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Differentiating the abnormalities of social and monetary reward processing associated with depressive symptoms

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Original Article

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Abstract

Background. Reward dysfunction is a major dimension of depressive symptomatology, but it remains obscure if that dysfunction varies across different reward types. In this study, we focus on the abnormalities in anticipatory/consummatory processing of monetary and social reward associated with depressive symptoms.

Methods. Forty participants with depressive symptoms and forty normal controls completed the monetary incentive delay (MID) and social incentive delay (SID) tasks with event-related potential (ERP) recording.

Results. In the SID but not the MID task, both the behavioral hit rate and the ERP component contingent negative variation (CNV; indicating reward anticipation) were sensitive to the interaction between the grouping factor and reward magnitude; that is, the depressive group showed a lower hit rate and a smaller CNV to large-magnitude (but not small-magnitude) social reward cues compared to the control group. Further, these two indexes were correlated with each other. Meanwhile, the ERP components feedback-related negativity and P3 (indicating reward consumption) were sensitive to the main effect of depression across the MID and SID tasks, though this effect was more prominent in the SID task.

Conclusions. Overall, we suggest that depressive symptoms are associated with deficits in both the reward anticipation and reward consumption stages, particularly for social rewards. These findings have a potential to characterize the profile of functional impairment that comprises and maintains depression.

Introduction

Reward dysfunction plays a pivotal role in the pathogenesis and progression of depression (Hagele et al., 2015). Anhedonia, which refers to diminished pleasure and/or reactivity to normally rewarding activities, is a core feature of depression and a hallmark of major depressive disorder (MDD) (Greenberg et al., 2015; Keedwell, Andrew, Williams, Brammer, & Phillips, 2005; Treadway & Zald, 2011). Reward dysfunction in depression manifests as decreased motivation to seek for rewards and difficulties in experiencing positive affect, which may lead to physiological and behavioral problems such as reduced energy, loss of interest, loss of appetite, and reinforcement learning deficits (Bishop & Gagne, 2018; Eshel & Roiser, 2010; Forbes, Shaw, & Dahl, 2007; Kumar et al., 2008; Robinson, Cools, Carlisi, Sahakian, & Drevets, 2012; Rothkirch, Tonn, Kohler, & Sterzer, 2017). In the past two decades, the neural mechanisms of depression-induced reward dysfunction have been the focus of numerous studies (Sankar et al., 2019; Satterthwaite et al., 2015). Generally speaking, neuroimaging studies have revealed hyporesponsivity of brain reward systems (especially frontostriatal networks) in both resting state and task-dependent conditions among MDD patients (Epstein et al., 2006; McCabe, Cowen, & Harmer, 2009; Pizzagalli et al., 2009; Schaefer, Putnam, Benca, & Davidson, 2006) as well as healthy people at a high risk of depression (Forbes & Dahl, 2012; McCabe, Woffindale, Harmer, & Cowen, 2012; Monk et al., 2008). Parallel findings have also been discovered by event-related potential (ERP) research on depression (for a review, see Proudfit, 2015). Most notably, an ERP component feedback-related negativity (FRN; see below for details) becomes smaller among depressed individuals compared to healthy controls, which is understandable regarding the association of this component with dopaminergic reward activity (Foti & Hajcak, 2009; Nelson, Perlman, Klein, Kotov, &

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Hajcak, 2016; Proudfit, Bress, Foti, Kujawa, & Klein, 2015). However, it should be pointed out that some other studies have observed intact reward processing in depression (or in some of its specific subtypes; see Foti, Carlson, Sauder, & Proudfit, 2014; Moutoussis *et al.* 2018; Rutledge *et al.* 2017). To our knowledge, studies to date have predominantly utilized monetary reward paradigms (i.e. winning a nominal amount of money in laboratory tasks), which might preclude a broader understanding of anhedonia severity in depression across reward types (Ait Oumeziane, Jones, & Foti, 2019; Sharma et al., 2016).

The concept of 'reward' is not a homogeneous construct (Nestler & Carlezon, 2006; Sescousse, Caldu, Segura, & Dreher, 2013), including not only hedonic reinforcement (monetary and material gains) but also social information that has rewarding properties (e.g. social approval, social belonging, and social agency; see Ruff & Fehr, 2014). Although there are many other kinds of stimuli (e.g. food, drink, and shelter) that are regarded as rewards (Sescousse et al., 2013), this study focused on monetary and social rewards since they are most relevant in the depression literature (Forbes & Dahl, 2012; Henriques & Davidson, 2000). Monetary and social reward processing are phenomenally and neurologically divided (Goerlich et al., 2017; Gu et al., 2019; Morelli, Sacchet, & Zaki, 2015) and are related to distinct behavioral characteristics (e.g. response accuracy, reaction time, and subjective rating) in diverse age groups according to developmental studies (Hardin, Schroth, Pine, & Ernst, 2007; Jazbec et al., 2006). For instance, 12-month-old infants' tendency to explore ambiguous situations is regulated more strongly by social rewards (e.g. caregiver's smile) compared to non-social rewards (Sorce, Emde, Campos, & Klinnert, 1985), while the concept of money may not develop before the age of 5 (Kohls, Peltzer, Herpertz-Dahlmann, & Konrad, 2009). On the neural level, both monetary and social rewards activate the midbrain dopaminergic system (Izuma, Saito, & Sadato, 2008) and the prefrontal cortex (Lin, Adolphs, & Rangel, 2012), but the brain regions underlying social processes (e.g. empathizing) might also be needed for the encoding of social rewards, such as the temporoparietal junction (Liu et al., 2020; Strombach et al., 2015). Separating social from nonsocial rewards has been proven to be a valuable approach for clinical research (e.g. Gonzalez-Gadea *et al.* 2016; Hanewald *et al.* 2017). For example, Lee et al. (2019) recently found that compared to non-social rewards, the neural sensitivity to social rewards reduced to a greater extent among patients with schizophrenia. Research in this direction has clinical implications by highlighting potential targets for treatment (Forbes & Dahl, 2012). Because reward dysfunction and its related symptoms (e.g. anhedonia) are shared by diverse diagnostic categories (Husain & Roiser, 2018; Whitton, Treadway, & Pizzagalli, 2015), differentiating various kinds of rewards helps identify unique (*v.* common) alterations in depression and thus facilitates more targeted interventions (Ait Oumeziane et al., 2019).

Social rewards contribute significantly to human functioning and behavior (Fehr & Camerer, 2007; Gunaydin et al., 2014) and are critical to understanding the development of depression (Morgan, Olino, McMakin, Ryan, & Forbes, 2013; Olino, Silk, Osterritter, & Forbes, 2015). Severe expression of depression symptoms and syndromes have been linked to blunted response to social rewards and social feedback (i.e. social anhedonia; for a review, see Kupferberg, Bicks, & Hasler, 2016). Alterations in social reward processing reduce depressed individuals' motivation to engage in social interactions (Brinkmann, Franzen, Rossier, & Gendolla, 2014; He, Liu, Zhao, Elliott, & Zhang, 2019a), impair their social functioning (Hirschfeld et al., 2000; Kupferberg et al., 2016), and increase their vulnerability to social stress (Pegg et al., 2019). From a developmental perspective, Davey, Yucel, and Allen (2008) proposed that repeated failure or frustration of social rewards could lead to suppression of the reward system and eventually result in the emergence of depression in adolescence (see also Silk, Davis, McMakin, Dahl, & Forbes, 2012). Adolescents often deal with peer rejection, emotional failure, and social evaluative concerns with limited social skills (Steinberg & Morris, 2001). Under the influence of these negative experiences, some adolescents might become pessimistic about the possibility of receiving social rewards, and thus withdraw from social activities to avoid interpersonal stress (Silk et al., 2012). Since adolescence is a critical period for the development of brain reward circuitry (Spear, 2000), low anticipation of social rewards and its behavioral consequences (e.g. social avoidance) could significantly disturb this development process and further cause anhedonia in depression (Davey et al., 2008; Forbes & Dahl, 2012). To our knowledge, there is a paucity of research investigating the neural representations of social reward processing in depression. Some recent studies suggest that depression severity is associated with reduced striatal activity (Enneking et al., 2019; Sharma et al., 2016) and an attenuated FRN (Distefano et al., 2018; Klawohn, Burani, Bruchnak, Santopetro, & Hajcak, 2020; Pegg et al., 2019) in response to social rewards. Though these results show similar patterns with previous findings based on monetary reward (see above), more works should be done to examine whether the influence of depression on reward processing varies in its different stages.

The monetary incentive delay (MID) task, of which the reliability has been verified by numerous studies, is a classic paradigm for the research on reward processing in both healthy and clinical populations (for reviews, see Balodis & Potenza, 2014; Gu *et al.* 2019; Wilson *et al.* 2018). In each MID trial, participants first observe an incentive cue indicating the amount of potential reward (anticipation stage), then respond to a target stimulus as quickly as possible before the presentation of performance feedback (consumption stage; see Knutson et al., 2000, 2001 for details). One of its popular variants is the social incentive delay (SID) task, which provides socially relevant information (e.g. friendly faces) rather than monetary feedback (Rademacher et al., 2010; Spreckelmeyer et al., 2009). Both the MID and SID tasks have contributed significantly to depression research (Arrondo et al., 2015; Knutson, Bhanji, Cooney, Atlas, & Gotlib, 2008; Smoski, Rittenberg, & Dichter, 2011; Stringaris et al., 2015). In our opinion, comparing MID and SID data in the same sample would provide an opportunity to clarify: (1) whether depressed individuals' deficits in monetary and social reward processing show different patterns; (2) whether those differences appear in the anticipation or the consumption stage. The latter issue is worth noting considering that the neural correlates of reward anticipation and consumption differ to some extent (Liu, Hairston, Schrier, & Fan, 2011; Oldham et al., 2018). Therefore, it is possible that domain-specific effects associated with depression selectively modulate the neural mechanisms of reward processing in specific stages (anticipation *v.* consumption).

The current study relied on the ERP technique since its high temporal resolution enables recognizing sub-stages within both anticipatory and consummatory reward processing (Ait Oumeziane et al., 2019). This study focused on three ERP

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components to investigate anticipatory and consummatory reward processing, the significance of which have been confirmed by previous studies using both the MID and SID tasks (Ait Oumeziane, Schryer-Praga, & Foti, 2017, Flores et al., 2015; Greimel et al., 2018; Gu, Jiang, Kiser, Luo, & Kelly, 2017). Regarding the reward anticipation stage, we focused on the contingent negative variation (CNV), a sustained, negative-going component that reflects preparation processes for an event of interest (Rohrbaugh, Syndulko, & Lindsley, 1976; Walter, Cooper, Aldridge, McCallum, & Winter, 1964). This fronto-central distributed component is elicited by a preceding signal and returns to baseline when the targeted event occurs (for a review, see Kononowicz & Penney, 2016). In the MID/SID context, researchers have used the CNV during cue presentation to investigate the anticipation of the target stimulus (Novak & Foti, 2015 Novak, Novak, Lynam, & Foti, 2016). However, the sensitivity of the CNV to depression level is largely unclear. Regarding the consumption stage, we were most interested in two ERP components associated with outcome processing, that is, the FRN and the P3 (for a review, see San Martín, 2012). The FRN reaches its maximum approximately 200–300 ms after the onset of outcome feedback, being more negative-going for unfavorable (e.g. monetary losses) than favorable feedback (Gehring & Willoughby, 2002; Miltner, Braun, & Coles, 1997). As mentioned above, the FRN is widely considered as an ERP index of depression severity, such that it becomes less negative-going among depressed individuals compared to controls (Barch et al., 2019; Brush, Ehmann, Hajcak, Selby, & Alderman, 2018; Foti et al., 2014; Foti & Hajcak, 2009). Finally, we also examined the P3 component, a positive-going waveform of which the emergence follows the FRN (Polezzi, Sartori, Rumiati, Vidotto, & Daum, 2010; San Martin, Appelbaum, Pearson, Huettel, & Woldorff, 2013). This component has been associated with various cognitive functions across different research fields (Polich, 2007; Polich & Criado, 2006). Compared to the FRN, researchers suggest that the P3 component reflects a more deliberate stage of information integration (Gu et al., 2011; Wu & Zhou, 2009). A majority of studies reveal that the P3 amplitude is negatively correlated with depression level (Gangadhar, Ancy, Janakiramaiah, & Umapathy, 1993; Karaaslan, Gonul, Oguz, Erdinc, & Esel, 2003; Urretavizcaya et al., 2003), though some others disagree (Feng et al., 2015; Zhang, He, Chen, & Wei, 2016; Zhang, Xie, He, Wei, & Gu, 2018).

In this research, we recruited participants with depressive symptoms (as well as normal controls) to examine their task performance and ERP patterns in both the MID and SID tasks. According to previous findings (see above), we expected to observe attenuated CNV, FRN, and P3 among individuals with depressive symptoms, indicating deficits in both reward anticipation and reward consumption. More importantly, we directly compared the effect of depression between the MID and SID tasks, so as to explore whether reward dysfunction associated with depressive symptoms is domain-general or domain-specific, and to determine whether the domain-specific effects (social *v.* nonsocial) occur in the anticipation or the consumption stage.

Methods

Participants

In a mental health screening of Shenzhen University, the Self-Rating Depression Scale (SDS: Zung, 1965) and the Beck Depression Inventory Second Edition (BDI-II: Beck, Steer, and Brown, 1996) were administered to approximately 2000 undergraduate students. The norms of the SDS suggest that a score >0.5 (range = 0.25–1.0) indicates mild depression, while those of BDI-II suggest that a score >13 (range = 0–63) indicates mild depression. Accordingly, individuals who scored higher than 0.5 on the SDS and higher than 13 on the BDI-II were determined as having a mild depressive state. The key exclusion criterion was any lifetime Axis I disorders other than depression according to Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Non-Patient Edition (SCID-I/NP: First, Gibbon, & Spitzer, 2002). Healthy controls were also recruited from the original sample, who had scores of SDS < 0.5, BDI-II < 13, and were screened with SCID-I/NP to exclude any lifetime Axis I disorders including depression. For all the participants, the exclusion criteria also included: (1) seizure disorder; (2) a history of head injury with possible neurological sequelae; (3) self-reported prior use of any psychoactive drugs especially medication for depression; (4) current alcohol or drug dependence.

Among the students who met the above criteria, 80 individuals (40 with mild depression and 40 controls) were invited to participate in the formal experiment. All participants were medical-free at the time of the experiment. As shown in Table 1, no significant difference was found between these two groups with respect to gender, age, and handedness. Written informed consent was obtained prior to the experiment. The study was approved by the Ethics Committee of Shenzhen University.

Self-reported measures

On the day of the experiment, each participant was required to complete four questionnaires: (1) the BDI-II measures depressive symptoms, with a high score indicating a high level of depressive tendency (range = 0–63); (2) the Trait form of Spielberger's State-Trait Anxiety Inventory (STAI-T: Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) measures trait anxiety, with a high score indicating a high level of trait anxiety (range = 20–80); (3) the Revised Social Anhedonia Scale (RSAS: Eckblad, Chapman, Chapman, & Mishlove, 1982) measures social anhedonia, with a higher score indicating less enjoyment from and need for social contact (range = 0–40); (4) the 'Sensitivity to Reward' subscale of the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ: Torrubia, Avila, Moltó, & Caseras, 2001) measures reward sensitivity, with a higher score indicating a higher level of sensitivity to reward (range = 0–24). The results of these measures from the two groups (on the day of the experiment) are reported in Table 1.

Procedure and experimental design

The current study relied on the electroencephalography (EEG) technique since its high temporal resolution enables recognizing sub-stages within both anticipatory and consummatory reward processing (Ait Oumeziane et al., 2019). Prior to EEG recording, the individual threshold of visual reaction time was assessed with a simple reaction time task (averaged across 40 trials) to set up the difficulty level of the formal task for each participant.

The formal task consisted of two MID and two SID blocks, the sequence of which was randomized across participants. In both the MID and SID blocks (Fig. 1), each trial began with a cue (i.e. anticipation stage) indicating a small ('I') or large ('II') amount of potential reward for 500 ms, followed by a delay for

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Table 1. Demographic characteristics of the participants (mean & standard deviation)

Items	Control group ($n = 40$)	Depressive group ($n = 40$)	Picture rating ($n = 40$)	Control *v.* Depressive	Control *v.* Picture rating
Gender (male/female)	21/19	20/20	20/20	$\chi^2 = 0.8$, $p = 1.000$	$\chi^2 = 0.8$, $p = 1.000$
Age (years)	21.3 (1.8)	20.6 (1.9)	21.6 (1.7)	$t = 1.6$, $p = 0.103$	$t = -0.6$, $p = 0.523$
Handedness, right/left	40/0	40/0	40/0		
BDI-II	2.5 (2.4)	19.4 (7.7)	3.2 (2.4)	$t = 13.3$, $p < 0.001$***	$t = -1.3$, $p = 0.195$
STAI-T	35.0 (6.3)	50.9 (9.3)	37.5 (9.2)	$t = 9.9$, $p < 0.001$***	$t = -1.4$, $p = 0.155$
RSAS	10.1 (5.4)	14.4 (6.3)	9.8 (5.6)	$t = 3.3$, $p = 0.001$**	$t = 0.2$, $p = 0.824$
SR	13.9 (3.6)	13.5 (3.2)	14.4 (3.8)	$t = -0.6$, $p = 0.535$	$t = -0.5$, $p = 0.586$

BDI-II, the Beck Depression Inventory Second Edition; STAI-T, the Trait form of Spielberger's State-Trait Anxiety Inventory; RSAS, the Revised Social Anhedonia Scale; SR, the subscale of Sensitivity to Reward of the Sensitivity to Punishment and Sensitivity to Reward Questionnaire. *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

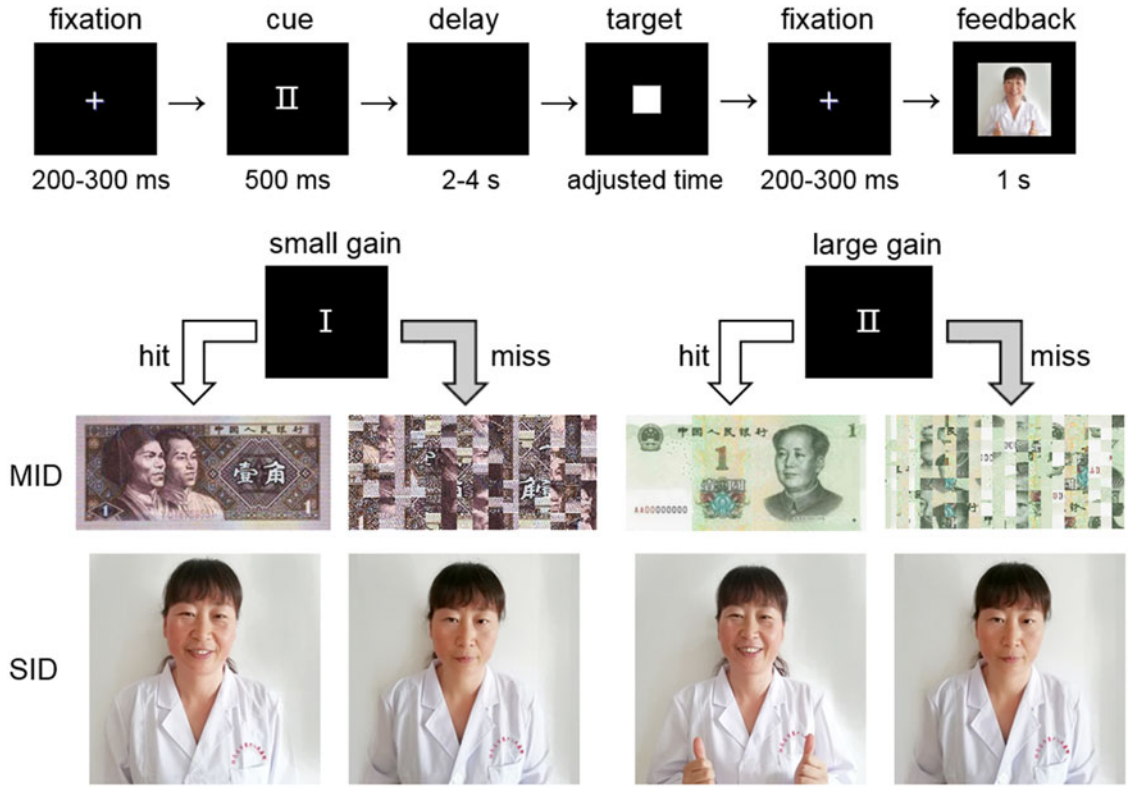


Fig. 1. Illustration of the experimental design. Upper panel: an exemplar trial of the social incentive delay (SID) task. Lower panel: the correspondence between cues and feedback (for hits or misses) in the monetary incentive delay (MID) and SID tasks.

a random duration ranging from 2 to 4 s. After that, participants were required to press the space button on the keyboard as quickly as possible upon the presentation of a white square (target) to gain reward. If participants' response time was shorter than the duration of target presentation, the ongoing trial would be labeled as a 'hit' trial; otherwise, it would be labeled a 'miss' trial. The presentation time of the target was initially set according to the average performance in the simple reaction time task (see above) for each participant and then was slightly adjusted (±10 ms) in a trial-by-trial manner so as to keep the hit rate at approximately 50% (Ait Oumeziane et al., 2017; Landes et al., 2018). After responding to the target, each participant received monetary or social feedback (i.e. consumption stage) for 1 s (see below for details). Each MID or SID block consisted of 40 small-reward trials and 40 large-reward trials. Inter-trial interval was 1 s. The formal task (320 trials in total) lasts for approximately 35 min.

Regarding the MID blocks, participants received a picture of ¥0.1 Chinese yuan (approximately US$0.015) after successfully hitting the target but a scrambled picture of ¥0.1 (indicating no monetary gain) after missing that target in each trial of the small-reward condition; meanwhile, the feedback was a picture (or scrambled picture) of ¥1 (approximately $0.15) in the large-reward condition. Regarding the SID blocks, participants received a picture showing a person with a smiling face after

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Table 2. 'Liking' and 'wanting' ratings of rewarding pictures on a 1-to-7 point scale (mean & standard deviation)

Item	Small monetary	Large monetary	Small social	Large social
Liking score	2.89 (1.67)	3.55 (1.84)	3.21 (1.44)	3.81 (1.71)
Wanting score	2.28 (1.85)	3.28 (2.05)	2.66 (1.37)	3.31 (1.86)

A high score indicates a high level of liking or wanting.

they successfully hit the target but a person with a neutral facial expression after they missed that target in each trial of the small-reward condition; meanwhile in the large-reward condition, feedback for hits was a picture showing a person who smiled and gave a thumbs up while feedback for misses was a person with neutral facial expression.

There were four sets of pictures for both the MID (paper or coin money in its front or backside) and SID (two actors and two actresses) blocks. Forty individuals with a low level of depressive tendency (Table 1), who were not involved in the formal experiment, were invited to assess the 'liking' and 'wanting' ratings of the rewarding pictures on a 1-to-7 point scale (Table 2). Their results showed that the 'liking' and 'wanting' scores of these pictures were only sensitive to the *reward magnitude* factor (large > small: $F_{(1,39)} = 34.7$, $p < 0.001$ for 'liking' rating; $F_{(1,39)} = 60.5$, $p < 0.001$ for 'wanting' rating), whereas neither *reward category* (monetary *v.* social) nor the interaction of two factors was significant ($Fs \leqslant 2.1$, $p \geqslant 0.151$).

Each participant was encouraged to maximize his/her performance prior to the task. For the MID blocks, his/her monetary gains in 'hit' trials were accumulated into the final payment; for the SID blocks, he/she was told that task performance was online assessed by strangers in the picture with an assumption that successfully hitting the target indicates a high level of cognitive ability. Unbeknownst to each participant, these strangers were four volunteers who provided consent for their portraits to be used for scientific purposes. After the whole task, each participant was asked if he/she believed that his/her cognitive ability was evaluated by other persons during the SID blocks. All the participants reported that they believed in the cover story.

EEG recording and analysis

Brain electrical activity was recorded by a 32-channel wireless amplifier with a sampling frequency of 250 Hz (NeuSen.W32, Neuracle, Changzhou, China). Data were on-line recorded referentially against left mastoid and off-line re-referenced to average activities over the scalp. EEG data were collected with electrode impedances kept below 10 kΩ. Ocular artifacts were removed from EEGs using a regression procedure implemented in NeuroScan software (Scan 4.3: NeuroScan, Inc., Herndon, VA).

This study focused on the anticipation and the consumption stage of reward processing, corresponding to the ERPs evoked by MID/SID cues and feedback. The recorded EEG data were filtered (half-amplitude cutoff: 0.1–30 Hz) and segmented beginning 200 ms prior to stimulus onset. The cue-evoked ERP epochs lasted from −200 ms to 2.5 s while the feedback-evoked epochs lasted from −200 ms to 800 ms. All epochs were baseline-corrected with respect to the mean voltage over the 200 ms preceding stimulus onset, followed by averaging for each experimental condition. Epochs containing artifacts with peak-to-peak deflection exceeding ± 100 μV were rejected.

The electrode sites and time window for each ERP component were selected before data analysis based on prior knowledge. For cue-evoked ERPs, this study focused on the CNV elicited by small ('I') and large-reward cues ('II') in the MID and SID blocks. The mean amplitude of the CNV was calculated using the arithmetic average of the electrode sites in the mid-frontocentral area (including Fz, FCz, FC1, and FC2) within a time window of 750–2500 ms post cue onset (Chronaki, Soltesz, Benikos, & Sonuga-Barke, 2017). For feedback-evoked ERPs, we focused on the FRN and P3 elicited by feedback for hits or misses in the MID and SID blocks. The mean amplitude of the FRN was calculated using the arithmetic average, also at electrode sites in the mid-frontocentral area (i.e. Fz, FCz, FC1, and FC2), within a time window of 200–300 ms post feedback onset (Gu et al., 2017; Zhu, Wang, Gao, & Jia, 2019). Finally, the mean amplitude of the P3 was calculated using the arithmetic average at electrode sites in the mid-centroparietal area (i.e. Pz, P3, P4, CP1, and CP2) within a time window of 200–400 ms post feedback onset (Greimel et al., 2018).

Since an ERP experiment usually has many measurements (e.g. the amplitude and latency of multiple ERP components on multiple electrode sites), the heightened likelihood of false-positive findings is a potential issue for statistical analysis; for this concern, reducing the number of factors in a given ANOVA (i.e. dimensionality reduction) would be helpful (Luck & Gaspelin, 2017). For this purpose, this study used the difference wave approach to measure the FRN amplitude, that is, the FRN evoked by neutral feedback (miss trials) minus the FRN evoked by positive feedback (hit trials) (Hajcak, Moser, Holroyd, & Simons, 2007; Holroyd & Krigolson, 2007; Holroyd, Pakzad-Vaezi, & Krigolson, 2008). In the same vein, this study measured the P3 amplitude as the difference wave (Kogler, Sailer, Derntl, & Pfabigan, 2017; Lei et al., 2019), that is, the P3 evoked by positive feedback (hit trials) minus the P3 evoked by neutral feedback (miss trials). Using this approach to measure feedback-related ERPs may also help removing the potential influence of unfamiliarity (Becker, Smith, & Schenk, 2017; Conde, Goncalves, & Pinheiro, 2015; Cycowicz, 2019): in our opinion, it was possible that unfamiliarity has affected feedback processing in both hit trials and miss trials of the SID because participants were unfamiliar with strangers' faces (while they were supposed to be familiar with monetary pictures).

Statistics

Descriptive data were presented as mean ± standard deviation, unless otherwise mentioned. The significance level was set at 0.05. Repeated-measures ANOVA was performed on behavioral and ERP measurements, with *reward category* (monetary *v.* social) and *reward magnitude* (small *v.* large) as the two within-subject factors, and *group* (depressive *v.* control) as the between-subjects factor. Regarding the strong relationship between anxiety and depression (Stavrakaki & Vargo, 1986), and that individual anxiety level might also influence social and monetary reward processing (Bishop & Gagne, 2018; Luo et al., 2014), we

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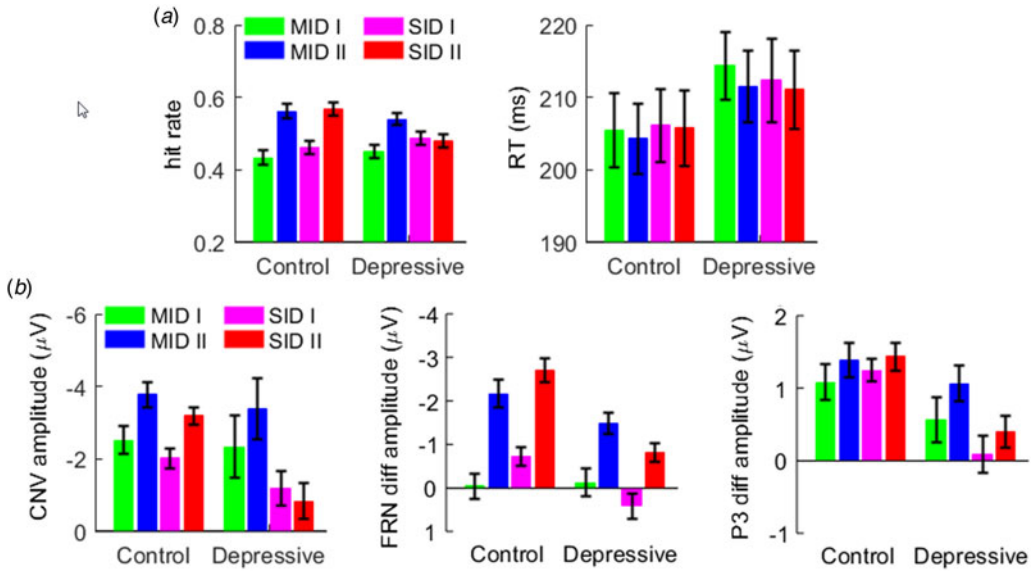


Fig. 2. The results of behavioral and event-related potential indexes. (*a*) The hit rate and reaction time in the depressive and control groups. (*b*) The amplitudes of the contingent negative variation (CNV), feedback-related negativity (FRN), and P3 in two groups. Error bars indicate one standard error.

considered the trait anxiety score (measured by STAI-T) as a covariate. Significant interactions were analysed using simple effects model.

Results

Behavioral data

Hit rate

The main effect of *reward magnitude* was significant ($F_{(1,77)}$ = 14.4, $p < 0.001$, η_p^2 = 0.157): the hit rate in the large-reward condition (0.537 ± 0.115) was higher than that in the small-reward condition (0.458 ± 0.118). The interaction of *reward magnitude* × *group* ($F_{(1,77)}$ = 18.5, $p < 0.001$, η_p^2 = 0.194) was also significant: while large rewards were associated with a higher hit rate in the control group compared to the depressive group ($F_{(1,77)}$ = 4.0, $p = 0.048$, η_p^2 = 0.050; control *v.* depressive = 0.562 ± 0.116 *v.* 0.512 ± 0.108), this group difference did not reach significance in the small-reward condition ($F < 1$; 0.451 ± 0.123 *v.* 0.466 ± 0.112).

Finally, the three-way interaction of *reward category* × *reward magnitude* × *group* was significant ($F_{(1,77)}$ = 5.3, $p = 0.024$, η_p^2 = 0.064; Fig. 2*a*). To break down this three-way interaction, we tested the two-way interaction of *reward magnitude* × *group* in the MID and SID blocks separately. In the MID blocks, only the main effect of *reward magnitude* was significant ($F_{(1,77)}$ = 7.7, $p = 0.007$, η_p^2 = 0.091; small *v.* large reward = 0.442 ± 0.118 *v.* 0.551 ± 0.111); meanwhile in the SID blocks, not only the main effect of *reward magnitude* ($F_{(1,77)}$ = 6.53, $p = 0.013$, η_p^2 = 0.078) but also the interaction of *reward magnitude* × *group* ($F_{(1,77)}$ = 33.5, $p < 0.001$, η_p^2 = 0.303) were significant: while large social rewards were associated with a higher hit rate in the control group compared to the depressive group ($F_{(1,77)}$ = 7.9, $p = 0.006$, η_p^2 = 0.093; control *v.* depressive = 0.564 ± 0.113 *v.* 0.483 ± 0.108), this group difference did not reach significance in the small-social-reward condition ($F < 1$; 0.464 ± 0.118 *v.* 0.486 ± 0.113).

Reaction time (RT)

The RT was averaged across hit trials in each condition (He, Zhang, Muhlert, & Elliott, 2019b). We defined the outlier threshold as mean ± 1.96 standard deviation (95% confidence interval). According to this criterion, two participants per group were removed from the datasets as outliers. After these outliers were removed, the RT data were normally distributed (i.e. all the *p* values of the K-S tests were larger than 0.05). For the outlier-removed RT datasets (n = 38 per group), the main effect of *reward magnitude* was marginally significant ($F_{(1,73)}$ = 3.91, $p = 0.052$, η_p^2 = 0.050): the RT for large rewards (213.5 ± 22.7 ms) showed a tendency to be shorter than that for small rewards (214.8 ± 23.7 ms). No other main or interaction effects were significant (Fig. 2*a*).

ERPs

Cue-evoked CNV

The main effect of *reward magnitude* was significant ($F_{(1,77)}$ = 7.20, $p = 0.009$, η_p^2 = 0.086): the CNV following large-reward cues (−2.80 ± 3.58 μV) was larger (i.e. more negative-going) than that following small-reward cues (−2.02 ± 3.53 μV).

The three-way interaction of *reward category* × *reward magnitude* × *group* was significant ($F_{(1,77)}$ = 4.14, $p = 0.045$, η_p^2 = 0.051; Figs 2b and 3). To break down this three-way interaction, we tested the two-way interaction of *reward magnitude* × *group* in the MID and SID blocks separately. In the MID blocks, only the main effect of *reward magnitude* was significant ($F_{(1,77)}$ = 5.76, $p = 0.019$, η_p^2 = 0.070; small *v.* large reward = −2.43 ± 4.24 *v.* −3.58 ± 4.02 μV). However in the SID blocks, not only the main effects of *group* ($F_{(1,77)}$ = 6.92, $p = 0.010$, η_p^2 = 0.083; control *v.* depressive = −2.63 ± 1.71 *v.* −0.99 ± 3.08 μV), but also the interaction of *reward magnitude* × *group* were significant ($F_{(1,77)}$ = 5.83, $p = 0.018$, η_p^2 = 0.070): while large-reward cues evoked a larger CNV in the controls than in the depressive group ($F_{(1,77)}$ = 10.9, $p = 0.001$, η_p^2 = 0.124; control *v.* depressive = −3.14 ± 1.49 *v.* −0.89 ± 3.19 μV), this group difference did not reach

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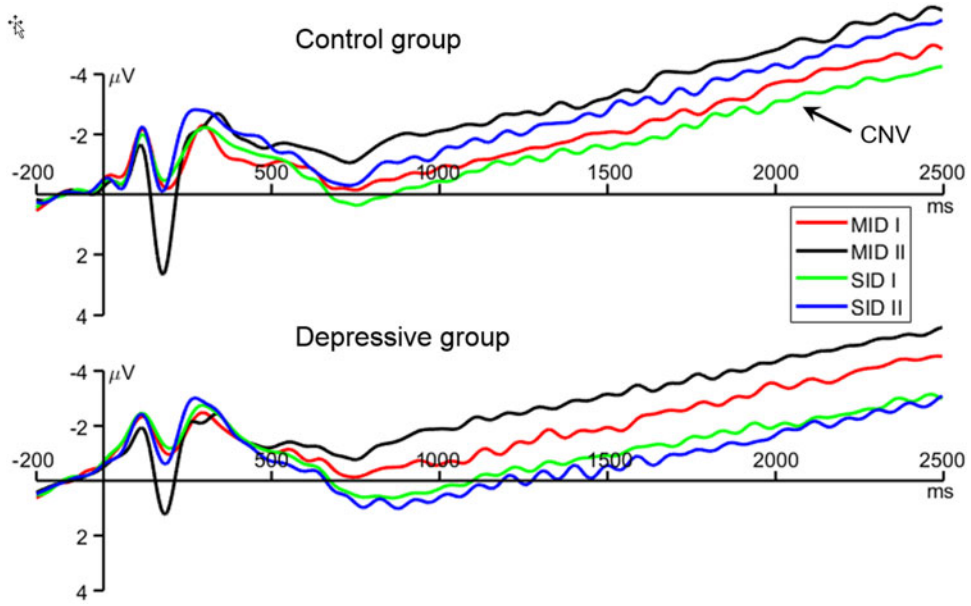


Fig. 3. Grand-average waveforms of the contingent negative variation (CNV). MID: monetary incentive delay task; SID: social incentive delay task; I: small cue; II: large cue. The waveforms were averaged across the electrode sites of Fz, FCz, FC1, and FC2.

Table 3. Correlation between hit rate and CNV amplitude ($n = 40$).

	Small monetary		Large monetary		Small social		Large social	
Group	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*
Control	0.496	0.001**	0.544	0.000***	0.335	0.035*	0.595	0.000***
Depressive	0.538	0.000***	0.648	0.000***	0.491	0.001**	0.637	0.000***

*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

significance for small-reward cues ($F_{(1,77)} = 2.46$, $p = 0.121$, $\eta_p^2 = 0.031$; -2.13 ± 1.73 *v.* $-1.08 \pm 2.99\,\mu$V).

Seeing that the results of hit rate and CNV amplitude showed a similar pattern (specifically, the *reward category* × *reward magnitude* × *group* interaction), we tested the two-tailed Pearson correlation between these two indexes. This analysis showed that the hit rate and CNV amplitude was positively correlated in each condition ($r = 0.335 \sim 0.648$, $p = 0.035 \sim 0.0005$; Table 3).

Feedback-evoked FRN

The FRN was measured as the mean amplitude of the difference wave between miss and hit trials (see the Methods section). The main effect of *group* was significant ($F_{(1,77)} = 15.0$, $p < 0.001$, $\eta_p^2 = 0.163$): the FRN amplitude was larger (i.e. more negative-going) in the control group ($-1.65 \pm 2.04\,\mu$V) than in the depressive group ($-0.26 \pm 1.85\,\mu$V). The main effect of *reward magnitude* was significant ($F_{(1,77)} = 12.1$, $p = 0.001$, $\eta_p^2 = 0.136$): the FRN following large rewards ($-1.79 \pm 1.81\,\mu$V) was larger than that following small rewards ($-0.12 \pm 1.82\,\mu$V).

The interaction of *reward category* × *group* was significant ($F_{(1,77)} = 10.5$, $p = 0.002$, $\eta_p^2 = 0.120$; Figs 2*b* and 4*a*). While social rewards evoked a larger FRN in the controls than in the depressive group ($F_{(1,77)} = 33.1$, $p < 0.001$, $\eta_p^2 = 0.301$; control *v.* depressive $= -2.09 \pm 1.83$ *v.* $0.17 \pm 1.70\,\mu$V), this group difference did not reach significance for monetary rewards ($F_{(1,77)} = 1.02$, $p = 0.316$, $\eta_p^2 = 0.013$; -1.20 ± 2.20 *v.* $-0.70 \pm 1.95\,\mu$V). Finally, the interaction of *reward magnitude* × *group* was significant ($F_{(1,77)} = 4.26$, $p = 0.042$, $\eta_p^2 = 0.052$; Figs 2*b* and 4*a*). While the FRN amplitude was larger (i.e. more negative-going) in the control group than in the depressive group, this group effect was more significant in the large-reward condition ($F_{(1,77)} = 20.6$, $p < 0.001$, $\eta_p^2 = 0.211$; control *v.* depressive $= -2.64 \pm 1.87$ *v.* $-0.94 \pm 1.52\,\mu$V) than in the small-reward condition ($F_{(1,77)} = 7.16$, $p = 0.009$, $\eta_p^2 = 0.085$; -0.66 ± 1.67 *v.* $0.42 \pm 1.93\,\mu$V).

Feedback-evoked P3

The P3 was measured as the mean amplitude of the difference wave between hit and miss trials. The main effect of *group* was significant ($F_{(1,77)} = 7.78$, $p = 0.007$, $\eta_p^2 = 0.092$): outcome feedback evoked a larger (i.e. more positive-going) P3 amplitude in the control group ($1.42 \pm 1.35\,\mu$V) than in the depressive group ($0.40 \pm 1.68\,\mu$V).

Furthermore, the interaction of *reward category* × *group* was significant ($F_{(1,77)} = 4.66$, $p = 0.034$, $\eta_p^2 = 0.057$; Figs 2*b* and 4*b*). While social rewards evoked a larger P3 in the controls than in the depressive group ($F_{(1,77)} = 16.1$, $p < 0.001$, $\eta_p^2 = 0.173$; control *v.* depressive $= 1.50 \pm 1.10$ *v.* $0.08 \pm 1.51\,\mu$V), this group effect did not reach significance for monetary rewards ($F_{(1,77)} = 1.82$, $p = 0.182$, $\eta_p^2 = 0.023$; 1.34 ± 1.56 *v.* $0.72 \pm 1.79\,\mu$V).

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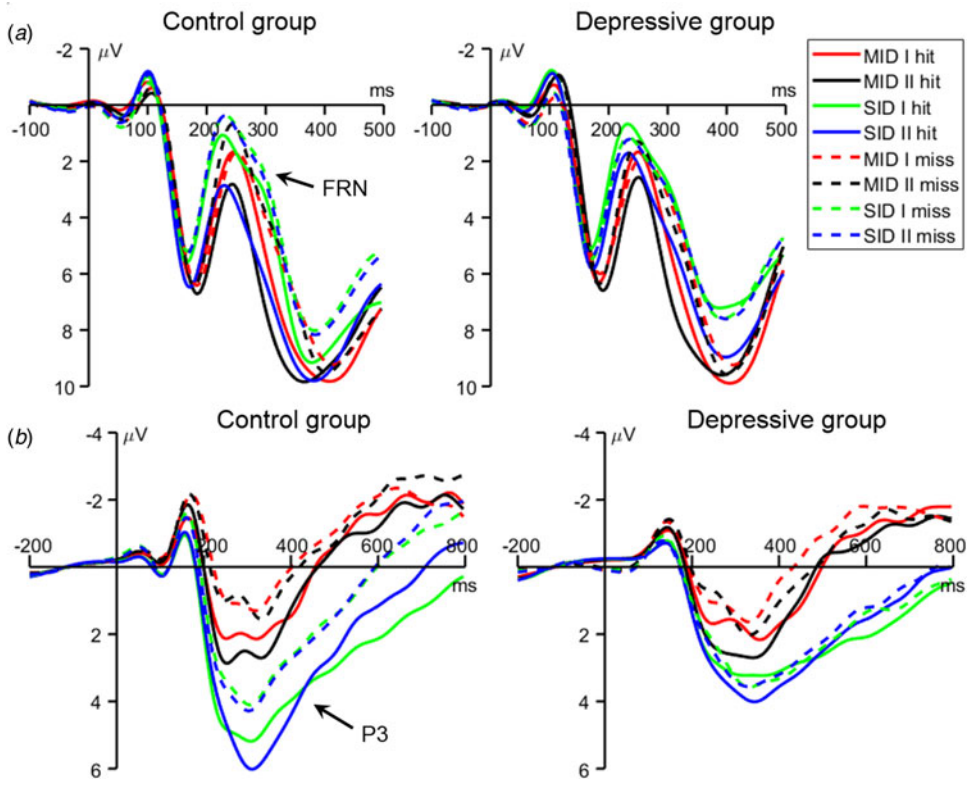


Fig. 4. Grand-average waveforms of the feedback-related negativity (FRN; upper panels) averaged across the electrode sites of Fz, FCz, FC1, and FC2, and the P3 (lower panels) averaged across the electrode sites of Pz, P3, P4, CP1, and CP2.

Discussion

To clarify whether reward dysfunction associated with depressive symptoms is general across different reward types or domain-specific, we analysed the behavioral and ERP data from the MID and SID tasks. At the behavioral level, there was a significant three-way *reward category* × *reward magnitude* × *group* interaction, such that the depressive group showed a lower hit rate when anticipating large (but not small) social rewards compared to the controls, while there was no between-group difference when anticipating monetary rewards. The same pattern was replicated on the ERPs in the anticipation stage, such that the CNV elicited by large (but not small) social reward cues was smaller in the depressive group than the controls. Finally, the FRN and P3 elicited by the consumption of both monetary and social rewards were generally weaker in the depressive group than the controls, though the group difference only reached significance for social rewards. These findings indicate potentially domain-specific and domain-general deficits associated with depressive symptoms in reward processing.

While the CNV has been related to various cognitive processes (for reviews, see Macar & Vidal, 2004; van Rijn, Kononowicz, Meck, Ng, & Penney, 2011), most researchers agree that the main function of this component is to prepare for an upcoming internal or external stimulus (see the Introduction; see also Kononowicz & Penney, 2016). In the current study, participants with depressive symptoms showed a smaller CNV in the anticipation stage of large social rewards compared to the controls, indicating that they were not well-prepared to seek for that kind of reward. This interpretation has been strongly supported by our behavioral results, such that the CNV amplitude increased as a function of the hit rate (see also Boehm, van Maanen, Forstmann, & van Rijn, 2014; Elbert, Ulrich, Rockstroh, & Lutzenberger, 1991), and that the hit rate for large social rewards was lower in the depressive group compared to the control group. Meanwhile, the small social reward condition was insensitive to depression level, possibly reflecting a 'floor effect' (see Yang, Zhou, Gu, & Wu, 2020 for similar results). In the reward anticipation stage, people should hold in mind their primary goals and the representation of future events (Davey et al., 2008). Our CNV results indicate that depressive symptoms are related to difficulties in conceiving of large social rewards, which could explain their reduced engagement in positive social activities (Setterfield, Walsh, Frey, & McCabe, 2016).

Social context plays a pivotal role in the development and maintenance of depressive disorders including the MDD (Sankar et al., 2019; Sheeber, Hops, & Davis, 2001). Depressive symptoms in youth are often caused by negative life events, including childhood maltreatment, peer rejection, and unfavorable social evaluation (Kendler, Hettema, Butera, Gardner, & Prescott, 2003; Monroe, Rohde, Seeley, & Lewinsohn, 1999; Nolan, Flynn, & Garber, 2003; Prinstein & Aikins, 2004; Silk et al., 2012). Over time, these social experiences could lead to interpersonal concerns and diminished pleasure from social interactions, which then precipitate and exacerbate depression (Allen et al., 2006; Allen & Badcock, 2003; Lee, Hankin, & Mermelstein, 2010; Slavich, Tartter, Brennan, & Hammen, 2014). As pointed out by Davey et al. (2008), depressive episodes often result from the omission of anticipated social rewards; when a person predicts that his/her endeavors would be unsuccessful in achieving interpersonal goals, he/she may reduce the risk of

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wasting resources by withdrawing from the pursuit of social rewards. This idea explains why impairment in social functioning is a prominent feature of depression (Badcock & Allen, 2003), and why social withdrawal (a key symptoms of depression) is related to alterations in seeking social rewards and experiencing positive affect in anticipation of those rewards (Forbes, 2009; Hsu et al., 2015; Silk et al., 2012). Thus, Pegg et al. (2019) suggested that compared to monetary reward, maladaptive responses to social reward is more relevant to understand anhedonia associated with depression (see also Copeland, Wolke, Angold, & Costello, 2013; George, Blazer, Hughes, & Fowler, 1989; Slavich, Thornton, Torres, Monroe, & Gotlib, 2009). That said, experimental data supporting the relationship between depression and social reward anticipation are still limited (Ait Oumeziane et al., 2019; Pegg et al., 2019). Our CNV findings could be considered as neurophysiological evidence that the anticipation of social reward among depressed individuals is anomalous, confirming the importance of this stage of social reward processing to depressive symptoms; meanwhile, the anticipation of monetary reward might be relatively intact. In line with our observation, Pizzagalli et al. (2009) reported that the effect of depression was much weaker in the anticipation stage than in the consumption stage during monetary reward processing. Further research could compare the CNV pattern between unipolar and bipolar depression since previous studies have reported dissociable differences in social valuation/processing between these two depressive disorders (Ehnvall et al., 2014; Ng & Johnson, 2013; Sharma et al., 2016).

In addition, both the FRN and P3 were smaller in the depressive group compared to the controls, regardless of whether these components were elicited by monetary or social feedback (i.e. the main effect of the group). In our opinion, these results reveal a general deficit of reward sensitivity spanning monetary and social conditions, which is in line with previous literature that emphasizes the significance of blunted reward responsiveness to depression (Admon & Pizzagalli, 2015; Keren et al., 2018; Pechtel, Dutra, Goetz, & Pizzagalli, 2013; Rolls, 2016). However, it is important to note that the interaction effect further showed that the group difference was significant for social but not for monetary rewards. Similar findings have been reported very recently, that is, Ait Oumeziane et al. (2019) examined the ERPs in both the MID and SID tasks, but only found a unique association between depression score and the P3 elicited by social reward (see also Jin, Sabharwal, Infantolino, Jarcho, & Nelson, 2019). One possible explanation is that when socially relevant and irrelevant information are represented in the same context, people (as social animals) tend to pay more attention on social information (Frazier et al., 2017; Mesoudi, Whiten, & Dunbar, 2006). However, this cognitive bias to social rewards might be suppressed among depressive individuals due to their pessimistic expectation (see above). Consequently, the difference between participants with depressive symptoms and normal controls became stronger in the social reward compared to the monetary reward condition. Further research is awaited to clarify whether there are domain-specific effects of depression in reward consumption.

Investigating the relationship between depression and reward-related aberrations with neuroscience methods has been fruitful (e.g. Bracht, Linden, & Keedwell, 2015; Heller *et al.* 2009; Naranjo, Tremblay, & Busto, 2001) and provides important messages for depression prevention intervention (Burkhouse et al., 2018). Altered processing of reward in depression may result in severe health consequences such as suicidal attempts (Marchand et al., 2011). Previous studies on depression have yielded mixed findings regarding neurobiologically and functionally distinct stages of reward processing that unfold in time dimension. Specifically, some studies suggest that depression is selectively associated with altered reward anticipation (McFarland & Klein, 2009; Sherdell, Waugh, & Gotlib, 2012; Ubl et al., 2015), but others have observed problems in both the anticipation and consumption stages among depressed individuals (Carl et al., 2016; Forbes et al., 2009; Smoski et al., 2009; Watson & Naragon-Gainey, 2010). By distinguishing between social and nonsocial rewards, we discovered both common and dissociable abnormalities of reward processing associated with depressive symptoms. Seeing that reward dysfunction is present in multiple psychiatric disorders, improving the knowledge about precise dysfunction in depression is critical to diagnostic and therapeutic efforts (Admon & Pizzagalli, 2015). That is to say, recognizing depressed individuals' diverse responses to different kinds of rewards in different temporal stages could be indicative of efficient treatment targets to reduce the onset or recurrence of depression (Silk et al., 2012; Stoy et al., 2012). Indeed, a meta-analysis on depressive symptoms has confirmed that selective prevention programs targeted at specific risk are more effective than universal programs (Horowitz & Garber, 2006).

Finally, some limitations of this study and potential future directions should be noted. First, this study did not include diagnosed patients. There has been some evidence supporting the continuity of depression in clinical and nonclinical populations (Flett, Vredenburg, & Krames, 1997); cognitive and behavioral correlates of depression differ quantitatively but may not qualitatively along a continuum of mild, moderate, and severe depression (Bradley, Mogg, & Millar, 1996; Wierzbicki & Rexford, 1989). Still, follow-up studies are needed to verify the robustness of our findings in clinical samples. Second, due to resource limitation, we did not set up a third group with a medium level of depression. Therefore, it is undetermined whether the relationship between depression and monetary/social reward processing is non-linear (see also Gu, Ao, Mo, & Zhang, 2020). Third, the MID and SID feedback stimuli (pictures of money *v.* persons) were different in their level of visual complexity, which might be a potential confounding factor. However, recent studies have indicated that the ERPs from the MID and SID tasks are morphologically similar (Ait Oumeziane et al., 2017). Also, it is important that we observed dissociated effects of depression in the anticipation stage (using the same stimuli 'I' and 'II' as MID and SID cues), which were not contaminated by visual complexity. Moreover, since the ERP technique is unable to capture non-phase-locked neural activity (Kalcher & Pfurtscheller, 1995; Pfurtscheller & Lopes da Silva, 1999), researchers could examine the effect of depression on non-phase-locked EEG synchronization and desynchronization associated with monetary and social reward processing (e.g. Gotlib, 1998; Thibodeau, Jorgensen, & Kim, 2006). Last but not least, our sample consisted of young people, which may limit the generalizability of the current findings. While it is still debated, recent studies suggest that anhedonia is more likely to be a state-like symptom in depression (e.g. Thomsen, Whybrow, & Kringelbach, 2015). Therefore, in our opinion, longitudinal investigations would be helpful to determine whether the reward processing patterns revealed in this study change over time (e.g. Burani et al., 2019). Seeing that social reward plays a more important role in adolescence than in adulthood, it would also be interesting to re-examine our ERP findings in older participants (see also Ethridge et al., 2017; Kujawa, Kessel, Carroll, Arfer, & Klein, 2017).

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Author contributions.
DZ conceived the experiment. JS, RB, FZ and YZ performed the experiment and collected data. DZ analyzed the data. DZ and RG wrote the manuscript. CF revised the manuscript.

Conflict of interest. The authors have declared that there is no conflict of interest in relation to the subject of this study.

Ethical standards. All procedures performed in this study were in accordance with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Data and code availability. The data and code of this study would be available upon request and with approvals of College of Psychology and Sociology, Shenzhen University. More information on making this request can be obtained from the first author, Prof. Dandan Zhang (zhangdd05@gmail.com).

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