Aberrant reward dynamics in trait anticipatory anhedonia

Shiyu Zhou, Lu Nie, Zhao Wang, Mengyao Wang, and Ya Zheng

Department of Psychology, Dalian Medical University, Dalian, China

Correspondence should be addressed to Ya Zheng, Department of Psychology, Dalian Medical University, No. 9 West Section, Lvshun South Road, Dalian 116044, China. E-mail: zhengya@dmu.edu.cn.

Abstract

As a cardinal feature of several psychiatric disorders, anhedonia includes a consummatory component (deficits in hedonic response to rewards) and an anticipatory component (a reduced motivation to pursue them). Although being conceptualized as impairments of reward system, the neural characterization of reward processing in anhedonia is hampered by the enormous heterogeneity in the reward phase ('wanting' vs 'liking') and comorbidity (inherent to disease states). The current event-related potential (ERP) study examined the reward dynamics of anticipatory anhedonia in a non-clinical sample. Anticipatory and consummatory ERP components were assessed with a monetary incentive delay task in a high anticipatory anhedonia (HAA) group and a low anticipatory anhedonia (LAA) group. HAA vs LAA group showed a diminished reward-related speeding during behavioral performance and reported overall reduced positive affect during anticipation and receipt of outcomes. Importantly, neural dynamics underlying reward processing were negatively associated with anticipatory anhedonia across the anticipatory phase indexed by the contingent negative variation and the consummatory phase indexed by the feedback P3. Our results suggest that anticipatory anhedonia in non-clinical individuals is linked to a poor modulation during both anticipatory and consummatory phases of reward processing.

Key words: anticipatory anhedonia; reward dynamics; event-related potentials

Introduction

The Research Domain Criteria (RDoC) launched by the National Institute of Mental Health proposes that classifying psychiatric illness should be grounded in core brain-behavior dimensions, rather than clinical observations (Insel et al., 2010; Insel & Cuthbert, 2015). One goal of the RDoC framework is to identify pathophysiological mechanisms that are common to various psychiatric disorders. Anhedonia, the reduced capacity to experience pleasure, fits into the RDoC Positive Valence Systems domain (Nusslock & Alloy, 2017). Anhedonia represents a prominent example in that it is associated with multiple psychiatric disorders as diverse as major depression disorder (MDD; Klein, 1984), schizophrenia (SZ; Meehl, 1962), bipolar disorder (Leibenluft et al., 2003), substance use disorder (Markou et al., 1998) and post-traumatic stress disorder (Nawijn et al., 2015). Characterization of the neural correlates underlying transdiagnostic anhedonia is thus of paramount importance in prevention and diagnosis of relevant psychiatric disorders.

Anhedonia has been studied under the framework of reward processing (Der-Avakian & Markou, 2012). In humans, functional magnetic resonance imaging (fMRI) studies have established an association between aberrant activation in brain areas implicated in reward processing (i.e. mesolimbic and mesocortical pathways) and anhedonia severity in individuals with SZ (Juckel et al., 2006; Park et al., 2009; Harvey et al., 2010) and MDD (Dunn et al., 2002; Keedwell et al., 2005; Gong et al., 2018), as well as those free of psychopathological conditions (Harvey et al., 2007; Keller et al., 2013). Despite its excellent spatial resolution, fMRI is mute to the neural dynamics of reward processing because of its inferior time resolution. This issue is non-trivial because reward is not a single construct but can be decomposed into at least
an anticipatory component and a consummatory component at both psychological and neural levels (Berridge & Robinson, 2003). Aligning with the multi-facets of the reward system, recent theories highlight that anhedonia consists of an anticipatory component, which is associated with a reduced motivation to pursue rewards, and a consummatory component, which is linked to deficits in hedonic response to rewards (Treadway & Zald, 2011; Romer Thomsen et al., 2015). The two components, anticipatory and consummatory anhedonia, can be captured by the Temporal Experience of Pleasure Scale (TEPS; Gard et al., 2006). Recent progress indicates that anticipatory anhedonia is more relevant to aberrant reward processing than consummatory anhedonia (Treadway & Zald, 2011; Romer Thomsen et al., 2015; Nusslock & Alloy, 2017).

With its superior temporal resolution, the event-related potential (ERP) technique is very suitable to decompose anticipatory and consummatory aspects in reward processing (Glazer et al., 2018), thus allowing for a direct characterization of reward processing in anhedonia in terms of neural dynamics. During the anticipatory phase of reward processing, three ERP components have been identified: cue-P3, the contingent negative variation (CNV) and the stimulus-preceding negativity (SPN), each with distinct functional significance. Cue-P3, a positive deflection between 300 and 600 ms with a parietal distribution, is usually larger for incentive than neutral cues and is thought to index the allocation of attention resources based on motivational significance (Broyd et al., 2012; Zhang et al., 2017). As a central negative-going deflection, the CNV is associated with anticipatory attention, motivation and response readiness for an imperative target (Walter et al., 1964; Tecce, 1972). Isolated from the CNV, the SPN is a broad slow negative-going wave during the waiting period of motivational feedback and is thought to reflect incentive anticipation without motor preparation (Damen & Brunia, 1987; Brunia, 1988; Zheng et al., 2017).

On the other hand, two ERP components, the feedback-related negativity (FRN) and the feedback P3 (fb-P3), are relevant to reward consumption. The FRN is a relative negativity peaking between 250 and 350 ms over frontocentral areas (Miltner et al., 1997). Whereas early research highlights that the FRN indexes a reward prediction error signal (Holroyd & Coles, 2002), recent research emphasizes that this component is directly associated with reward consumption such that a reward positivity elicited by rewards is superimposed on a baseline negativity elicited by both rewards and non-rewards/losses (Holroyd et al., 2008; Proudfit, 2015). The ensuing fb-P3 is a parietal positivity occurring between 300 and 600 ms and has been proposed to reflect motivational salience during feedback processing (Nieuwenhuis et al., 2005; San Martin, 2012).

To date, few ERP studies have addressed the reward dynamics in anhedonia using clinical populations with SZ (Wynn et al., 2010; Vignapiano et al., 2016) and MDD (Liu et al., 2013), as well as non-clinical populations (Simons et al., 1982; Padrao et al., 2013; Chen et al., 2018). With regard to clinical populations, Vignapiano et al. (2016) reported that cue-P3 but not the CNV elicited in the reward-anticipation phase was negatively correlated with trait anhedonia in patients with SZ. In patients with MDD, Liu et al. (2015) found a negative correlation between anhedonia severity and the FRN elicited in the reward-attainment phase. Additionally, Wynn et al. (2010) found no reliable relationship between two anticipatory ERP components, the CNV and SPN, and trait anhedonia among individuals with SZ. With respect to non-clinical populations, an early study observed an enhanced CNV when anticipating high interest (sexual-related slides) vs low interest (neutral slides) stimuli for non-anhedonic participants. In contrast, anhedonic subjects were insensitive to the content of the anticipated stimuli (Simons et al., 1982). Another study found comparable FRN responses between extreme groups of anhedonic and non-anhedonic participants (Padrao et al., 2013), indicating preserved consummatory processing in anhedonia. A recent study reported that participants with anticipatory anhedonia showed a reduced cue-P3 but a comparable SPN during the anticipatory phase and a less positive FRN and a blunted fb-P3 during the consummatory phase compared to those with consummatory anhedonia (Chen et al., 2018).

Taken together, these previous findings indicate a potential association between anhedonia, either in clinical or in non-clinical populations, and neural dynamics of reward processing, either anticipatory or consummatory ERP components. However, several potential limitations preclude the neural characterization of reward processing in anhedonia. For studies using clinical populations, one cannot determine whether the abnormally electrophysiological activity is associated specifically with anhedonia or results from other dimensions of the disorder (Harvey et al., 2007). Moreover, given the multi-facet nature of reward processing (Glazer et al., 2018), it remains ambiguous whether anhedonia is manifested as abnormal mechanisms in the anticipatory phase or the consummatory phase of reward processing, or both. Finally, previous research, except for our recent one (Chen et al., 2018), failed to discriminate between anticipatory anhedonia and consummatory anhedonia, corresponding to the so-called symptom heterogeneity in clinical diagnosis of anhedonia (Treadway & Zald, 2011). One way to overcome these limitations is to examine the neural dynamics of reward processing in a purer type of anhedonia among individuals free of psychopathological status. Studies with non-clinical samples are important to rule out confounding variables inherent to clinical status and provide in turn strong support that anhedonia constitutes a vulnerability marker of clinical diseases including MDD and SZ (Gottesman, 2003).

Here, we sought to characterize the neural dynamics of reward processing in trait anhedonia using a non-clinical sample to exclude potential confounding effects of psychiatric disorders. Because of a recent emphasis on the anticipatory aspect of anhedonia (Treadway & Zald, 2011), we focused on anticipatory anhedonia by varying anticipatory anhedonia but stabilizing consummatory anhedonia. We recorded anticipatory (i.e. cue-P3, CNV and SPN) and consummatory (the FRN and fb-P3) ERP components in a high anticipatory anhedonia (HAA) group and a low anticipatory anhedonia (LAA) group while they were performing a modified monetary incentive delay (MID) task, a well-known task that can separate the anticipatory phase from the consummatory phase in reward processing (Knutson et al., 2000). We hypothesized that elevated anticipatory anhedonia would be associated with abnormally electrophysiological activities during the anticipatory phase, rather than the consummatory phase, of reward processing.

Methods
Participants
A group of 54 right-handed volunteers with a mean age of 19.0 years (SD = 1.00) were recruited as participants based on their scores on the 20-item Chinese version of the TEPS (Gard et al., 2006; Chan et al., 2012), which was administered in a sample of 585 university students. The TEPS is a 6-point Likert scale (1 = very false for me and 6 = very true for me) designed to...
measure anticipatory pleasure and consummatory pleasure. Lower scores obtained from the anticipatory and consummatory subscales indicate higher levels of anticipatory and consummatory anhedonia, respectively. The Chinese version of the TEPS has been demonstrated to have a good reliability in previous studies (Chan et al., 2010; Chan et al., 2012). Cronbach’s alphas for the anticipatory and consummatory subscales in the present sample were 0.70 and 0.74, respectively. An HAA group and an LAA group were created as follows: firstly, the mean and standard deviation (SD) of the distribution of the consummatory-pleasure scores of the sample (N = 585) were computed (M = 43.01, SD = 7.74), resulting in 252 potential participants whose consummatory-pleasure scores were M ± 0.5 SD; secondly, respondents scoring in top and bottom quartiles of the distribution of the anticipatory-pleasure scores of the potential sample (N = 252) were classified into HAA and LAA groups, respectively. This sampling strategy was used to isolate anticipatory anhedonia from consummatory anhedonia, that is, varying anticipatory anhedonia but stabilizing consummatory anhedonia. Potential participants were subsequently invited separately from the HAA and LAA groups, leading to a final sample of 27 subjects with HAA and 27 subjects with LAA. None of the participants had current psychiatric disorders, as determined by the Structured Clinical Interview for DSM-IV, Patient Edition (First et al., 1995). Participants also reported no history of head trauma, neurological illnesses or substance abuse.

Participants also completed the Snaith–Hamilton Pleasure Scale (SHAPS; Harvey et al., 2007) to assess the severity of state anhedonia and the Behavioral Inhibition System/Behavioral Activation System Scales (BIS/BAS; Carver & White, 1994) to measure their approach and avoidance motivational tendencies. Participants received monetary compensation of ¥30 for participation, as well as a bonus depending on their performance in the task. All participants gave written informed consent prior to the experiment. This study was approved by the Institutional Review Board of the Dalian Medical University in accordance with the Helsinki Declaration as revised 1989.

Procedure
To dissociate anticipatory and consummatory phases of reward processing, participants completed a modified MID task (Zhang et al., 2017), which can exclude the confounding influences of outcome probability and visual characteristic on feedback processing (see details below). In the MID task (Figure 1), participants were encouraged to maximize their rewards by making a response as quickly as possible to a visual target. Each trial began with a cue (either a circle or a square) for 1000 ms signaling a potential monetary reward (a gain context) or no money at stake (a neutral context). The type of the cue was counterbalanced across participants. Following a jittered interstimulus interval (ISI; 2000–2500 ms), participants responded to the target (a white square) by pressing a button quickly using their right index finger. Target duration was constantly adapted to render an ~50% of success rate in each context at the individual level. To this end, a staircase algorithm was implemented for gain and neutral trials separately in which the duration of the target would be decreased by 25 ms following a successful response (i.e. a button response occurred during target presentation) and increased by 25 ms following an unsuccessful response (i.e. a button response occurred after target presentation), with a minimum of 100 ms and a maximum of 400 ms duration (initial value: 250 ms). Following their response, a second ISI lasted for 2000 ms during which participants anticipated their performance outcome. A successful response was signaled by a white tick, an unsuccessful response by a white cross; the performance feedback was presented for 1000 ms. In the gain context, the tick feedback indicated a winning of ¥1 whereas the cross feedback resulted in ¥0. In the neutral context, both tick and cross feedback resulted in ¥0. Each trial ended with an intertrial interval ranging from 1200 to 1500 ms. Participants were instructed to respond as fast as possible irrespective of cue type to control for the confounding influence of motor preparation associated with the incentive cue (Vignapiano et al., 2016).

The task included four blocks of 40 trials (20 gain and 20 neutral trials), and a rest break was provided between blocks. Every two blocks, participants rated their affective responses for the anticipation and receipt of outcomes in both contexts by completing a 5-point Likert scale in terms of valence (1 = very negative and 5 = very positive) and arousal (1 = not arousing at all and 5 = very arousing). Prior to the experiment, 20 practice trials were provided to familiarize participants with the task.

Recording and analysis
The EEG was recorded continuously from a set of 64 Ag/AgCl electrodes at the extended International 10/20 locations and referenced to the left mastoid electrode. Horizontal eye movements were monitored with a pair of electrodes placed at the external canthi of both eyes. Vertical eye movements and blinks were detected via a pair of electrodes placed above and below the
left eye. Electrode impedances were kept <5 kΩ throughout the experiment. The EEG signals were amplified and digitized using a Neuroscan SynAmps² amplifier with a low pass of 100 Hz in DC acquisition mode and a sample rate of 500 Hz.

The EEG data were analyzed using MATLAB 2014a (Math-Works, Natick, MA) and EEGLAB toolbox v13.1.1 (Delorme & Makeig, 2004). The EEG data were re-referenced offline to the mean of the activity at the left and right mastoids. For the CNV analysis, the raw EEG data were filtered with a low pass at 30 Hz (roll-off 6 dB/octave) and then epoched from −200 to 0 ms serving as the baseline; for the SPN analysis, the raw EEG data were filtered with a low-pass at 30 Hz (roll-off 6 dB/octave) and then epoched from −4000 to 3000 ms relative to feedback onset with the activity from −200 to 0 ms serving as the baseline; for the FRN, the FRN and other ERP components including the preceding P2 were isolated, a difference waveform (unsuccessful vs successful outcomes) was created separately for the gain context and neutral contexts, which could minimize the overlap between cue-P3 and feedback-P3 analyses, the raw EEG data were filtered with a bandpass of 0.1 and 30 Hz (roll-off 6 dB/octave) and then epoched from −1000 to 1500 ms relative to cue onset (cue-P3) or feedback (the FRN and fb-P3) onset with the activity from −200 to 0 ms serving as the baseline. All epoched data were screened manually for artifacts (e.g., spikes, drifts and nonbiological signals) and then were entered into an Infomax independent components analysis (runica). Individual components were inspected, and blink components were removed. To remove additional artifacts, epochs containing a voltage difference more than 50 μV between sample points, a voltage difference exceeding 200 μV within an epoch or a maximum voltage difference less than 0.5 μV within 100-ms intervals were automatically rejected. The clean data were then averaged for each condition for each participant. The number of accepted trials (around 90%) was comparable between groups for each ERP component and task condition (ps > .05). For visualization, the CNV and SPN data were filtered with a low-pass cutoff at 7 Hz as implemented in the ERLAB Toolbox (Lopez-Calderon & Luck, 2014).

ERP components were measured as the mean activity of different time windows using a region-of-interest (ROI) approach. Time window and electrodes for each component were selected based on averaged ERPs across groups in waveforms and topographic maps, which thus was orthogonal to the conditions of interest (Luck & Gaspelin, 2017). Specifically, cue-P3 was scored from 400 to 550 ms relative to cue onset over a parietal ROI (P1, Pz, P2, PO2); the CNV from 2800 to 3000 ms relative to cue onset over a central ROI (FCz, C1, Cz, C2); the SPN from −200 to 0 ms relative to feedback onset over a frontotemporal ROI (F7, F8, FT7, FT8); and fb-P3 from 330 to 430 ms post-feedback onset over a centroparietal ROI (Cz, CP1, CPz, CP2). To isolate the FRN component, a difference waveform (unsuccessful minus successful outcomes) was created separately for the gain and neutral contexts, which could minimize the overlap between the FRN and other ERP components including the preceding P2 and the following P3 (Sambrook & Goslin, 2015). The FRN was then scored as the mean activity of the difference waveforms from 230 to 330 ms post-feedback onset over a frontocentral ROI (Fz, FCz). The widths of the windows and specific electrodes are consistent with prior studies (Novak & Foti, 2015; Zhang et al., 2017).

Table 1. Sample characteristics and behavioral data (M ± SD)

|                      | HAA (N = 27) | LAA (N = 27) |
|----------------------|-------------|-------------|
| Gender (M/F)         | 6/21        | 4/23        |
| Age (years)          | 19.04 ± 1.13| 18.93 ± 0.87|
| Education (years)    | 12.89 ± 1.09| 12.78 ± 0.80|
| TEPS                 |             |             |
| Anticipatory pleasure| 27.41 ± 3.26| 41.30 ± 3.23|
| Consumatory pleasure | 41.70 ± 2.49| 42.67 ± 2.54|
| SHAPS                | 24.33 ± 4.91| 21.67 ± 4.10|
| BIS/BAS scales       |             |             |
| BIS                  | 20.41 ± 2.39| 20.26 ± 3.57|
| BAS                  |             |             |
| Drive                | 10.22 ± 1.55| 11.11 ± 1.97|
| Fun seeking          | 13.56 ± 1.87| 15.19 ± 2.25|
| Reward responsiveness| 12.52 ± 1.37| 13.93 ± 1.73|
| Success rates        |             |             |
| Gain context         | 0.50 ± 0.01 | 0.50 ± 0.01 |
| Neutral context      | 0.50 ± 0.01 | 0.49 ± 0.01 |
| Reaction times (ms)  |             |             |
| Gain context         | 235.25 ± 41.32| 232.85 ± 27.01|
| Neutral context      | 246.51 ± 38.14| 259.50 ± 41.01|

Note: HAA = high anticipatory anhedonia; LAA = low anticipatory anhedonia. TEPS = Temporal Experience of Pleasure Scale; SHAPS = Snaith-Hamilton Pleasure Scale; BIS/BAS = Behavioral Inhibition System/Behavioral Activation System. Significant group differences are indicated in bold.

²We also analyzed FRN data with the factor outcome (successful vs unsuccessful) included, as did for fb-P3 data. The FRN was more positive in the gain context than in the neutral context, F(1, 52) = 29.93, P < .001, η² = .37, and more positive for successful compared to unsuccessful outcomes, F(1, 52) = 88.91, P < .001, η² = .63. The outcome effect was more pronounced in the gain than neutral context, resulting in a significant interaction between context and outcome, F(1, 52) = 10.28, P = .002, η² = .17. Although the main effect of group was not significant, F(1, 52) = 2.96, P = .091, η² = .05, the interaction between context and group was significant, F(1, 52) = 6.09, P = .017, η² = .11. Post hoc comparisons revealed that HAA elicited a less positive FRN than LAA in the gain context (8.73 ± 9.19 μV, P = .032), but not in the neutral context (7.43 ± 8.47 μV, P = .363). No other significant effects were obtained, F(1, 52) = 1.44–0.59, P = .235–.445. However, it should be noted that these effects might be driven by fb-P3 modulation, as the correlations between the two components were highly significantly correlated, r > .59, P < .0001. Therefore, our FRN findings would focus on those obtained using the difference wave approach.
Table 2. Affective rating data (M ± SD) for gain and neutral trials as a function of group

|                | HAA Gain   | HAA Neutral | LAA Gain   | LAA Neutral |
|----------------|------------|-------------|------------|-------------|
| Anticipation   |            |             |            |             |
| Valence        | 3.26 ± 0.63| 3.09 ± 0.54 | 3.67 ± 0.64| 3.24 ± 0.58 |
| Arousal        | 3.35 ± 0.65| 3.13 ± 0.66 | 3.70 ± 0.76| 3.44 ± 0.85 |
| Successful outcome |       |             |            |             |
| Valence        | 3.70 ± 0.84| 3.76 ± 0.81 | 4.07 ± 0.74| 4.04 ± 0.69 |
| Arousal        | 3.39 ± 0.87| 3.35 ± 0.73 | 3.83 ± 0.76| 3.52 ± 1.15 |
| Unsuccessful outcome |     |             |            |             |
| Valence        | 2.56 ± 0.58| 2.48 ± 0.58 | 2.70 ± 0.65| 2.69 ± 0.48 |
| Arousal        | 3.11 ± 0.63| 2.96 ± 0.68 | 3.48 ± 0.70| 3.17 ± 0.91 |

Note. Data were averaged across the first and second ratings. HAA = high anticipatory anhedonia; LAA = low anticipatory anhedonia. Significant group differences are indicated in bold.

Statistical analyses were conducted in SPSS v22 (IBM, Armonk, NY). Success rates and reaction times (RTs) were analyzed separately with a mixed repeated-measures analysis of variance (ANOVA), with group (HAA vs LAA) as a between-subject factor and context (gain vs neutral) as a within-subject factor. Affective rating data were analyzed using a Group × Context ANOVA for outcome anticipation and a Group × Context × Outcome (successful vs unsuccessful) ANOVA for outcome delivery; ERP data were analyzed with a Group × Context ANOVA for cue-P3, CNV, SPN and FRN separately and a Group × Context × Outcome ANOVA for fb-P3. Greenhouse–Geisser epsilon correction was applied when appropriate, with the Bonferroni correction for post hoc comparisons. In addition, to provide quantified evidence for the null hypothesis significance testing, we performed a Bayesian repeated-measures ANOVA for each ERP component separately with the JASP software v0.9.2 (Wagenmakers et al., 2018) with the default prior settings.

Results

Demographic and behavioral data

Table 1 shows the demographic and behavioral data for HAA and LAA groups, respectively. The two groups were matched in terms of gender, age and education level (P > .8). As expected, HAA group scored significantly lower than LAA group on the anticipatory, t(52) = −18.04, P < .001, Cohen’s d = 5.00, but not consummatory, t(52) = −1.41, P = .166, Cohen’s d = 0.39, subscale of the TEPS. Further, the HAA group had significantly higher scores on the SHAPS than the LAA group, t(52) = 2.17, P = .035, Cohen’s d = 0.60, indicating a higher level of state anhedonia in the former group. Additionally, the HAA group compared to the LAA group showed significantly reduced tendencies in approach motivation in terms of fun seeking, t(52) = −2.89, P = .006, Cohen’s d = 0.80, and reward responsiveness, t(52) = −3.31, P = .002, Cohen’s d = 0.92, but not drive, t(52) = −1.84, P = .071, Cohen’s d = 0.51. No group difference was found for avoidance motivation as reflected by the BIS score, t(52) = 0.18, P = .858, Cohen’s d = 0.05.

The ANOVA performed on success rates yielded a significant interaction between context and group, F(1, 52) = 5.58, P = .022, ηp2 = .10. Post hoc comparisons indicated that whereas the LAA group displayed higher success rates for the gain context than for the neutral context (P = .030), the HAA group showed comparable success rates across contexts (P = .270). Moreover, RTs were significantly faster in the gain context than in the neutral context, F(1,52) = 38.64, P < .001, ηp2 = .43. This reward-related speeding, however, was less pronounced for the HAA group (ΔM = 11 ms) than for the LAA group (ΔM = 27 ms), as revealed by a significant interaction between group and context, F(1,52) = 6.37, P = .015, ηp2 = .11.

Affective rating data

Table 2 shows valence- and arousal-rating scores (averaged across the first and second ratings) for the anticipation and delivery of outcomes during the two contexts as a function of group. Results revealed that the gain context was rated as more pleasant, F(1, 52) = 9.92, P = .003, ηp2 = .16, and more arousing, F(1, 52) = 7.61, P = .008, ηp2 = .13, than the neutral context. Successful outcomes were rated as more pleasant, F(1, 52) = 96.89, P < .001, ηp2 = .65, and more arousing, F(1, 52) = 15.76, P < .001, ηp2 = .23, than unsuccessful outcomes. Critically, the HAA relative to LAA group reported overall reduced positive affect for both outcome anticipation, F(1, 52) = 4.43, P = .040, ηp2 = .08, and outcome delivery, F(1, 52) = 5.59, P = .022, ηp2 = .10. No other significant effects were obtained (P > .05).

ERP data

Anticipatory ERP results. Figure 2 shows grand-averaged ERP waveforms elicited during the cue-evaluation, motor-preparation and feedback-anticipation stages of the anticipatory phase, as well as scalp topographic maps for cue-P3, CNV and SPN. As in previous research, all these anticipatory ERP components displayed a canonical scalp distribution.

Cue-P3 was larger (more positive) for the gain context than for the neutral context, F(1, 52) = 19.09, P < .001, ηp2 = .27. The HAA group tended to elicit a smaller cue-P3 than did the LAA group, as revealed by a marginally significant main effect of group, F(1, 52) = 3.68, P = .061, ηp2 = .07. However, the interaction effect between context and group failed to achieve statistical significance, F(1, 52) = 2.29, P = .136, ηp2 = .04 (see Table S1 in Supplementary Materials for Bayesian statistics of the cue-P3 data).

The ANOVA performed on CNV data revealed no significant main effects of context, F(1, 52) = 2.06, P = .157, ηp2 = .04, and group, F(1, 52) = 0.72, P = .400, ηp2 = .01. However, there was a significant interaction between context and group, F(1, 52) = 5.49, P = .023, ηp2 = .10. Post hoc comparisons indicated that CNV amplitudes were significantly enhanced (more negative) for the gain context (−9.71 μV) vs the neutral context (−7.44 μV) in the LAA group (P = .010). In contrast, CNV amplitudes were comparable between
Fig. 2. Grand-averaged ERP waveforms as a function of group during the anticipation phase, where the shaded areas demarcate the time windows during which cue-P3 (400–550 ms), CNV (2800–3000 ms) and SPN (−200–0 ms) were scored. Topographical distribution maps for these ERP components are also shown.

the gain context (−9.54 μV) and the neutral context (−10.09 μV) in the HAA group (P = .524) (see Table S2 in Supplementary Materials for Bayesian statistics of the CNV data).

As the CNV has been associated with target anticipation and motor preparation, a series of Pearson’s correlations were performed to examine the relationship between CNV amplitudes and RTs in the two groups, respectively. As shown in Figure 3, for the LAA group, greater CNV amplitudes were associated with faster RTs in the gain context, r = .42, P = .028, but not the neutral context, r = .34, P = .087. By contrast, for the HAA group, greater CNV amplitudes were associated with faster RTs in both the gain context, r = .61, P = .001, and the neutral context, r = .49, P = .009. However, the correlation coefficients were not significantly different between groups for both the gain context, z = 0.87, P = .382, and the neutral context, z = 0.66, P = .512.

SPN was larger (more negative) for the gain context than for the neutral context, F(1, 52) = 9.53, P = .003, ηp² = .16. Neither the main effect of group, F(1, 52) = 2.80, P = .100, ηp² = .05, nor the interaction effect between context and group, F(1, 52) = 0.02, P = .920, ηp² < .01, was significant (see Table S3 in Supplementary Materials for Bayesian statistics of the SPN data).

Consummatory ERP results. Figure 4 illustrates grand-averaged waveforms elicited during the consummatory phase, as well as scalp topographic maps for the FRN and fb-P3. As in previous research, whereas the FRN showed a classical frontocentral distribution, fb-P3 displayed a centroparietal distribution.

FRN data revealed a significant main effect of context, F(1, 52) = 10.28, P < .001, ηp² = .17, with an enhanced (more negative) FRN for the gain vs neutral context. Neither the main effect of group, F(1, 52) = 1.44, P = .235, ηp² = .03, nor the interaction between group and context, F(1, 52) = 0.59, P = .445, ηp² = .01, was significant (see Table S4 in Supplementary Materials for Bayesian statistics of the FRN data). With regard to fb-P3, there was a significant main effect of context, F(1, 52) = 38.20, P < .001, ηp² = .42, indicating that fb-P3 was larger for the gain than neutral context. The main effect of group was also significant, F(1, 52) = 4.07, P = .049, ηp² = .07, with a smaller fb-P3 for the HAA group than for the LAA group. This group effect was present for the gain context (14.35 vs 18.28 μV, P = .018), but not for the neutral context (12.59 vs 14.12 μV, P = .219), as revealed by a significant interaction between context and group, F(1, 52) = 6.28, P = .015, ηp² = .11. No other significant effects were obtained,
Fig. 3. Scatterplots of the correlations between CNV amplitudes and RTs as a function of group in gain and neutral contexts.

Fig. 4. Grand-averaged ERP waveforms as a function of group during the consummatory phase, where shaded areas demarcate the time windows during which the FRN (230–330 ms) and fb-P3 (330–430 ms) were scored. Topographical distribution maps for these ERP components are also shown. SC = successful; US = unsuccessful.

F(1,52) = 0.20–0.02, P = .655–.889 (see Table S5 in Supplementary Materials for Bayesian statistics of the fb-P3 data).

Relationships between cue-P3 and fb-P3 as a function of group. Since we found a (marginally) significant effect of anhedonia at the P3 at both anticipatory (cue-P3) and consummatory (fb-P3) phases, Person’s correlation was performed to evaluate the relationship between cue-P3 and fb-P3 (Table 3). As in a previous study (Zheng et al., 2017), the two components were positively correlated with each other in both contexts. However, the relationships were modulated by anticipatory anhedonia. For LAA, cue-P3 was significantly correlated with fb-P3 in the gain context but not the neutral context. For HAA, the relationship was reversed such that cue-P3 was significantly correlated with fb-P3 in the neutral context but not the gain context.

Discussion

This study examined the neural dynamics of reward processing in anticipatory anhedonia using a non-clinical sample. To our knowledge, this is the first study to decompose the relative contributions of anticipatory vs consummatory aspects of reward processing in trait anhedonia. The HAA relative to LAA group reported an enhanced trait anticipatory anhedonia and a higher level of state anhedonia, as well as a reduced tendency in approach but not avoidance motivation. Behaviorally, the HAA relative to LAA group showed a diminished reward priority, as
reflected by their comparable success rates across contexts and less pronounced reward-related speeding. Similarly, the HAA vs LAA group reported overall reduced positive affect during anticipation and receipt of outcomes. Importantly, neural dynamics underlying reward processing were negatively associated with anticipatory anhedonia across anticipatory (as indexed by the CNV) and consummatory (as indexed by fb-P3) phases. In sum, these findings provide novel insights into the relationship between anhedonic symptoms and neural dynamics of reward processing.

In previous studies, anhedonia has been regarded as a single structure, and thus, it remains ambiguous whether the observed aberrant reward processing is associated with anticipatory anhedonia or consummatory anhedonia (Treadway & Zald, 2011; Romer Thomsen et al., 2015; Nusslock & Alloy, 2017). The fact that we recruited subjects with HAA and LAA based on their extreme anticipatory anhedonic score while stabilizing their consummatory anhedonic score on the TEPS differentiates our study from previous ones (Simons et al., 1982; Harvey et al., 2007; Wacker et al., 2009; Keller et al., 2013; Padrao et al., 2013). By this way, the current study revealed aberrant reward dynamics among non-clinical individuals high in anticipatory anhedonia without confounding influences from consummatory anhedonia.

Before exploring the reward dynamics of anhedonia, it is important to verify whether the observed aberrant reward processing is associated with anticipatory anhedonia or consummatory anhedonia (Treadway & Zald, 2011; Romer Thomsen et al., 2015; Nusslock & Alloy, 2017). The fact that we recruited subjects with HAA and LAA based on their extreme anticipatory anhedonic score while stabilizing their consummatory anhedonic score on the TEPS differentiates our study from previous ones (Simons et al., 1982; Harvey et al., 2007; Wacker et al., 2009; Keller et al., 2013; Padrao et al., 2013). By this way, the current study revealed aberrant reward dynamics among non-clinical individuals high in anticipatory anhedonia without confounding influences from consummatory anhedonia.

Table 3. Correlations between the cue-P3 and the fb-P3

|                | Gain context | Neutral context |
|----------------|--------------|-----------------|
|                | SC fb-P3     | US fb-P3        | SC fb-P3          | US fb-P3        |
| Cue-P3: Total  | .54***       | .48***          | .40**             | .39**           |
| Cue-P3: LAA    | .65***       | .55**           | .24              | .34             |
| Cue-P3: HAA    | .29          | .30             | .50**            | .42*            |

Note. LAA = low anticipatory anhedonia; HAA = high anticipatory anhedonia; SC = successful; US = unsuccessful. *p < .05. **p < .01. ***p < .001.
fb-P3 significantly decreased as the level of anhedonia increased. This modulation was present in the gain context but not in the neutral context. Given that fb-P3 has been linked to motivational salience during feedback evaluation (Nieuwenhuis et al., 2005; San Martin, 2012), our fb-P3 findings suggest that anticipatory anhedonia is associated with disrupted consummatory reward processing, which is in line with our recent finding that anticipatory anhedonia was negatively correlated with fb-P3 across gain and loss contexts during a simple gambling task (Chen et al., 2018). The inclusion of the neutral context here during which no group difference was obtained indicates that dysfunctional fb-P3 is specific to feedback processing in a potential reward condition (gain and non-gain outcomes), rather than a general deficit in feedback processing.

In contrast, we observed no modulation of anticipatory anhedonia on the FRN (calculated as the difference between unsuccessful and successful feedback), which is consistent with a previous study using a non-clinical sample (Padrao et al., 2013) but at odds with another previous research using a sample with MDD (Liu et al., 2013). The discrepancy between our findings and those reported by Liu and colleagues may be due in part to the use of a clinical sample in their study. It is possible that the relationship between anhedonia and the FRN is attributable to other factors inherent to the disease. Actually, the relationship became non-significant after controlling for depressive symptoms (Liu et al., 2013). Together, it seems that the effects of anhedonia on the FRN might be driven by depressive symptoms and therefore probably hedonic disruptions. These disruptions might not be that strong in a non-clinical sample. Future research should extend our findings to clinical populations with MDD or SZ to determine whether the neural correlates of anhedonia in patients is quantitatively or qualitatively different from those in non-clinical individuals (Harvey et al., 2010).

Finally, we found that the HAA group elicited a smaller cue-P3 than the LAA group at a trend level, which is consistent with a recent study reporting a reduced cue-P3 among non-clinical individuals high in anticipatory anhedonia (Chen et al., 2018), as well as with a previous research finding a negative correlation between cue-P3 and social anhedonia with a sample of healthy controls and subjects with SZ (Vignapiano et al., 2016). Given that a reduced fb-P3 was observed for HAA vs LAA, it is possible that the two P3 components may capture the allocation of attention across anticipatory and consummatory reward processing in a common way, as supported by reliable correlations between the two components (Zheng et al., 2017). Interestingly, we observed independent modulation of cue-P3 and fb-P3 by anticipatory anhedonia. On the one hand, whereas cue-P3 effect emerged irrespective of the type of the context, fb-P3 effect appeared for the gain context but not the neutral context. Therefore, anticipatory anhedonia may be associated with reduced motivational salience in the cue-evaluation stage but with a diminished reward salience in the feedback-evaluation stage. On the other hand, whereas LAA showed a correlation between cue-P3 and fb-P3 in the gain but not the neutral context, HAA exhibited a correlation in the neutral but not the gain context. Together with the CNV findings, the correlation findings suggest that anticipatory anhedonia may be associated with a reduced ability to use reward incentive to modulate decision-making processes.

Several potential limitations should be pointed out. The relationship between consummatory processing as indexed by fb-P3 and anticipatory anhedonia is contrary to our hypothesis. One possibility is that electrophysiological activity during the anticipatory phase is not independent from that during the consummatory phase, as revealed by significant correlations between cue-P3 and fb-P3 across contexts, as well as the CNV and fb-P3 in the gain context ($r = -.45, P = .001$). Further, we did not include a loss context in the MID task and thus were unable to specify whether the findings reported here were driven by incentive valence specifically or by incentive salience generally. However, given that anhedonia is more linked to lacking approach instead of avoidance motivation, it seems relatively reasonable that the abnormal ERP responses were associated with aberrant reward dynamics in anhedonia. Finally, our sample size was relatively small, which might have reduced the power of finding more subtle effects.

Although anhedonia is a cardinal feature of several psychiatric disorders, our results demonstrate that disturbances in reward dynamics in anticipatory anhedonia can also be observed in non-clinical individuals. Our findings suggest that anhedonia severity in this psychiatrically healthy sample is linked to a poor modulation of both the CNV component during the preparation for and anticipation of an upcoming target and fb-P3 component during the receipt of outcomes incurred by the action. Further, anhedonia may be associated with a reduced ability to use incentive information to modulate effort allocation and decision-making processes, because individuals high in anticipatory anhedonia showed a behavior–neural (RTs and CNV) association and a neural–neural (cue-P3 and fb-P3) association in the neutral context instead of the gain context. Given that elevated anhedonia measured in non-clinical individuals has been demonstrated as a marker of vulnerability for the future development of psychiatric disorders including MDD (Loas, 1996) and SZ (Blanchard et al., 2001), it is conceivable that the dysfunctional reward dynamics, the abnormal CNV and fb-P3, linked to anticipatory anhedonia in non-clinical populations may represent a vulnerability marker for these psychiatric disorders. Currently, anhedonia symptoms in these disorders are inadequately addressed by available pharmacological or psychosocial treatments (Calabrese et al., 2014; Whitton et al., 2015). Our findings of the selectively aberrant ERP components (i.e. the CNV and fb-P3) have the potential to provide important insights into the treatment of anhedonia as a clinical symptom in reward-related disorders. Future studies using longitudinal designs are needed to examine the role and effect of these brain abnormalities on the potential development of specific psychiatric disorders.

**Supplementary data**

Supplementary data are available at SCAN online.

**Funding**

This work was supported by the National Natural Science Foundation of China (Grant 31971027).

**Conflict of interest** None declared.

**References**

Berridge, K.C., Robinson, T.E. (2003). Parsing reward. Trends in Neurosciences, 26(9), 507–13.

Blanchard, J.J., Horan, W.P., Brown, S.A. (2001). Diagnostic differences in social anhedonia: a longitudinal study of schizophrenia and major depressive disorder. Journal of Abnormal Psychology, 110(3), 363–71.
Broyd, S.J., Richards, H.J., Helps, S.K., Chronaki, G., Bamford, S., Sonuga-Barke, E.J. (2012). An electrophysiological monetary incentive delay (e-MID) task: a way to decompose the different components of neural response to positive and negative monetary reinforcement. Journal of Neuroscience Methods, 209(1), 40–9.

Brunia, C.H. (1988). Movement and stimulus preceding negativity. Biological Psychology, 26(1–3), 165–78.

Brunia, C.H., Hackley, S.A., van Boxtel, G.J., Kotani, Y., Ohgami, Y. (2011). Waiting to perceive: reward or punishment? Clinical Neurophysiology, 122(5), 588–68.

Brunia, C.H., van Boxtel, G., Böcker, K. (2012). Negative slow waves as indices of anticipation: the bereitsehaftspotential, the contingent negative variation, and the stimulus-preceding negativity. In: Luck, S.J., Kappenman, E.S., editors. The Oxford Handbook of Event-Related Potential Components, USA: New York, Oxford University Press.

Calabrese, J.R., Fava, M., Garibaldi, G., et al. (2014). Methodological approaches and magnitude of the clinical unmet need associated with amotivation in mood disorders. Journal of Affective Disorders, 168, 439–51.

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), 319–33.

Chan, R.C., Wang, Y., Huang, J., et al. (2010). Anticipatory and consummatory components of the experience of pleasure in schizophrenia: cross-cultural validation and extension. Psychiatry Research, 175(1–2), 181–3.

Chan, R.C., Shi, Y.F., Lai, M.K., Wang, Y.N., Wang, Y., Kring, A.M. (2012). The Temporal Experience of Pleasure Scale (TEPS): exploration and confirmation of factor structure in a healthy Chinese sample. PLoS One, 7(4), e35352.

Chen, Y., Xu, J., Zhou, L., Zheng, Y. (2018). The time course of incentive processing in anticipatory and consummatory anhedonia. Journal of Affective Disorders, 238, 442–50.

Damen, E.J., Brunia, C.H. (1987). Changes in heart rate and slow brain potentials related to motor preparation and stimulus anticipation in a time estimation task. Psychophysiology, 24(6), 700–13.

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.

Der-Avakian, A., Markou, A. (2012). The neurobiology of anhedonia and other reward-related deficits. Trends in Neurosciences, 35(1), 68–77.

Dunn, R.T., Kimberl, T.A., Ketter, T.A., Frye, M.A., Willis, M.W., Luckenbaugh, D.A., Post, R.M. (2002). Principal components of the Beck Depression Inventory and regional cerebral metabolism in unipolar and bipolar depression. Biological Psychiatry, 51(5), 387–99.

First, M.B., Spitzer, R.L., Gibbon, M., Williams, J.B. (1995). Structured clinical interview for DSM-IV axis I disorders. In: Patient Edition (SCID-P) Version 2.0, Washington, DC: American Psychiatric Press.

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), 1086–102.

Geaney, J.T., Treadway, M.T., Smillie, L.D. (2015). Trait anticipatory pleasure predicts effort expenditure for reward. PLoS One, 10(6), e0131357.

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.

Gong, L., He, C., Zhang, H., Zhang, H., Zhang, Z., Xie, C. (2018). Disrupted reward and cognitive control networks contribute to anhedonia in depression. Journal of Psychiatric Research, 103, 61–8.

Gottesman, I.I & Gould, T.D. (2003). ‘The endophenotype concept in psychiatry: etymology and strategic intentions’, American Journal of Psychiatry, 160(4), pp. 636–45.

Gu, R., Jiang, Y., Kiser, S., Black, C.L., Broster, L.S., Luo, Y.J., Kelly, T.H. (2017). Impulsive personality dimensions are associated with altered behavioral performance and neural responses in the monetary incentive delay task. Neuropsychologia, 103, 59–68.

Harvey, P.O., Puressner, J., Czechowska, Y., Lepage, M. (2007). Individual differences in trait anhedonia: a structural and functional magnetic resonance imaging study in non-clinical subjects. Molecular Psychiatry, 12(8), 703–767–775.

Harvey, P.O., Armony, J., Malla, A., Lepage, M. (2010). Functional neural substrates of self-reported physical anhedonia in non-clinical individuals and in patients with schizophrenia. Journal of Psychiatric Research, 44(11), 707–16.

Holroyd, C.B., Coles, M.G. (2002). The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. Psychological Review, 109(4), 679–709.

Holroyd, C.B., Pakzad-Vaezi, K.L., Krigolson, O.E. (2008). The feedback correct-related positivity: sensitivity of the event-related brain potential to unexpected positive feedback. Psychophysiology, 45(5), 688–97.

Husain, M., Roiser, J.P. (2018). Neuroscience of apathy and anhedonia: a transdiagnostic approach. Nature Reviews. Neuroscience, 19(8), 470–84.

Insel, T.R., Cuthbert, B.N. (2015). Medicine. Brain disorders? Precisely. Science, 348(6234), 499–500.

Insel, T.R., Cuthbert, B.N., Garvey, M., et al. (2010). Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. American Journal of Psychiatry, 167(7), 748–51.

Juckel, G., Schlagenhauf, F., Koslowski, M., et al. (2006). Dysfunction of ventral striatal reward prediction in schizophrenia. Neurolmage, 29(2), 409–16.

Keedwell, P.A., Andrew, C., Williams, S.C., Brammer, M.J., Phillips, M.L. (2005). The neural correlates of anhedonia in major depressive disorder. Biological Psychiatry, 58(11), 843–53.

Keller, J., Young, C.B., Kelley, E., Prater, K., Levitin, D.J., Menon, V. (2013). Trait anhedonia is associated with reduced reactivity and connectivity of mesolimbic and paralimbic reward pathways. Journal of Psychiatric Research, 47(10), 1319–28.

Klein, D. (1984). Depression and anhedonia. In: Clark, D.C., Fawcett, J., editors. Anhedonia and Affect Deficit States, New York: PMA Publishing.

Knutson, B., Westdorp, A., Kaiser, E., Hommer, D. (2000). FMRI visualization of brain activity during a monetary incentive delay task. Neurolmage, 12(1), 20–7.

Leibenluft, E., Charney, D.S., Pine, D.S. (2003). Researching the pathophysiology of pediatric bipolar disorder. Biological Psychiatry, 53(11), 1099–20.

Liu, W.H., Wang, L.Z., Shang, H.R., Shen, Y., Li, Z., Cheung, E.F, Chan, R.C. (2013). The influence of anhedonia on feedback negativity in major depressive disorder. Neuropsychologia, 53C, 213–20.
Loas, G. (1996). Vulnerability to depression: a model centered on anhedonia. *Journal of Affective Disorders*, 41(1), 39–53.

Lopez-Calderon, J., Luck, S.J. (2014). ERPLAB: an open-source toolbox for the analysis of event-related potentials. *Frontiers in Human Neuroscience*, 8, 213.

Luck, S.J., Gaspelin, N. (2017). How to get statistically significant effects in any ERP experiment (and why you shouldn’t). *Psychophysiology*, 54(1), 146–57.

Markou, A., Kosten, T.R., Koob, G.F. (1998). Neurobiological similarities in depression and drug dependence: a self-medication hypothesis. *Neuropsychopharmacology*, 18(3), 135–74.

Mehler, P.E. (1962). Schizotypia, schizotypy, schizophrenia. *American Psychologist*, 17(12), 827–38.

Mei, S., Li, Q., Liu, X., Zheng, Y. (2018). Monetary incentives modulate feedback-related brain activity. *Scientific Reports*, 8(1), 11913.

Miltner, W.H., Braun, C.H., Coles, M.G. (1997). Event-related brain potentials following incorrect feedback in a time-estimation task: evidence for a “generic” neural system for error detection. *Journal of Cognitive Neuroscience*, 9(6), 788–98.

Nawijn, L., van Zuiden, M., Frijling, J.L., Koch, S.B., Veltman, D.J., Olf, M. (2015). Reward functioning in PTSD: a systematic review exploring the mechanisms underlying anhedonia. *Neuroscience and Biobehavioral Reviews*, 51, 189–204.

Nieuwenhuis, S., Aston-Jones, G., Cohen, J.D. (2005). Decision making, the P3, and the locus coeruleus-norepinephrine system. *Psychological Bulletin*, 131(4), 510–32.

Novak, K.D., Foti, D. (2015). Teasing apart the anticipatory and consummatory processing of monetary incentives: an event-related potential study of reward dynamics. *Psychophysiology*, 52(11), 1470–82.

Novak, B.K., Novak, K.D., Lynam, D.R., Foti, D. (2016). Individual differences in the time course of reward processing: stage-specific links with depression and impulsivity. *Biological Psychology*, 119, 79–90.

Nusslock, R., Alloy, L.B. (2017). Reward processing and mood-related symptoms: an RDoC and translational neuroscience perspective. *Journal of Affective Disorders*, 216, 3–16.

Padrao, G., Mallorqui, A., Cucurell, D., Marco-Pallares, J., Rodriguez-Fornells, A. (2013). Neuropsychiological differences in reward processing in anhedonics. *Cognitive, Affective, & Behavioral Neuroscience*, 13(1), 102–15.

Park, I.H., Kim, J.J., Chun, J., Jung, Y.C., Seok, J.H., Park, H.J., Lee, J.D. (2009). Medial prefrontal default-mode hypoactivity affecting trait physical anhedonia in schizophrenia. *Psychiatry Research*, 171(3), 155–65.

Pessiglione, M., Vinckier, F., Bouret, S., Daunizeau, J., Le Bouc, R. (2018). Why not try harder? Computational approach to motivation deficits in neuro-psychiatric diseases. *Brain*, 141(3), 629–50.

Pfabigan, D.M., Seidel, E.M., Paul, K., et al. (2015). Context-sensitivity of the feedback-related negativity for zero-value feedback outcomes. *Biological Psychology*, 104, 184–92.

Proudfit, G.H. (2015). The reward positivity: from basic research on reward to a biomarker for depression. *Psychophysiology*, 52(4), 449–59.

Romer Thomsen, K., Whybrow, P.C., Kringlebach, M.L. (2015). Reconceptualizing anhedonia: novel perspectives on balancing the pleasure networks in the human brain. *Frontiers in Behavioral Neuroscience*, 9, 49.

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), 213–35.

San Martin, R. (2012). Event-related potential studies of outcome processing and feedback-guided learning. *Frontiers in Human Neuroscience*, 6, 304.

Santesso, D.L., Bogdan, R., Birk, J.L., Goetz, E.L., Holmes, A.J., Pizzagalli, D.A. (2012). Neural responses to negative feedback are related to negative emotionality in healthy adults. *Social Cognitive and Affective Neuroscience*, 7(7), 794–803.

Schevernels, H., Krebs, R.M., Santens, P., Woldorff, M.G., Bohler, C.N. (2014). Task preparation processes related to reward prediction override those related to task-difficulty expectation. *NeuroImage*, 84, 629–47.

Simons, R.F., MacMillan, F.W., 3rd, Ireland, F.B. (1982). Anticipatory pleasure deficit in subjects reporting physical anhedonia: slow cortical evidence. *Biological Psychology*, 14(3–4), 298–310.

Tecce, J.J. (1972). Contingent negative variation (CNV) and psychological processes in man. *Psychological Bulletin*, 77(2), 73–108.

Treadway, M.T., Zald, D.H. (2011). Reconsidering anhedonia in depression: lessons from translational neuroscience. *Neuroscience and Biobehavioral Reviews*, 35(3), 537–55.

Treadway, M.T., Buckholtz, J.W., Schwartzman, A.N., Lambert, W.E., Zald, D.H. (2009). Worth the ‘EEFRT?’ The effort expenditure for rewards task as an objective measure of motivation and anhedonia. *PLoS One*, 8(4), e6598.

Vignapiano, A., Mucci, A., Ford, J., Montefusco, V., Pescia, G.M., Bucci, P., Galderisi, S. (2016). Reward anticipation and trait anhedonia: an electrophysiological investigation in subjects with schizophrenia. *Clinical Neurophysiology*, 127(4), 2149–60.

Wacker, J., Dillon, D.G., Pizzagalli, D.A. (2009). The role of the nucleus accumbens and rostral anterior cingulate cortex in anhedonia: integration of resting EEG, fMRI, and volumetric techniques. *NeuroImage*, 46(1), 327–37.

Wagenmakers, E.J., Love, J., Marsman, M., et al. (2018). Bayesian inference for psychology. Part II: example applications with JASP. *Psychonomic Bulletin & Review*, 25(1), 58–76.

Walter, W.G., Cooper, R., Aldridge, V.J., McCallum, W.C., Winter, A.L. (1964). Contingent negative variation: an electric sign of sensorimotor association and expectancy in the human brain. *Nature*, 203, 380–4.

Whitton, A.E., Treadway, M.T., Pizzagalli, D.A. (2015). Reward processing dysfunction in major depression, bipolar disorder and schizophrenia. *Current Opinion in Psychiatry*, 28(1), 7–12.

Wynn, J.K., Horan, W.P., Kring, A.M., Simons, R.F., Green, M.F. (2010). Impaired anticipatory event-related potentials in schizophrenia. *International Journal of Psychophysiology*, 77(2), 141–9.

Zhang, Y., Li, Q., Wang, Z., Liu, X., Zheng, Y. (2017). Temporal dynamics of reward anticipation in the human brain. *Biological Psychology*, 128, 89–97.

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), 1040–53.