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Aberrant reward dynamics in trait anticipatory anhedonia

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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

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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.* (2013) 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 MMD 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

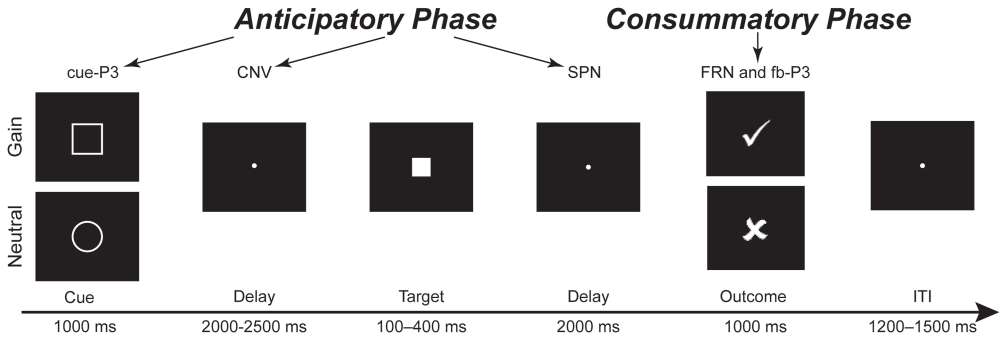

Fig. 1. Schematic representation of the monetary incentive delay task. Relevant ERP components in the anticipatory and consummatory phases are also shown. ITI = intertrial interval.

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 ($M = 43.01$, $SD = 7.74$),[1] resulting in 252 potential participants whose consummatory-pleasure scores were $M \pm 0.5$ *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

[1]The mean and SD of the distribution of the anticipatory-pleasure scores of the sample ($N = 585$) were 35.81 and 6.71, respectively.

Table 1. Sample characteristics and behavioral data ($M \pm SD$)

	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 ± 2.32**
Consummatory 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.

left eye. Electrode impendances were kept <5 KΩ throughout the experiment. The EEG signals were amplified and digitalized using a Neuroscan SynAmps[2] 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 (MathWorks, 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 −4000 to 3000 ms relative to cue onset with the activity 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 −1900 to −1700 ms serving as the baseline; for cue-P3, FRN and fb-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 (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 non-biological 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 ERPLAB 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 over all conditions 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, POz); 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).

[2]We 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, $F(1, 52) = 29.93$, $P < .001$, $\eta_p^2 = .37$, and more positive for successful outcomes than for unsuccessful outcomes, $F(1, 52) = 88.91$, $P < .001$, $\eta_p^2 = .63$. The outcome effect was more pronounced in the gain than neutral context, resulting in a significant interaction between context and outcome, $F(1, 52) = 10.28$, $P = .002$, $\eta_p^2 = .17$. Although the main effect of group was not significant, $F(1, 52) = 2.96$, $P = .091$, $\eta_p^2 = .05$, the interaction between context and group was significant, $F(1, 52) = 6.09$, $P = .017$, $\eta_p^2 = .11$. Post hoc comparisons revealed that HAA elicited a less positive FRN than LAA in the gain context (8.73 *vs* 11.92 μV, $P = .032$), but not in the neutral context (7.43 *vs* 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, $r > .59$, $P < .00001$. Therefore, our FRN findings would focus on those obtained using the difference wave approach.

Table 2. Affective rating data ($M \pm SD$) for gain and neutral trials as a function of group

	HAA		LAA	
	Gain	Neutral	Gain	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	3.74 ± 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 ($P > .6$). 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, $F(1, 52) = 5.58$, $P = .022$, $\eta_p^2 = .10$. 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, $F(1,52) = 38.64$, $P < .001$, $\eta_p^2 = .43$. This reward-related speeding, however, was less pronounced for the HAA group ($\Delta M = 11$ ms) than for the LAA group ($\Delta M = 27$ ms), as revealed by a significant interaction between group and context, $F(1,52) = 6.37$, $P = .015$, $\eta_p^2 = .11$.

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, $F(1, 52) = 9.92$, $P = .003$, $\eta_p^2 = .16$, and more arousing, $F(1, 52) = 7.61$, $P = .008$, $\eta_p^2 = .13$, than the neutral context. Successful outcomes were rated as more pleasant, $F(1, 52) = 96.89$, $P < .001$, $\eta_p^2 = .65$, and more arousing, $F(1, 52) = 15.76$, $P < .001$, $\eta_p^2 = .23$, than unsuccessful outcomes. Critically, the HAA relative to LAA group reported overall reduced positive affect for both outcome anticipation, $F(1, 52) = 4.43$, $P = .040$, $\eta_p^2 = .08$, and outcome delivery, $F(1, 52) = 5.59$, $P = .022$, $\eta_p^2 = .10$. 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, $F(1, 52) = 19.09$, $P < .001$, $\eta_p^2 = .27$. 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, $F(1, 52) = 3.68$, $P = .061$, $\eta_p^2 = .07$. However, the interaction effect between context and group failed to achieve statistical significance, $F(1, 52) = 2.29$, $P = .136$, $\eta_p^2 = .04$ (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, $F(1, 52) = 2.06$, $P = .157$, $\eta_p^2 = .04$, and group, $F(1, 52) = 0.72$, $P = .400$, $\eta_p^2 = .01$. However, there was a significant interaction between context and group, $F(1, 52) = 5.49$, $P = .023$, $\eta_p^2 = .10$. 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

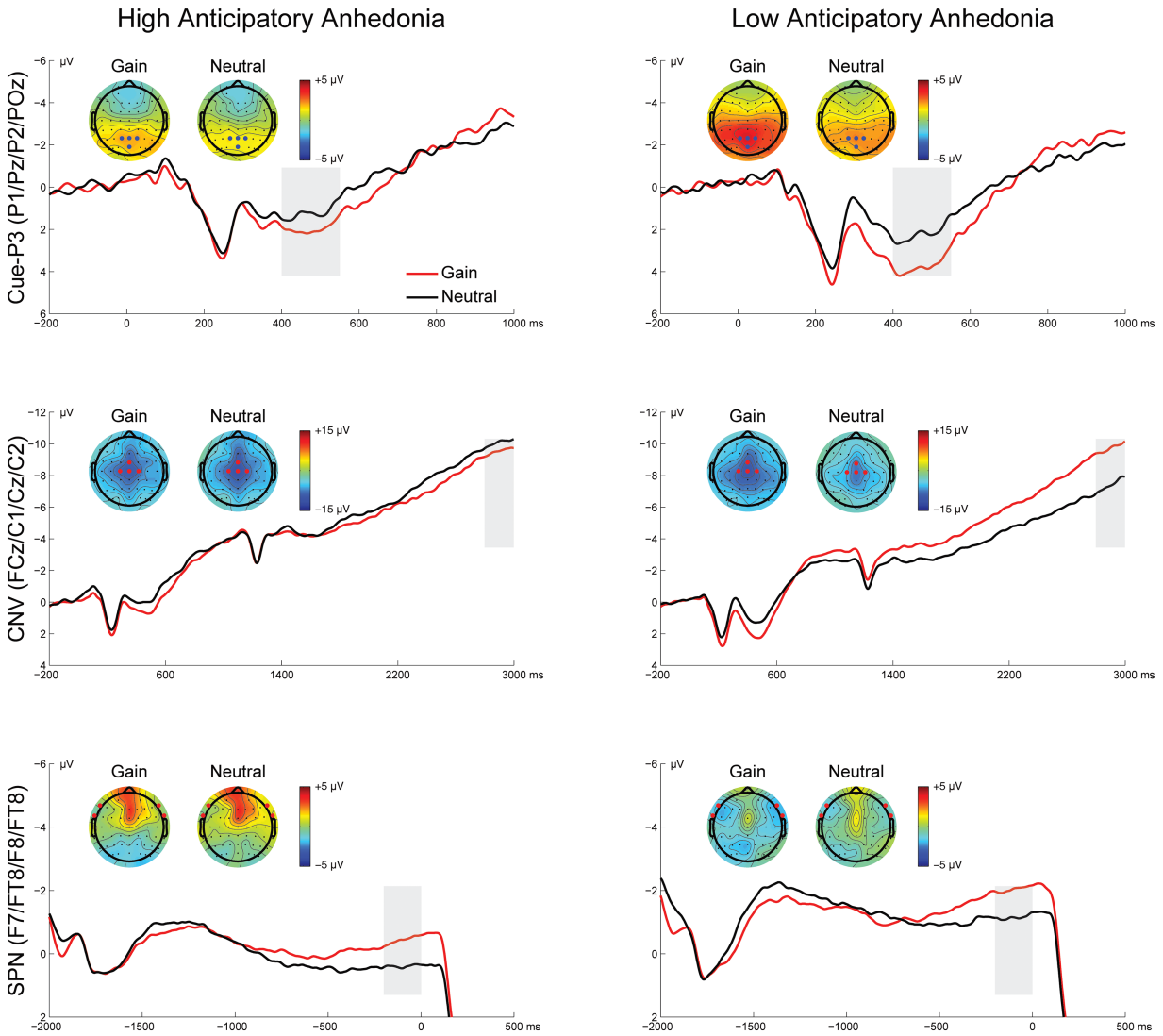

Fig. 2. Grand-averaged ERP waveforms as a function of group during the anticipation phase, where the shaded areas demarcate the time windows during which cue-P3 (400–550 ms), CNV (2800–3000 ms) and SPN (−200–0 ms) were scored. Topographical distribution maps for these ERP components are also shown.

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, $F(1, 52) = 9.53$, $P = .003$, $\eta_p^2 = .16$. Neither the main effect of group, $F(1, 52) = 2.80$, $P = .100$, $\eta_p^2 = .05$, nor the interaction effect between context and group, $F(1, 52) = 0.02$, $P = .902$, $\eta_p^2 < .01$, 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, $F(1, 52) = 10.28$, $P = .002$, $\eta_p^2 = .17$, with an enhanced (more negative) FRN for the gain *vs* neural context. Neither the main effect of group, $F(1, 52) = 1.44$, $P = .235$, $\eta_p^2 = .03$, nor the interaction between group and context, $F(1, 52) = 0.59$, $P = .445$, $\eta_p^2 = .01$, was significant (see Table S4 in Supplementary Materials for Bayesian statistics of the FRN data).[2] With regard to fb-P3, there was a significant main effect of context, $F(1, 52) = 38.20$, $P < .001$, $\eta_p^2 = .42$, indicating that fb-P3 was larger for the gain than neutral context. The main effect of group was also significant, $F(1, 52) = 4.07$, $P = .049$, $\eta_p^2 = .07$, 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, $F(1, 52) = 6.28$, $P = .015$, $\eta_p^2 = .11$. No other significant effects were obtained,

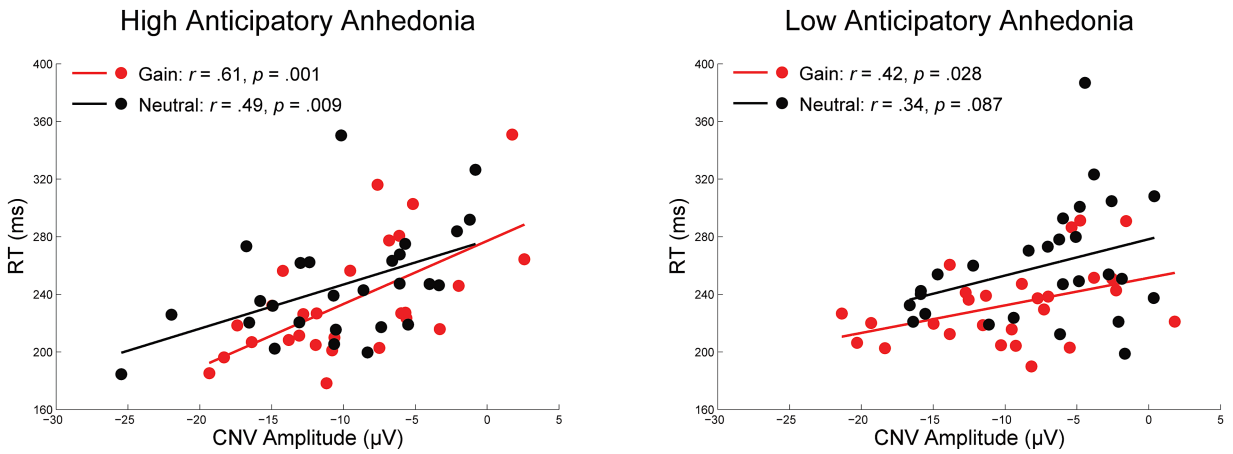

Fig. 3. Scatterplots of the correlations between CNV amplitudes and RTs as a function of group in gain and neutral contexts.

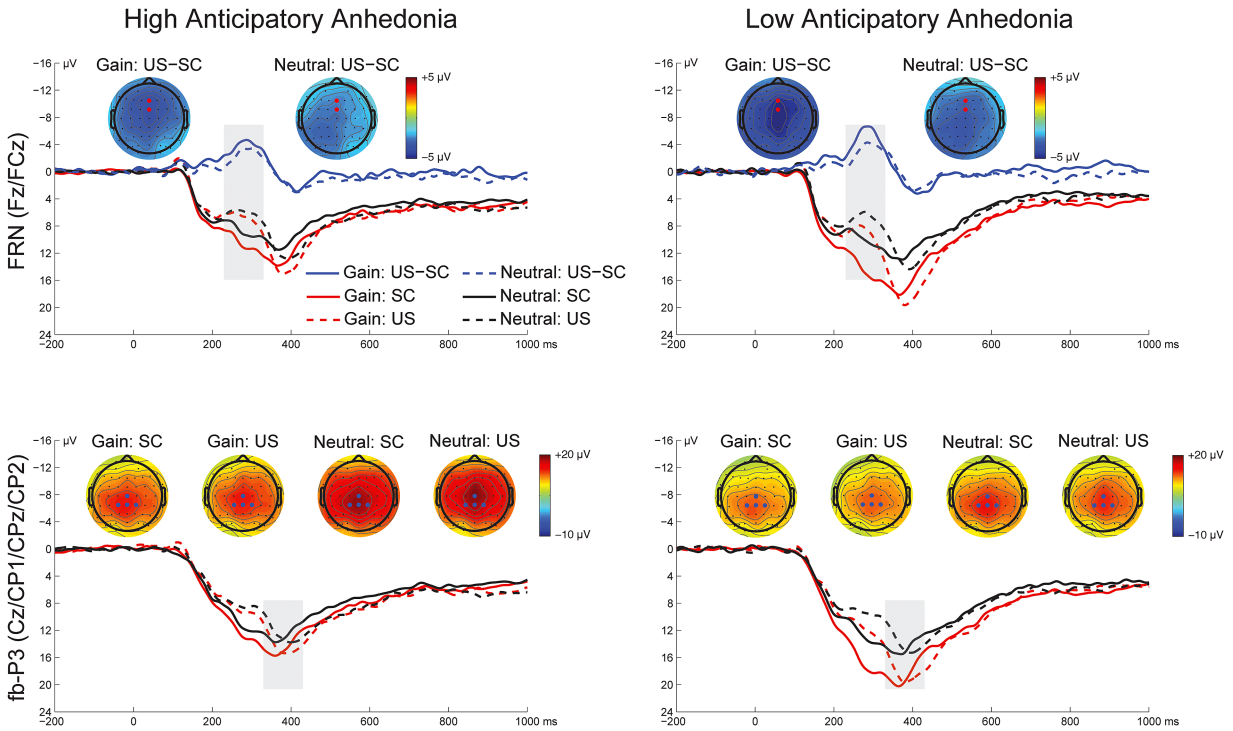

Fig. 4. Grand-averaged ERP waveforms as a function of group during the consummatory phase, where shaded areas demarcate the time windows during which the FRN (230–330 ms) and fb-P3 (330–430 ms) were scored. Topographical distribution maps for these ERP components are also shown. SC = successful; US =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

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. *$p < .05$. **$p < .01$. ***$p < .001$.

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 ERP paradigm (i.e. the MID task) could elicit, in both groups, reward-related ERP components as demonstrated in the literature. Consistent with previous research employing similar tasks (Broyd *et al.*, 2012; Santesso *et al.*, 2012; Pfabigan *et al.*, 2015; Novak *et al.*, 2016; Gu *et al.*, 2017; Zhang *et al.*, 2017), our results revealed that a series of ERP components from the anticipatory phase (i.e. cue-P3, CNV and SPN) to consummatory phase (i.e. the FRN and fb-P3) were greater in amplitude for the gain context than for the neutral context, indicating a widespread influence of reward on human neural circuits in terms of temporal dynamics (Glazer *et al.*, 2018). Interestingly, anticipatory anhedonia was specifically linked to only a subset of reward-related ERP components, instead of the entire reward dynamics.

The first ERP component modulated by anhedonia is the CNV, an ERP component associated with anticipatory and motivational attention for the upcoming stimulus, as well as preparation for the movement taking place simultaneously (Walter *et al.*, 1964; Tecce, 1972; Brunia *et al.*, 2012). In the present study, the LAA group showed a larger CNV in the gain than neutral context, reflecting their higher level of target anticipation of or response preparation for potential rewards. By contrast, this reward effect was not observed in the HAA group, as revealed by comparable CNV amplitudes across the gain and neutral contexts. These results are in line with an early study finding that anhedonic subjects in a non-clinical sample showed less differential CNV responses while anticipating interesting relative to neutral slides than did non-anhedonic subjects (Simons *et al.*, 1982). As shown in Figure 2, the lack of difference between contexts in the HAA group seemed to be caused by an enhanced CNV in anticipation of the neutral target, rather than a reduced CNV in response to the potential gain target. In support of this observation, correlation analyses revealed that although larger CNV amplitudes were associated with faster RTs in the gain context across groups, the relationship between CNV amplitudes and RTs in the neutral context showed a different pattern: whereas no significant correlation was found for the LAA group, subjects with HAA displayed a strong correlation between their CNV amplitudes and RTs, though the correlation coefficients were not significantly different across groups. These findings indicate that anticipatory anhedonia is more associated with greater performance, as evinced by RTs and the CNV, in the neutral context instead of the gain context. It seems that the HAA group is more intrinsically motivated and therefore not influenced by the context manipulation. However, we argued that anticipatory anhedonia is more associated with abnormal and inefficient response preparation for a target without reward incentive. Recent research highlights that anhedonia should be understood in terms of effort-based decision making for reward (Husain & Roiser, 2018; Pessiglione *et al.*, 2018) such that anhedonia is associated with reduced willingness to exert effort to obtain rewards (Treadway *et al.*, 2009; Geaney *et al.*, 2015). Our findings are compatible with this framework and further suggest that anticipatory anhedonia may be associated with a reduced ability to use incentive information to modulate effort allocation and decision-making processes in pursuit of goal-directed behaviors.

Interestingly, although the SPN was larger for the gain relative to neutral context, it was not modulated by anhedonia. The SPN is another anticipatory ERP component that reflects passive reward anticipation, which is similar to the CNV but free of motor preparation (Damen & Brunia, 1987; Brunia, 1988). Stimulus anticipation involved in the CNV (anticipating a visual target) may be different from that involved in the SPN (anticipating performance feedback). Considering its association with RTs, the CNV could reflect the motivational component, effort mobilization or task engagement (Schevernels *et al.*, 2014), whereas the SPN could be more linked to unresolved expectation and information processing (Brunia *et al.*, 2011). Our findings indicate that anticipatory anhedonia might be more associated with effort anticipation, rather than passive reward anticipation. Therefore, the discrepant effects of anhedonia on the two components may be not solely attributable to motor preparation, which could be clarified by examining readiness potential in anhedonia in the future study.

We adapted the MID task in the current study to exclude the confounding influences of outcome probability and visual characteristic on feedback processing (Mei *et al.*, 2018), which were not well controlled in previous ERP studies (Broyd *et al.*, 2012; Pfabigan *et al.*, 2015). We observed that fb-P3 was affected negatively by anticipatory anhedonia such that the amplitude of

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 also 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.

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