (RewP), may have contributed to the inconsistency in the literature. It's been shown that RewP is most commonly investigated within the reinforcement learning paradigm, and that RewP is associated with prediction violation and is considered an index for reward prediction error (Glazer et al., 2018; Sambrook & Goslin, 2014). Since our paradigm did not incorporate reward learning and prediction, the Feedback-P3 results were temporally distinct from other ERP components, and we concluded that Feedback-P3 was not sensitive to reward valences of win and non-win conditions. We also observed that our P3

had a tendency to shift back close to occipital instead of centroparietal where P3 is most commonly observed. This could be due to human error during experiment setup or because both our reward cues and reward feedback stimuli are visually salient, therefore we observed heightened neural response carried to later P3 component. The task had elevated negativity at late-stage anticipation before feedback onset but there was no effect of reward magnitudes in SPN as previous literature has suggested (Kotani et al., 2003; Zheng et al., 2015, 2017). Previous research also noted that enhanced SPN is associated with higher uncertainties in outcomes (Catena et al., 2012; Megías et al., 2018) which is more commonly manipulated as risk levels in decision-making or probabilistic tasks, which was lacking in our paradigm. With purely reward evaluation without fluctuation in reward probabilities to create levels of uncertainties, there might not be enough sensitivity to evoke SPN differences in low and high reward magnitudes.

In the revised paradigm (Experiment 2), we identified and substituted two visual stimuli that had caused incongruent early visual processing between conditions in the first experiment. The 5 tickets cue stimulus was asymmetrically organized and resulted in a negative potential at early visual processing. Our non-win feedback stimulus (i.e., thumb-down) also showed an inversion processing effect that caused enhanced negativity around N170 which is an ERP best known for face recognition and the face inversion effects. We used a different set of stimuli that contained the same visual incentive information and conducted the experiment with a different group of participants. From the grand average waveforms, we confirmed our suspicions that the early 150ms – 170ms differences between conditions were purely caused by early visual processing of the visual stimuli, and not related to the processing of reward information. For future experiments, we need to pay more attention to the paradigm and stimuli design and ensure that undesired effects are not evoked which could contaminate ERP results.

One of the limitations of the study is the salience of our reward. We used chances to win lottery tickets towards monetary gain instead of direct monetary rewards. Even though we observed sensitivity from behavioural responses of anticipatory and consummatory ratings, ERPs might not be most sensitive to the current reward condition. The task was also manipulated by researchers so that we acquired the same number of trials in win and non-win in each reward magnitude, and participants were blinded to this setup. However, by the end of the task, a number of participants had suspected that their response to the target would not influence task outcomes. From the results of the questionnaire conducted at the end of the second experiment, about half of the participants (47.83%) speculated that their actions did not influence experiment outcome, and some (26.09%) believed outcomes were not affected at all. It's reasonable to suspect that motivation would decrease as participants had come to terms that their actions did not cause changes in outcomes. However, a number of fMRI studies on passive reward expectancy and outcome processing showed activation at the ventral striatum and medial orbitofrontal cortex (Chumbley et al., 2014; Diekhof et al., 2012; Van Leijenhorst et al., 2010). It's reasonable to assume anticipation and consumption reward processing would evoke neural responses just by the visual presentation of salient stimuli.

In both experiments, we had relatively small sample sizes which precluded an adequately powered correlation analysis between the self-reported scales, behavioural ratings, and ERP amplitudes, and possibly contributed to low power in the main analysis of Experiment 2. We also recruited from a college population of young adults rather than a clinical setting, so the majority of the sample had low to medium symptom levels and only a few had high symptom levels. In future studies, a greater sample size, and greater variance in depression/anxiety symptoms should

be recruited to better investigate symptoms of anhedonia in relation to anticipatory and consummatory reward processing.

In summary, we assessed the use of an altered MID paradigm as a means to measure electrophysiological responses to anticipatory and consummatory reward processing. The paradigms demonstrated the ability to evaluate early reward anticipation in different reward magnitudes as reflected by Cue-P3, but did not have an effect on late anticipation (SPN) and feedback evaluation (Feedback-P3). Since the paradigm omitted reward learning and decision-making, which are psychological constructs outside of reward anticipation and consumption, it allows a refined examination of the reward-related ERP components and the effects of reward magnitudes and valences. This makes our task a potentially useful paradigm to investigate reward-related symptoms such as subtypes of anhedonia, especially deficits in early reward anticipation, that are observed in various clinical disorders. Given the dearth of research on reward anticipation in clinical populations, this paradigm could be used in studies as an index to early anticipation toward monetary reward, and future studies could use it to investigate reward processing-related symptoms and their neurophysiological markers in patients with mental health disorders.

References

Admon, R., & Pizzagalli, D. A. (2015). Dysfunctional reward processing in depression. In Current Opinion in Psychology (Vol. 4, pp. 114–118). Elsevier. https://doi.org/10.1016/j.copsyc.2014.12.011

Alloy, L. B., Olino, T., Freed, R. D., & Nusslock, R. (2016). Role of Reward Sensitivity and Processing in Major Depressive and Bipolar Spectrum Disorders. www.elsevier.com/locate/bt

American Psychiatric Association. (2013). Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. American Psychiatric Association Publishing.

Angst, J., Adolfsson, R., Benazzi, F., Gamma, A., Hantouche, E., Meyer, T. D., Skeppar, P., Vieta, E., & Scott, J. (2005). The HCL-32: Towards a self-assessment tool for hypomanic symptoms in outpatients. Journal of Affective Disorders, 88(2), 217–233. https://doi.org/10.1016/j.jad.2005.05.011

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1960). An Inventory for Measuring Depression The difficulties inherent in obtaining. https://jamanetwork.com/

Berridge, K. C., Robinson, T. E., & Aldridge, J. W. (2009). Dissecting components of reward: "liking", "wanting", and learning. In Current Opinion in Pharmacology (Vol. 9, Issue 1, pp. 65–73). https://doi.org/10.1016/j.coph.2008.12.014

Bigdely-Shamlo, N., Mullen, T., Kothe, C., Su, K.-M., & Robbins, K. A. (2015). The PREP pipeline: standardized preprocessing for large-scale EEG analysis. Frontiers in Neuroinformatics | Www.Frontiersin.Org, 1, 16. https://doi.org/10.3389/fninf.2015.00016

Borsini, A., Wallis, A. S. J., Zunszain, P., Pariante, C. M., & Kempton, M. J. (2020). Characterizing anhedonia: A systematic review of neuroimaging across the subtypes of reward processing deficits in depression. In Cognitive, Affective and Behavioral Neuroscience (Vol. 20, Issue 4, pp. 816–841). Springer. https://doi.org/10.3758/s13415-020-00804-6

Burkhouse, K. L., Gorka, S. M., Klumpp, H., Kennedy, A. E., Karich, S., Francis, J., Ajilore, O., Craske, M. G., Langenecker, S. A., Shankman, S. A., Hajcak, G., & Luan Phan, K. (2018). Neural responsiveness to reward as an index of depressive symptom change following cognitive-behavioral therapy and SSRI treatment. Journal of Clinical Psychiatry, 79(4). https://doi.org/10.4088/JCP.17m11836

Calabro, F. J., Montez, D. F., Larsen, B., Laymon, C. M., Foran, W., Hallquist, M. N., Price, J. C., & Luna, B. (2023). Striatal dopamine supports reward expectation and learning: A simultaneous PET/fMRI study. NeuroImage, 267. https://doi.org/10.1016/j.neuroimage.2022.119831

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS Scales. Journal of Personality and Social Psychology, 67(2), 319–333.

Catena, A., Perales, J. C., Megías, A., Cándido, A., Jara, E., & Maldonado, A. (2012). The brain network of expectancy and uncertainty processing. PLoS ONE, 7(7). https://doi.org/10.1371/journal.pone.0040252

Chumbley, J. R., Tobler, P. N., & Fehr, E. (2014). Fatal attraction: Ventral striatum predicts costly choice errors in humans. NeuroImage, 89, 1–9. https://doi.org/10.1016/j.neuroimage.2013.11.039

Davis, C., & Woodside, D. B. (2002). Sensitivity to the rewarding effects of food and exercise in the eating disorders. Comprehensive Psychiatry, 43(3), 189–194. https://doi.org/10.1053/comp.2002.32356

Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. Journal of Neuroscience Methods, 134(1), 9–21. https://doi.org/10.1016/j.jneumeth.2003.10.009

Der-Avakian, A., Barnes, S. A., Markou, A., & Pizzagalli, D. A. (2016). Translational assessment of reward and motivational deficits in psychiatric disorders. In Current Topics in Behavioral Neurosciences (Vol. 28, pp. 231–262). Springer Verlag. https://doi.org/10.1007/7854_2015_5004

Diekhof, E. K., Kaps, L., Falkai, P., & Gruber, O. (2012). The role of the human ventral striatum and the medial orbitofrontal cortex in the representation of reward magnitude - An activation likelihood estimation meta-analysis of neuroimaging studies of passive reward expectancy and outcome processing. Neuropsychologia, 50(7), 1252–1266. https://doi.org/10.1016/j.neuropsychologia.2012.02.007

Dillon, D. G., Holmes, A. J., Jahn, A. L., Bogdan, R., Wald, L. L., & Pizzagalli, D. A. (2007). Dissociation of neural regions associated with anticipatory versus consummatory phases of incentive processing. https://doi.org/10.1111/j.1469-8986.2007.00594.x

Eshel, N., & Roiser, J. P. (2010). Reward and punishment processing in depression. In Biological Psychiatry (Vol. 68, Issue 2, pp. 118–124). Elsevier USA. https://doi.org/10.1016/j.biopsych.2010.01.027

Foti, D., Weinberg, A., Dien, J., & Hajcak, G. (2011). Event-related potential activity in the basal ganglia differentiates rewards from nonrewards: Temporospatial principal components analysis and source localization of the feedback negativity. Human Brain Mapping, 32(12), 2207–2216. https://doi.org/10.1002/hbm.21182

Gard, D. E., Gard, M. G., Kring, A. M., & John, O. P. (2006). Anticipatory and consummatory components of the experience of pleasure: A scale development study. Journal of Research in Personality, 40(6), 1086–1102. https://doi.org/10.1016/j.jrp.2005.11.001

Gard, D. E., Kring, A. M., Gard, M. G., Horan, W. P., & Green, M. F. (2007). Anhedonia in schizophrenia: Distinctions between anticipatory and consummatory pleasure. Schizophrenia Research, 93(1–3), 253–260. https://doi.org/10.1016/j.schres.2007.03.008

Glazer, J. E., Kelley, N. J., Pornpattananangkul, N., Mittal, V. A., & Nusslock, R. (2018). Beyond the FRN: Broadening the time-course of EEG and ERP components implicated in reward processing. International Journal of Psychophysiology, 132, 184–202. https://doi.org/10.1016/j.ijpsycho.2018.02.002

Guyer, A. E., Nelson, B., Li, Y., Oumeziane, B. A., Jones, O., & Foti, D. (2019). Neural Sensitivity to Social and Monetary Reward in Depression: Clarifying General and Domain-

Specific Deficits. Frontiers in Behavioral Neuroscience, 13. https://doi.org/10.3389/fnbeh.2019.00199

Hackley, S. A., Valle-Inclán, F., Masaki, H., & Hebert, K. (2013). Stimulus-Preceding Negativity (SPN) and Attention to Rewards. In Cognitive Electrophysiology of Attention: Signals of the Mind (pp. 216–225). Elsevier Inc. https://doi.org/10.1016/B978-0-12-398451-7.00017-8

Hajcak, G., Holroyd, C. B., Moser, J. S., & Simons, R. F. (2005). Brain potentials associated with expected and unexpected good and bad outcomes. Psychophysiology, 42(2), 161–170. https://doi.org/10.1111/j.1469-8986.2005.00278.x

Hajcak, G., Moser, J. S., Holroyd, C. B., & Simons, R. F. (2007). It's worse than you thought: The feedback negativity and violations of reward prediction in gambling tasks. Psychophysiology, 44(6), 905–912. https://doi.org/10.1111/j.1469-8986.2007.00567.x

Hughes, G., Mathan, S., & Yeung, N. (2013). EEG indices of reward motivation and target detectability in a rapid visual detection task. NeuroImage, 64(1), 590–600. https://doi.org/10.1016/j.neuroimage.2012.09.003

Johnson, S. L., Turner, R. J., & Iwata, N. (2003). BIS/BAS levels and psychiatric disorder: An epidemiological study. Journal of Psychopathology and Behavioral Assessment, 25(1), 25–36. https://doi.org/10.1023/A:1022247919288

Julian, L. J. (2011). Measures of anxiety: State-Trait Anxiety Inventory (STAI), Beck Anxiety Inventory (BAI), and Hospital Anxiety and Depression Scale-Anxiety (HADS-A). Arthritis Care and Research, 63(SUPPL. 11). https://doi.org/10.1002/acr.20561

Kelly, C. A., Freeman, K. B., & Schumacher, J. A. (2022). Treatment-resistant depression with anhedonia: Integrating clinical and preclinical approaches to investigate distinct phenotypes. In Neuroscience and Biobehavioral Reviews (Vol. 136). Elsevier Ltd. https://doi.org/10.1016/j.neubiorev.2022.104578

Klein, D. F. (1984). Depression and Anhedonia. In Clark C & Fawcett J (Eds.), In Anhedonia and affect deficit states (pp. 1–14). PMA.

Knutson, B., Adams, C. M., Fong, G. W., Hommer, D., Varner, J., Kerich, M., Lionetti, T., & Walker, J. (2001). Anticipation of Increasing Monetary Reward Selectively Recruits Nucleus Accumbens. In The Journal of Neuroscience (Vol. 21). www.jneurosci.org.http://www.jneurosci.org/cgi/content/full/5472

Knutson, B., Fong, G. W., Adams, C. M., Varner, J. L., & Hommer, D. (2001). Dissociation of reward anticipation and outcome with event-related fMRI. Brain Imaging, 12(17), 3683–3687.

Knutson, B., Westdorp, A., Kaiser, E., & Hommer, D. (2000). FMRI visualization of brain activity during a monetary incentive delay task. NeuroImage, 12(1), 20–27. https://doi.org/10.1006/nimg.2000.0593

Kotani, Y., Kishida, S., Hiraku, S., Suda, K., Ishii, M., & Aihara, Y. (2003). Effects of information and reward on stimulus-preceding negativity prior to feedback stimuli. Psychophysiology, 40(5), 818–826. https://doi.org/10.1111/1469-8986.00082

Lopez-Calderon, J., & Luck, S. J. (2014). ERPLAB: An open-source toolbox for the analysis of event-related potentials. Frontiers in Human Neuroscience, 8(1 APR). https://doi.org/10.3389/fnhum.2014.00213

Luby, J. L., Mrakotsky, C., Heffelfinger, A., Brown, K., & Edward Spitznagel, B. (2004). Article Characteristics of Depressed Preschoolers With and Without Anhedonia: Evidence for a Melancholic Depressive Subtype in Young Children. In Am J Psychiatry (Vol. 161). http://ajp.psychiatryonline.org

Markou, A., Kosten, T. R., & Koob, G. F. (1998). Neurobiological Similarities in Depression and Drug Dependence: A Self-Medication Hypothesis. In Neuropsychopharmacology (Vol. 18, Issue 3).

Mathalon, D. (2009). Neurophysiological distinction between schizophrenia and schizoaffective disorder. Frontiers in Human Neuroscience. https://doi.org/10.3389/neuro.09.070.2009

Mattox, S. T., Valle-Inclán, F., & Hackley, S. A. (2006). Psychophysiological evidence for impaired reward anticipation in Parkinson's disease. Clinical Neurophysiology, 117(10), 2144–2153. https://doi.org/10.1016/j.clinph.2006.05.026

McCabe, C., Mishor, Z., Cowen, P. J., & Harmer, C. J. (2010). Diminished Neural Processing of Aversive and Rewarding Stimuli During Selective Serotonin Reuptake Inhibitor Treatment. Biological Psychiatry, 67(5), 439–445. https://doi.org/10.1016/j.biopsych.2009.11.001

Meadows, C. C., Gable, P. A., Lohse, K. R., & Miller, M. W. (2016). The effects of reward magnitude on reward processing: An averaged and single trial event-related potential study. Biological Psychology, 118, 154–160. https://doi.org/10.1016/j.biopsycho.2016.06.002

Meehl, P. E. (1962). Schizotaxia, schizotypy, schizophrenia. American Psychologist, 17, 827–838. https://doi.org/10.1037/h0041029

Megías, A., Navas, J. F., Perandrés-Gómez, A., Maldonado, A., Catena, A., & Perales, J. C. (2018). Electroencephalographic Evidence of Abnormal Anticipatory Uncertainty Processing in Gambling Disorder Patients. Journal of Gambling Studies, 34(2), 321–338. https://doi.org/10.1007/s10899-017-9693-3

Mei, S., Li, Q., Liu, X., & Zheng, Y. (2018). Monetary Incentives Modulate Feedback-related Brain Activity. Scientific Reports, 8(1). https://doi.org/10.1038/s41598-018-30294-z

Meyer, G. M., Marco-Pallarés, J., Sescousse, G., & Boulinguez, P. (2021). Electrophysiological underpinnings of reward processing: Are we exploiting the full potential of EEG? NeuroImage, 242. https://doi.org/10.1016/j.neuroimage.2021.118478

Ohgami, Y., Kotani, Y., Tsukamoto, T., Omura, K., Inoue, Y., Aihara, Y., & Nakayama, M. (2006). Effects of monetary reward and punishment on stimulus-preceding negativity. Psychophysiology, 43(3), 227–236. https://doi.org/10.1111/j.1469-8986.2006.00396.x

Oldham, S., Murawski, C., Fornito, A., Youssef, G., Yücel, M., & Lorenzetti, V. (2018). The anticipation and outcome phases of reward and loss processing: A neuroimaging meta-analysis of the monetary incentive delay task. Human Brain Mapping, 39(8), 3398–3418. https://doi.org/10.1002/hbm.24184

Pfabigan, D. M., Seidel, E. M., Sladky, R., Hahn, A., Paul, K., Grahl, A., Küblböck, M., Kraus, C., Hummer, A., Kranz, G. S., Windischberger, C., Lanzenberger, R., & Lamm, C. (2014). P300 amplitude variation is related to ventral striatum BOLD response during gain and loss anticipation: An EEG and fMRI experiment. NeuroImage, 96, 12–21. https://doi.org/10.1016/j.neuroimage.2014.03.077

Richter, P., Werner, J., Heerlein, A., Kraus, A., & Sauer, H. (1998). On the Validity of the Beck Depression Inventory A Review. Psychopathology, 4(31), 160–168. www.karger.comhttp://BioMedNet.com/karger

Rizvi, S. J., Pizzagalli, D. A., Sproule, B. A., & Kennedy, S. H. (2016). Assessing anhedonia in depression: Potentials and pitfalls. In Neuroscience and Biobehavioral Reviews (Vol. 65, pp. 21–35). Elsevier Ltd. https://doi.org/10.1016/j.neubiorev.2016.03.004

Rizvi, S. J., Quilty, L. C., Sproule, B. A., Cyriac, A., Michael Bagby, R., & Kennedy, S. H. (2015). Development and validation of the Dimensional Anhedonia Rating Scale (DARS) in a community sample and individuals with major depression. Psychiatry Research, 229(1–2), 109–119. https://doi.org/10.1016/j.psychres.2015.07.062

Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: an incentive-sensitization theory of addiction.

Rugg, M. D. (2001). Event-related/Evoked Potentials. International Encyclopedia of the Social & Behavioral Sciences, 4962–4966. https://doi.org/10.1016/B0-08-043076-7/03410-0

Salisbury, D. F., Rutherford, B., Shenton, M. E., & Mccarley, R. W. (2001). Button-pressing affects P300 amplitude and scalp topography. Clinical Neurophysiology, 112, 1676–1684. www.elsevier.com/locate/clinph

Sambrook, T. D., & Goslin, J. (2014). Mediofrontal event-related potentials in response to positive, negative and unsigned prediction errors. Neuropsychologia, 61(1), 1–10. https://doi.org/10.1016/j.neuropsychologia.2014.06.004

Sambrook, T. D., & Goslin, J. (2015). A Neural reward prediction error revealed by a meta-analysis of ERPs using great grand averages. Psychological Bulletin, 141(1), 213–235. https://doi.org/10.1037/bul0000006

San Martín, R. (2012). Event-related potential studies of outcome processing and feedback-guided learning. Frontiers in Human Neuroscience, OCTOBER 2012, 1–40. https://doi.org/10.3389/fnhum.2012.00304

Sato, A., Yasuda, A., Ohira, H., Miyawaki, K., Nishikawa, M., Kumano, H., & Kuboki, T. (2005). Effect of value and reward magnitude on feedback negativity and P300. Cognitive Neuroscience and Neuropsychology, 16(4), 407–411. http://journals.lww.com/neuroreport

Sherdell, L., Waugh, C. E., & Gotlib, I. H. (2012). Anticipatory pleasure predicts motivation for reward in major depression. Journal of Abnormal Psychology, 121(1), 51–60. https://doi.org/10.1037/a0024945

Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., & Jacobs, G. A. (1983). Manual for the State-Train Anxiety Inventory .

Spitzer, R. L., Kroenke, K., Williams, J. B. W., & Löwe, B. (2006). A Brief Measure for Assessing Generalized Anxiety Disorder The GAD-7. Arch Intern Med, 166, 1092–1097. https://jamanetwork.com/

Teti Mayer, J., Compagne, C., Nicolier, M., Grandperrin, Y., Chabin, T., Giustiniani, J., Haffen, E., Bennabi, D., & Gabriel, D. (2021). Towards a functional neuromarker of impulsivity: Feedback-related brain potential during risky decision-making associated with self-reported impulsivity in a non-clinical sample. Brain Sciences, 11(6). https://doi.org/10.3390/brainsci11060671

Treadway, M. T., & Zald, D. H. (2011). Reconsidering anhedonia in depression: Lessons from translational neuroscience. In Neuroscience and Biobehavioral Reviews (Vol. 35, Issue 3, pp. 537–555). https://doi.org/10.1016/j.neubiorev.2010.06.006

Treadway, M. T., & Zald, D. H. (2013). Parsing Anhedonia: Translational Models of Reward-Processing Deficits in Psychopathology. Current Directions in Psychological Science, 22(3), 244–249. https://doi.org/10.1177/0963721412474460

Uher, R., Perlis, R. H., Henigsberg, N., Zobel, A., Rietschel, M., Mors, O., Hauser, J., Dernovsek, M. Z., Souery, D., Bajs, M., Maier, W., Aitchison, K. J., Farmer, A., & McGuffin, P. (2012). Depression symptom dimensions as predictors of antidepressant treatment outcome: Replicable evidence for interest-activity symptoms. Psychological Medicine, 42(5), 967–980. https://doi.org/10.1017/S0033291711001905

Van Leijenhorst, L., Zanolie, K., Van Meel, C. S., Westenberg, P. M., Rombouts, S. A. R. B., & Crone, E. A. (2010). What motivates the adolescent? brain regions mediating reward sensitivity across adolescence. Cerebral Cortex, 20(1), 61–69. https://doi.org/10.1093/cercor/bhp078

Vinckier, F., Gourion, D., & Mouchabac, S. (2017). Anhedonia predicts poor psychosocial functioning: Results from a large cohort of patients treated for major depressive disorder by general practitioners. European Psychiatry, 44, 1–8. https://doi.org/10.1016/j.eurpsy.2017.02.485

Walentowska, W., Paul, K., Severo, M. C., Moors, A., & Pourtois, G. (2018). Relevance and uncertainty jointly influence reward anticipation at the level of the SPN ERP component.

International Journal of Psychophysiology, 132, 287–297. https://doi.org/10.1016/j.ijpsycho.2017.11.005

Wang, J., Huang, J., Yang, X. hua, Lui, S. S. Y., Cheung, E. F. C., & Chan, R. C. K. (2015). Anhedonia in schizophrenia: Deficits in both motivation and hedonic capacity. Schizophrenia Research, 168(1–2), 465–474. https://doi.org/10.1016/j.schres.2015.06.019

Winer, E. S., Jordan, D. G., & Collins, A. C. (2019). Conceptualizing anhedonias and implications for depression treatments. In Psychology Research and Behavior Management (Vol. 12, pp. 325–335). Dove Medical Press Ltd. https://doi.org/10.2147/PRBM.S159260

Wu, Y., & Zhou, X. (2009). The P300 and reward valence, magnitude, and expectancy in outcome evaluation. Brain Research, 1286, 114–122. https://doi.org/10.1016/j.brainres.2009.06.032

Yeung, N., & Sanfey, A. G. (2004). Independent coding of reward magnitude and valence in the human brain. Journal of Neuroscience, 24(28), 6258–6264. https://doi.org/10.1523/JNEUROSCI.4537-03.2004

Zhao, Q., Li, H., Hu, B., Wu, H., & Liu, Q. (2017). Abstinent heroin addicts tend to take risks: ERP and source localization. Frontiers in Neuroscience, 11(DEC). https://doi.org/10.3389/fnins.2017.00681

Zheng, Y., Li, Q., Wang, K., Wu, H., & Liu, X. (2015). Contextual valence modulates the neural dynamics of risk processing. Psychophysiology, 52(7), 895–904. https://doi.org/10.1111/psyp.12415

Zheng, Y., Li, Q., Zhang, Y., Li, Q., Shen, H., Gao, Q., & Zhou, S. (2017). Reward processing in gain versus loss context: An ERP study. Psychophysiology, 54(7), 1040–1053. https://doi.org/10.1111/psyp.12855