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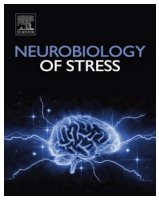

Acute stress selectively blunts reward anticipation but not consumption: An ERP study

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ABSTRACT

Stress-induced dysfunction of reward processing is documented to be a critical factor associated with mental illness. Although many studies have attempted to clarify the relationship between stress and reward, few studies have investigated the effect of acute stress on the temporal dynamics of reward processing. The present study applied event-related potentials (ERP) to examine how acute stress differently influences reward anticipation and consumption. In this study, seventy-eight undergraduates completed a two-door reward task following a Trier Social Stress Task (TSST) or a placebo task. The TSST group showed higher cortisol levels, perceived stress, anxiety, and negative affect than the control group. For the control group, a higher magnitude of reward elicited a reduced cue-N2 but increased stimulus-preceding negativity (SPN), suggesting that controls were sensitive to reward magnitude. In contrast, these effects were absent in the stress group, suggesting that acute stress reduces sensitivity to reward magnitude during the anticipatory phase. However, the reward positivity (RewP) and P3 of both groups showed similar patterns, which suggests that acute stress has no impact on reward responsiveness during the consummatory phase. These findings suggest that acute stress selectively blunts sensitivity to reward magnitude during the anticipatory rather than the consummatory phase.

1. Introduction

Stress can be defined as a process that occurs when an individual is aroused and experiences anxiety in relation to an uncontrollable and unpredictable challenge (Fink, 2016; Humiston and Lansing, 2021). Stress responses include physiological, psychological, and behavioral processes that allow an individual to cope with stressors (Humiston and Lansing, 2021; Shields et al., 2016). Acute stress causes rapid activation of the sympathetic adrenomedullary (SAM) system and relatively slow activation of the hypothalamic-pituitary-adrenocortical (HPA) axis, leading to increased heart rate (HR) and levels of circulating glucocorticoids (Allen et al., 2014; Murison, 2016; Russell and Lightman, 2019). Acute stress also induces altered alertness, sensory processing, memory encoding, and emotional responses, which recruit large-scale areas of the brain, including the default and salience networks (Hermans et al., 2011; Murison, 2016; van Oort et al., 2017).

Previous studies have contributed to clarifying how stress increases the risk of major depressive disorder (McEwen and Akil, 2020; Tsigos et al., 2020), and approximately 80% of people experiencing depression in community samples encountered major life stressors (Monroe et al., 2009). Although extensive research has linked depression to stress-induced impairment of cognitive control (Clark and Beck, 2010; Kaser et al., 2017; Rutherford et al., 2023), recent studies have attempted to highlight both cognitive control and reward system dysfunctions in depression (Grahek et al., 2019; Kaser et al., 2017). Anhedonia, typically defined as loss of pleasure, is one of the most promising endophenotypes of mental disorders and of depression in particular. It has been characterized as a dysfunction in reward processing (Lambert et al., 2018; Rizvi et al., 2016; Treadway and Zald, 2013). More specifically, Pizzagalli (2014) proposed a synthesis and integrated model and interpreted anhedonia as a pathological condition derived from dysfunctional interactions between stress and the reward system.

An influential model of the function of the reward system is the incentive salience theory. According to this theory, reward processing can be divided into an anticipatory phase before reward stimuli and a consummatory phase following reward feedback onset; both phases have independent functions and mechanisms (Berridge and Robinson, 2003; Berridge et al., 2009; Kringelbach and Berridge, 2009). Specifically, reward anticipation mainly recruits the mesolimbic dopaminergic system and causes a phasic release of dopamine, while reward

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consumption mainly recruits the nucleus accumbens and increases endogenous opioid secretion (Berridge and Kringelbach, 2015; Kringelbach and Berridge, 2009). An increasing number of studies have attempted to understand the psychopathological process of anhedonia in depression from the perspective of the temporal dynamics of reward processing (Kieslich et al., 2021; Rizvi et al., 2016; Romer Thomsen, Whybrow and Kringelbach, 2015). While some evidence suggests impaired reward consumption but relatively robust reward anticipation in anhedonia within depression (Foti et al., 2018; Webb et al., 2017; Whitton et al., 2016), further efforts are needed to clarify the relationship between anhedonia and the temporal dynamics of reward processing.

To investigate these dynamics of reward processing, event-related potentials (ERPs) at a high temporal resolution has been widely applied. Previous studies have usually measured the cue-elicited N2 and stimulus-preceding negativity (SPN) during the anticipatory phase of reward processing (Glazer et al., 2018). The cue-N2 is a negative component elicited by the reward-related cue onset over the frontal-central area, and it peaks at around 200 ms–350 ms (Folstein and Van Petten, 2008; N. Pornpattananangkul and Nusslock, 2015). Previous studies found that the cue-N2 was augmented following loss or even neutral cues compared with reward cues (Glazer et al., 2018; Novak and Foti, 2015; Potts, 2011). According to the template mismatch theory, individuals form a positive bias towards reward cues; therefore loss or neutral cues deviate from this preformed expectation and elicit a more negative N2 deflection (Folstein and Van Petten, 2008; Glazer et al., 2018; Lo, 2018; Potts, 2011). The SPN is a negative component over the frontal-temporal area that rises gradually and reaches a plateau at 200 ms prior to the onset of feedback stimulus (Brunia et al., 2011; Kotani et al., 2003; Ohgami et al., 2006). It originates within the anterior insula and reflects mesolimbic dopaminergic excitation (Foti and Hajcak, 2012). Previous research has shown that the SPN is enhanced prior to an upcoming reward feedback compared with neutral or loss feedback (Brunia et al., 2011; Mei et al., 2018), as well as anticipating high reward magnitude relative to low reward magnitude (Kotani et al., 2003; Yi et al., 2018), suggesting that the SPN reflects reward anticipation based on motivation salience (Brunia et al., 2011; Masaki et al., 2010; Ohgami et al., 2023).

For the consummatory phase of reward processing previous work has mainly implicated the Reward positivity (RewP) and P3 (Glazer et al., 2018). The RewP is a positive component that reaches a peak at about 200–300 ms over the frontal-central area and originates in the anterior cingulate cortex (ACC) and ventral striatum (Glazer et al., 2018; Proudfit, 2015). Previous studies have shown that RewP is enhanced following reward delivery rather than reward omission and loss (Proudfit, 2015; Threadgill and Gable, 2016), and it is also associated with a high reward magnitude compared with a low reward magnitude (Meadows et al., 2016; Paul et al., 2020; Sambrook and Goslin, 2015; Yaple et al., 2018), which suggests that RewP mirrors reward responsiveness (Burani et al., 2021; Proudfit, 2015; Threadgill and Gable, 2016). The P3 is a positive-going component with centro-parietal distributions usually measured at approximately 300–500 ms following reward feedback onset (Glazer et al., 2018). Its amplitude reflects attention allocation based on motivational salience of a feedback stimulus (Polich, 2007; San Martín, 2012).

A growing number of recent studies have investigated how acute stress influences the temporal dynamics of reward processing; however, the results have been contradictory (Dutcher and Creswell, 2018; Pizzagalli, 2014; Xin, 2020). Animal models have revealed that stressed mice show blunted motivation to receive a reward (i.e., reward anticipation) (Bergamini et al., 2016; Hollon et al., 2015) or a reduction in sucrose preference (i.e., reward consumption) (Krishnan et al., 2007; Tye et al., 2013), and acute stress not only inhibits dopamine neural firing in the reward system but also increases the dopamine level (Bouarab et al., 2019; Holly and Miczek, 2016). As in humans, the cortisol receptors are widely distributed in the reward system, including in the ventral tegmental area (VTA), nucleus accumbens (NAcc), and prefrontal cortex (Lopez and Flagel, 2021). During the reward anticipatory phase, individuals show reduced motivation to obtain rewards under a stress condition compared with those under a non-stress condition (Vriens, 2021). However, functional magnetic resonance imaging (fMRI) evidence has demonstrated that acute stress can activate reward-related areas (Choi et al., 2013; Dagher et al., 2009; Gaillard et al., 2019; Gorka et al., 2018; Kruse et al., 2018; Kumar et al., 2014) or suppress these areas (Gaillard et al., 2020; Ossewaarde et al., 2011). In addition, some fMRI studies have found that acute stress blunts activation of the reward system during the reward consummatory phase (Born et al., 2010; Kumar et al., 2014; Oei, Both, van Heemst and van der Grond, 2014) while other studies have found that these areas exhibit enhancement under stress (Gaillard et al., 2019; Porcelli et al., 2012). Furthermore, two ERP studies examined how acute stress modulates reward consumption, as indexed by the RewP, and the results showed that acute stress either blunts reward positivity (Burani et al., 2021) or does not affect it (Ethridge et al., 2020).

These contradictory results need to be further examined. One critical aspect is that most studies have only examined stress effects during either the anticipatory or consummatory phase, and the acute stressors and reward tasks have varied. Moreover, Porcelli et al. (2012) have found that acute stress decreased dorsal striatum and orbitofrontal cortex sensitivity to the magnitude of monetary outcome. The effects of acute stress on the dynamics of reward processing may stem from its impact on sensitivity to reward magnitude. Therefore, in this study, we aimed to use ERP technology to investigate the effects of acute stress on the temporal dynamics of reward processing under different reward magnitudes. Participants were instructed to complete a two-door reward task with EEG signal recording following a Trier Social Stress Test (TSST) or a placebo task. The TSST is a standard acute stress induction protocol and has been proven to be an ecologically valid stressor producing a consistent hypothalamic-pituitary-adrenal (HPA) axis response in humans (Allen et al., 2014; Kirschbaum et al., 1993; Narvaez Linares, Charron, Ouimet, Labelle and Plamondon, 2020). The two-door reward task is widely applied in reward processing research and is an effective tool for distinguishing the dynamics of reward processing (Proudfit, 2015).

As mentioned, the results of previous studies have been contradictory, and some have found that acute stress not only enhances but also blunts reward anticipation; however, Pizzagalli (2014) proposed that anhedonia in depression might arise from the detrimental effects of stressors on mesocorticolimbic DA pathways. Therefore, we speculated that acute stress could reduce reward anticipation and even decrease the discrepancy of the SPN between high and low reward conditions in stressed participants. Given the controversial results of acute stress effects on reward consumption (Burani et al., 2021; Porcelli et al., 2012), we did not construct an explicit hypothesis with respect to how acute stress influences reward processing during the reward consummatory phase.

2. Materials and methods

2.1. Participants

Of the 78 undergraduates from Shenzhen University who participated in this study, four were excluded owing to EEG recording errors. The remaining participants were randomized into a stress group (N = 40, 24 males and 16 females; age: $M = 19.95 \pm 1.36$ years) and a control group (N = 34, 19 males and 15 females; age: 19.65 ± 1.28 years), and there were no significant differences in age ($t(72) = 0.99$, $p = 0.326$, Cohen's $d = 0.23$) or gender ($\chi 2(1) = 0.13$, $p = 0.721$) between the groups. All participants had normal or corrected-to-normal vision, and were right-handed, reported no neurological and psychological conditions, current use of medication, drug abuse, and smoking. Female participants had regular menstrual cycles and were tested during their

follicular phase (4–12 days after the cessation of the last menstrual period) to exclude the impact of the follicular phase (Hamidovic et al., 2020; Maki et al., 2015). Each participant received compensation and provided informed consent based on the protocol approved by the Shenzhen University Institutional Review Board.

2.2. General procedure

As illustrated in Fig. 1, participants visited our laboratory during 13:00 to 18:00 for the circadian fluctuation of cortisol levels (Russell and Lightman, 2019). After arrival, they were instructed to complete pre-TSST tests (e.g., the Perceived Stress Scale (PSS), the Behavioral Activation System/Behavioral Inhibition System (BIS/BAS) scales, and the self-rating depression scale (SDS)) and then rest for 10-min while reading a travel guide (tourist brochure). Subsequently, the physiological (i.e., salivary cortisol) and psychological responses (i.e., the visual analog scale of stress (VAS-S), the negative affect scale of positive and negative affect schedule (PANAS-NA), and the state version of the State-Trait Anxiety Inventory (STAI-S)) of acute stress were collected (baseline, 36 min before the TSST task). After EEG preparation, participants in the stress group were given a TSST task, and those in the control group were given a placebo-TSST. Thereafter, they were instructed to complete a two-door reward task to win monetary rewards while EEG signals were recorded (approximately 30 min). After the task and EEG, participants were seated quietly to read the travel guide (tourist brochure) for 20 min to recover from their stressed state. Salivary cortisol samples and VAS-S were also collected immediately after the TSST task (S2, at +17 min), after every reward task block (S3, at +26 min S4, at +34 min S5, at +42 min S6, at +50 min), and every 10 min during the rest phase (S7, at +60 min and S8, at +70 min). The visual analog scale of control (VAS-C) was administered only at S2, and the STAI-S and PANAS-NA were administered at S2, S6, and S8.

2.3. Questionnaires

To control for the influence of chronic stress and depression on the stress responses between the stress and control groups, PSS and SDS were applied. The PSS is a 10-item self-reported questionnaire that has been proven to be a valid and reliable measure of chronic perceived stress (Cohen, 1988). Participants rated how frequently they have experienced each stress item over the previous month, ranging from 1 (never) to 5 (always). A higher score denotes that a higher stress level was perceived by the participant during the past month. The SDS is a self-reported questionnaire containing 20 items that measure depression levels (Zung, 1965). Participants assess the number of times they have experienced each statement on the questionnaire in the past week, ranging from 1 (never) to 4 (always). To enable comparison and analysis, the raw sum score is converted to a 100-point scale, which provides the self-rating depression index.

As previous studies have also suggested that reward sensitivity influences reward processing and stress responses (Bress and Hajcak, 2013; Daniel P. Moriarity et al., 2020; Harden et al., 2018), the Chinese version of the BIS/BAS scales (Carver and White, 1994; Li et al., 2008) were also applied in the current study. The 22-item questionnaire contains a behavioral inhibition system dimension (BIS) and three behavioral activation system dimensions representing reward responsiveness (BAS-RR), drive (BAS-D), and fun-seeking subscales (BAS-FS). Participants rate how strongly they agree with each item, from 1 (strongly agree) to 4 (strongly disagree). Lower BIS and BAS scores indicate a higher level of reward sensitivity. Other measures were detailed in Supplementary Material.

2.4. Stress induction

Acute stress was induced by a revised TSST task (Buchanan et al., 2012), which could produce a robust effect on cortisol levels and has been approved for good validation. The TSST or placebo-TSST task consisted of a preparation period (5 min), a speech task (5 min), and an arithmetic task (5 min). In the stress group, participants were given 5 min to prepare a speech to defend themselves against a charge of shoplifting. They were allowed to make notes in the preparation room; however, they could not take the notes in the interview room. They then entered the interview room where they stood in front of a microphone and a three-member committee. All members of the committee were professionally trained; they wore white coats and maintained a neutral facial expression throughout the task. Participants were required to give a 5-min speech and complete a 5-min mental arithmetic task (performing serial subtraction of 13 from 1022 or 14 from 1023) while being videotaped. During the mental arithmetic task period, participants were asked to respond as quickly and accurately as possible and restart if they made a mistake.

The placebo-TSST was equally physically and mentally demanding as the stress task, but not stressful through omitting the social and self-relevant components (Buchanan et al., 2014; Chu et al., 2023; Gilbertson et al., 2019). In the placebo–TSST condition, participants were instructed to deliver a speech according to a neutral travel article in the preparation room without any committee. They first spent 5 min prepared for the speech, followed by delivering the prepared speech in front of a video camera for 5 min. Subsequently, they engaged in a 5-min session of performing simple mathematical calculations, with no specific performance requirement and evaluation.

2.5. Two-door reward task

Immediately after the TSST task, participants were given a two-door reward task adapted from Dunning and Hajcak (2007). During the task, every trial began with a 500-ms cue (square or circle, counterbalanced between subjects) that indicated the magnitude of the reward. Thereafter, subsequent to a fixation that appeared for 500 ms–700 ms, two doors were presented on the center of a computer screen. Participants

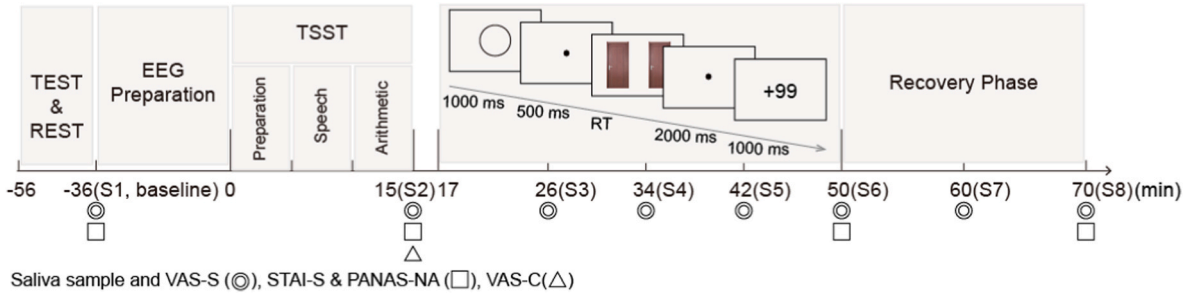

Fig. 1. Schematic representation of the experiment. Participants were instructed to complete a two-door reward task following a Trier Social Stress Test (TSST) or a placebo task. S1-8 = salivary cortisol samples 1–8, VAS-S = visual analog scale of perceived stress, STAI-S = the state version of the State-Trait Anxiety Inventory, PANAS-NA = negative affect subscale of Positive and Negative Affect Schedule, VAS-C = visual analog scale of control.

were required to choose a door that may hide a reward, and the doors then disappeared. Participants then had to wait 2000 ms for the appearance of reward feedback, and a reward feedback stimulus then appeared for 1000 ms to inform participants whether the reward delivered or not. Participants had the opportunity to win 9 points in the low reward condition (+9 or 0) or 99 points in the high reward condition (+99 or 0). However, the outcome of each trial was predetermined, and there was only a 50% probability of gaining a reward in both conditions. The task was divided into four blocks, each containing 60 trials, and a brief break of approximately 2–3 min was given after every block. Before the formal experiment, each participant underwent a practice procedure that included eight trials to familiarize themselves with the task.

2.6. Saliva sampling and cortisol analysis

Saliva samples were collected using Salivettes (Sarstedt, Rommelsdorf, Germany) and stored in a medical refrigerator at −22 °C until analysis. All samples were dissolved and centrifuged at 3,000 rpm/min for 10 min. Cortisol measurements were conducted using an electrochemiluminescence immunoassay (Cobase 601, Roche Diagnostics, Numbrecht, Germany). The lower limit of detection of cortisol was 0.5 nmol/L. The intra-and inter- assay variations were below 10%. The intra-and inter- assay variations were below 10%.

2.7. EEG recording and preprocessing

Continuous electroencephalogram (EEG) signals were recorded through a set of Ag/AgCl electrodes according to the extended 64-channel 10–20 international system. All signals were referenced to the left mastoid. The horizontal electrooculogram was recorded by a pair of electrodes placed approximately 1 cm from the outer of bilateral canthi, eye blinks were monitored through a pair of electrodes placed under/below ocular of the left eye. Every electrode resistance was maintained below 5 kΩ throughout the experiment and signals were amplified in DC mode and low-pass filtered at 100 Hz using a Neuroscan Synamp2 Amplifier (Scan 4.5).

Offline preprocessing of the EEG signals was conducted using MATLAB 19.0 (MathWorks, Natick, Ma, USA) and EEGLAB 2022.0 (Delorme and Makeig, 2004). The EEG data were re-referenced to the average value of bilateral mastoids. For the SPN, EEG data were filtered with a band pass of 0.01–30 Hz (roll-off 6 dB/octave) and segmented into −2500 ms–200 ms with a baseline from −1900 to −1700 ms in relation to the feedback onset (Masaki et al., 2010). For cue-N2, RewP, and P3, EEG data were filtered with a band pass of 0.1 Hz–30 Hz (roll-off 6 dB/octave) and segmented into −200 ms–1000 ms with a baseline correction from −200 to 0 ms before the cue or feedback onset. Thereafter, artifacts were deleted manually, and blinks were detected using an independent component analysis (ICA) algorithm. For the cue-N2 and SPN, epochs exceeding ±100 μV were excluded. For the RewP, a procedure was employed to exclude extra epochs if an epoch had a voltage difference exceeding 50 μV between two neighbor sample points, a voltage difference exceeding 200 μV, or a maximum voltage difference smaller than 0.5 μV within 100-ms intervals. Finally, cleaned data were averaged across conditions per participant.

To conduct the ERP analysis, the time window of each component was selected based on previous research, visual inspections of the grand average waveform, and topographical maps across conditions. Based on previous studies and topographical maps, the mean amplitudes of cue-N2 between 300 and 350 ms in response to the cue at the central-frontal area (Fz, FCz) were selected (Folstein and Van Petten, 2008), and the mean SPN amplitude between −200 and 0 ms at the fronto-temporal area prior to feedback was obtained (F5/F6 and FC5/FC6) (Holtgraves and Kraus, 2018; León-Cabrera et al., 2017). Subsequently, the following were obtained: the mean amplitude of RewP between 200 and 300 ms in relation to feedback stimulus onset over the central-frontal area (Fz, FCz) (Aziz et al., 2020; Wang et al., 2020) and the mean amplitude of P3 between 300 and 450 ms over the parietal area (CPz, Pz) (Hopstaken, van der Linden, Bakker, Kompier and Leung, 2016; Polich, 2007).

2.8. Data analysis

Statistical analyses of behavioural and cortisol data were conducted in SPSS (IBM SPSS Statistics for Windows, Version 22.0; IBM Corp., Armonk, NY, USA). Physiological (cortisol) and psychological (VAS-S, PANAS-NA, and STAI-S) responses indicated effects of stress induction. For the cortisol and VAS-S data, group (stress, control) × time (baseline, S2, S3, S4, S5, S6, S7, and S8) repeated measure analysis of variance (ANOVA) was conducted, and for the PANAS-NA and STAI-S data, group (stress, control) × time (baseline, S2, S6, and S8) ANOVAs were conducted.

To verify the effectiveness of reward manipulation and clarify the impacts of acute stress on reward processing, we analyzed behavioral data, subjective ratings, and ERP components. A group (stress, control) × reward magnitude (low reward, high reward) ANOVA was conducted on the response time (RT). For the subjective ratings of the reward task (i.e., interest, control, and regularity), group (stress, control) × reward magnitude (low reward, high reward) ANOVAs were conducted. To control for influences of stress and depression, t-tests were conducted between the stress and control groups for the VAS-C, PSS. During the reward anticipatory phase, we conducted a group (stress, control) × magnitude (low reward, high reward) × site (Fz, FCz) ANOVA for the cue-N2 and a group (stress, control) × magnitude (low reward, high reward) × site (F5/F6 and FC5/FC6) × hemisphere (left, right) ANOVA for the SPN to test acute stress enhancement effects on reward anticipation. During the reward consummatory phase, group (stress, control) × magnitude (low reward, high reward) × valance (gain, non-gain) × site ANOVAs were conducted on the RewP (FCz, Cz) and P3 (CPz, Pz). Greenhouse–Geisser epsilon correction and Bonferroni correction were employed when factors had more than two levels (Jennings and Wood, 1976).

3. Results

3.1. Descriptive, behavioral, and rating data

Chronic stress levels and depression did not differ between participants in the control and stress groups, as evidenced by repeated measure ANOVAs conducted on PSS ($t(72) = 1.79$, $p = 0.078$, Cohen'$d = 2.52$) and SDS scores ($t(72) = 0.50$, $p = 0.620$, Cohen'$d = 0.71$), respectively. Similarly, there were no significant main effects of group with respect to dimensions of the BAS/BIS, including the BIS ($t(72) = 0.20$, $p = 0.846$, Cohen'$d = 0.05$), BAS-R ($t(72) = 0.83$, $p = 0.408$, Cohen'$d = 0.20$), BAS-D ($t(72) = 0.45$, $p = 0.656$, Cohen'$d = 0.15$), and BAS-F ($t(72) = 1.12$, $p = 0.265$, Cohen'$d = 0.26$).

When choosing between two doors in the two-door reward task, there was no significant difference in the RT between the stressed participants and non-stressed controls (592 ms *vs.* 712 ms), $F(1, 72) = 3.12$, $p = 0.082$, $\eta_p^2 = 0.42$. In addition, the main effect of the reward magnitude did not reach significance, $F(1, 72) = 0.27$, $p = 0.605$, $\eta_p^2 = 0.04$, and participants responded as fast in the high reward condition ($M = 658$ ms, $SE = 40$) as in the low reward condition ($M = 646$ ms, $SE = 31$). Furthermore, the interaction between the group and the reward magnitude on reaction times was not significant, $F(1, 72) = 2.81$, $p = 0.098$, $\eta_p^2 = 0.04$. Rating data results were presented in supplementary material.

3.2. Physiological response to stress

As shown in Fig. 2, the physical and psychological variations indicated that stress was successfully induced by the TSST administration. For cortisol data, a significant interaction was obtained between time

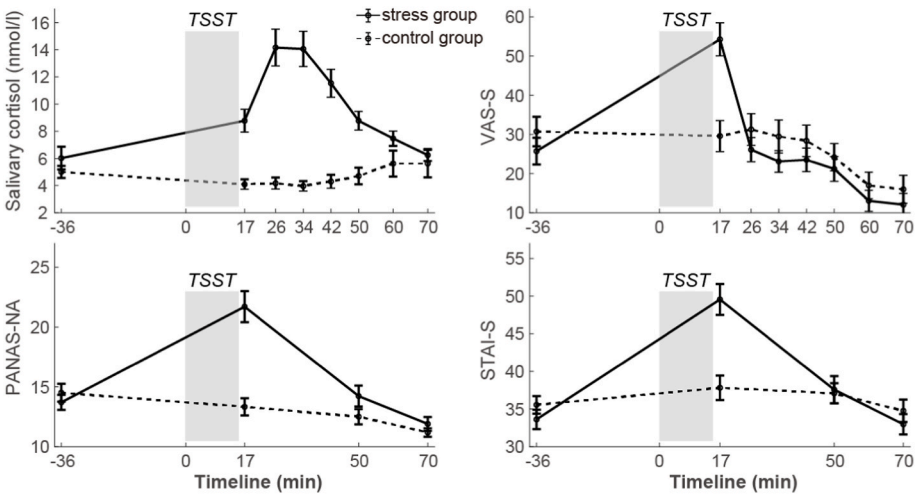

Fig. 2. Temporal fluctuations in salivary cortisol levels, VAS-S, PANAS-NA, and STAI-S. Error bars denote standard errors of means.

and group, F (7, 504) = 22.84, $p < 0.001$, $\eta_p^2 = 0.24$. Post hoc comparisons revealed that the cortisol concentrations of the stress and control groups were comparable at baseline (6.03 *vs.* 5.00, $p = 0.309$). Cortisol levels were significantly increased in the stress group relative to those in the control group post TSST task (S2, 8.78 *vs.* 4.09, $p < 0.001$) and at S3 to S6 (S3: 14.16 *vs.* 4.17, S4: 14.06 *vs.* 4.17, S5: 11.53 *vs.* 4.30, and S6: 8.77 *vs.* 4.70, *ps* < 0.001); however, this increase disappeared during the rest phase (S7: 7.47 *vs.* 5.62, and S8: 6.25 *vs.* 5.62, *ps* > 0.05). Compared to the baseline, cortisol levels in the stress group were enhanced post TSST task (S2, $p = 0.031$); they reached a peak at S2 and S3 (*ps* < 0.001) (approximately 26–34 min after TSST onset) and then gradually reduced until there was no significant difference after S6 ($p = 0.053$).

For the VAS-S scores, there was a significant interaction between the group and time, F (7, 504) = 13.57, $p < 0.001$, $\eta_p^2 = 0.16$. Participants in the stress group reported higher subjective stress levels immediately after the TSST than those in the control group (55.05 *vs.* 29.59, $p <$ 0.001) but comparable subjective stress levels at other time points, including baseline (*ps* > 0.1). Similarly, there were significant group × time interactions both on the PANAS-NA, F (3, 216) = 21.49, $p < 0.001$, $\eta_p^2 = 0.23$, and the STAI-S scale, F (3, 216) = 17.48, $p < 0.001$, $\eta_p^2 = 0.20$. Post hoc analysis revealed that stressed participants experienced an increased negative affect from the TSST compared with the controls (21.73 *vs.* 13.35, $p < 0.001$), and their anxiety levels were higher (49.55 *vs.* 37.82); however, the negative affect and anxiety were comparable at other time points (*ps* > 0.1).

3.3. Electrophysiological data

3.3.1. The anticipatory phase: cue-N2 and SPN

Fig. 3 shows that the Cue-N2 is a negative-going component elicited by the cue stimulus onset, and it reached its peak at around 300–350 ms during the anticipatory phase. As revealed by a repeated measure ANOVA, there was a significant main effect of the reward magnitude, F (1, 72) = 4.56, $p = 0.036$, $\eta_p^2 = 0.06$, and the amplitude of the N2 was more negative in association with a low reward than with a high reward (−0.84 μV *vs.* −0.26 μV). However, the main effect of group did not reach a significant level, F(1, 72) = 2.21, $p = 0.141$, $\eta_p^2 = 0.03$. In addition, the interaction between group and reward magnitude reached a significant level, F(1,72) = 4.37, $p = 0.040$, $\eta_p^2 = 0.06$. The post hoc analysis revealed that the amplitude of N2 was increased in the low reward condition ($M = -0.60$ μV, $SE = 0.55$) compared with that in the high reward condition ($M = 0.57$ μV, $SE = 0.61$) in the control group (p = 0.005); however, the enhancement is not observable in the stress group (−1.10 μV *vs.* −0.57 μV, $p = 0.973$).

The SPN waveform rose gradually and approached a plateau at approximately 200 ms prior to reward delivery, as depicted in Fig. 4. The group × reward magnitude ANOVA showed a significant main effect of the reward magnitude, F(1, 72) = 5.77, $p = 0.019$, $\eta_p^2 = 0.07$, but not the group, F(1, 72) = 0.02, $p = 0.880$, $\eta_p^2 < 0.01$. The SPN was enhanced in the high reward condition ($M = -2.69$ μV, $SE = 0.43$) compared with that in the low reward condition ($M = -1.93$ μV, $SE = 0.39$). Furthermore, the interaction between the group and reward magnitude was significant, F(1, 72) = 4.38, $p = 0.039$, $\eta_p^2 = 0.06$. Post hoc comparisons revealed that the SPN was enhanced in the high reward condition (M = −3.08 μV, $SE = 0.64$) compared with that in the low reward condition ($M = -1.65$ μV, $SE = 0.57$) only in the control group ($p = 0.004$), but not in the stress group (p = 0.824). Moreover, there was a significant interaction between reward magnitude and hemisphere, F(1, 72) = 6.38, $p = 0.014$, $\eta_p^2 = 0.08$. Post hoc comparisons indicated that high reward ($M = -3.01$ μV, $SE = 0.45$) elicited a higher SPN amplitude relative to low reward ($M = -1.86$ μV, $SE = 0.45$) over the right hemisphere (p = 0.002) but not the left hemisphere ($p = 0.294$).

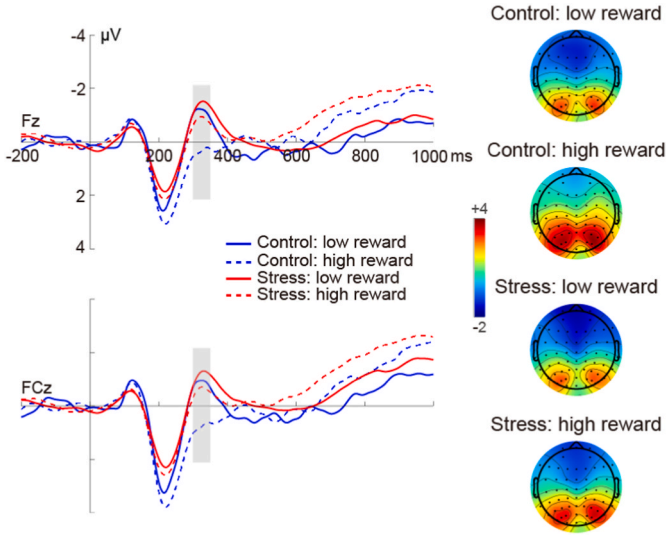

Fig. 3. Grand average amplitude and scalp distributions of N2. Gray-shaded rectangles represent the time window (300 ms–350 ms) in which the mean amplitude of N2 was scored.

3.3.2. The consummatory phase: RewP and P3

As depicted in Fig. 5 and S1, the RewP was peaked at around 200–300 ms, and the P3 was peaked at around 300–500 ms following

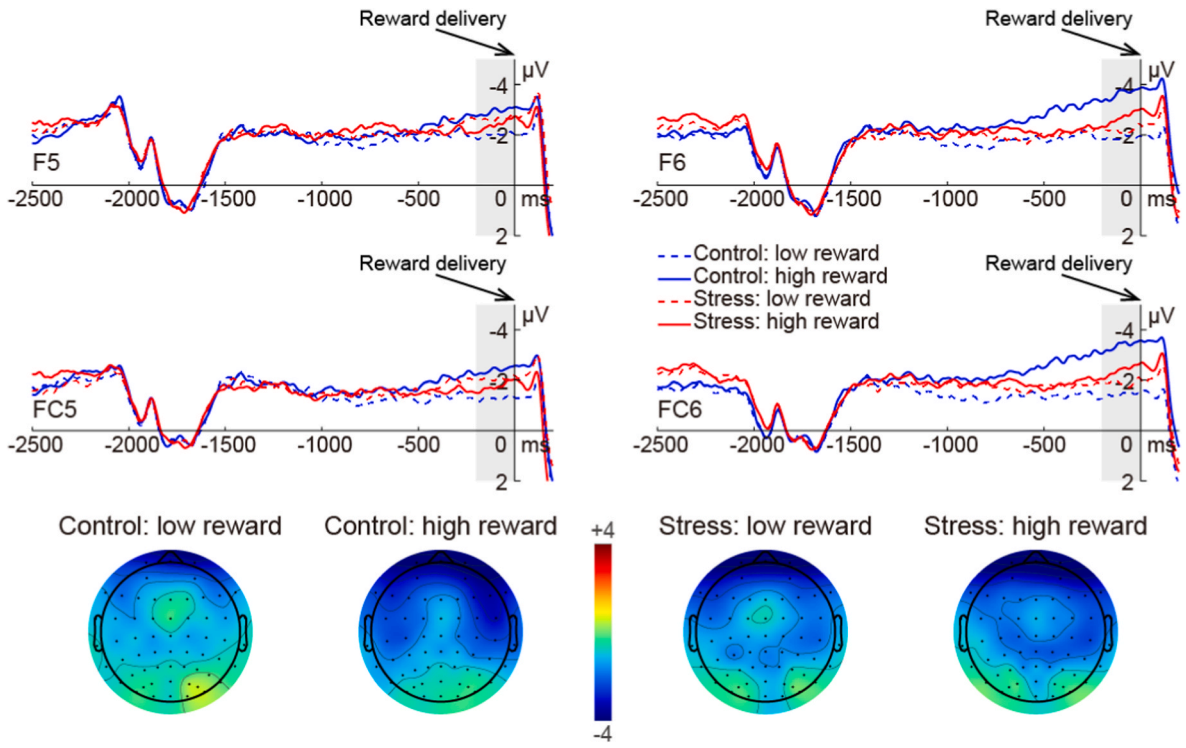

Fig. 4. Grand average amplitude and scalp distributions of SPN. Gray-shaded areas indicate the 200-ms time window in which the mean amplitude of SPN was scored.

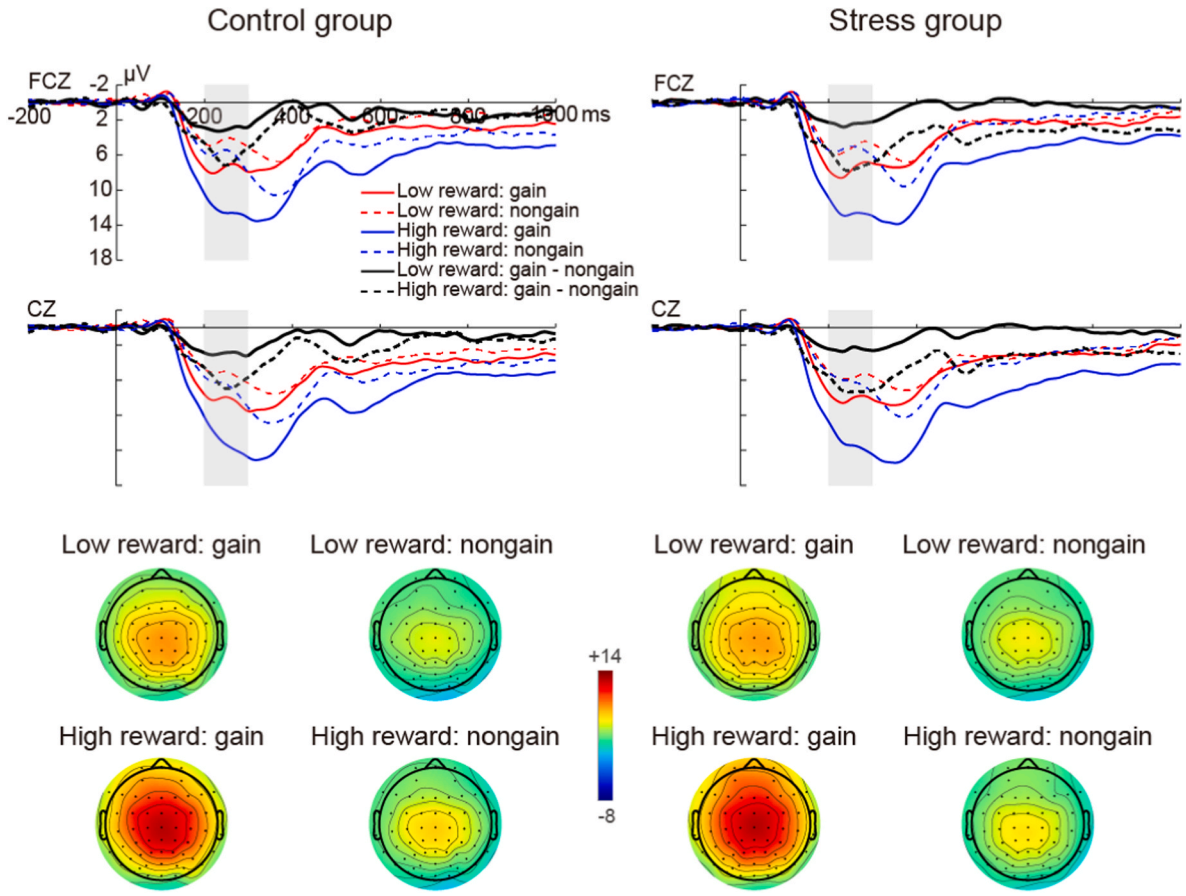

Fig. 5. Grand average amplitude and scalp distributions of RewP. Shaded areas represent the time window (200–300 ms) in which the mean RewP amplitude was scored.

the onset of the reward feedback stimulus. The group × magnitude × valance × site ANOVA on the RewP revealed the main effects of the valance, $F(1, 72) = 138.47$, $p < 0.001$, $\eta p^2 = 0.66$, and reward magnitude, $F(1, 72) = 85.62$, $p < 0.001$, $\eta p^2 = 0.54$. A higher amplitude of RewP was elicited in the gain condition ($M = 10.38$ μV, $SE = 0.64$) than in the non-gain condition ($M = 5.69$ μV, $SE = 0.43$), as well as in the high reward condition ($M = 9.48$ μV, $SE = 0.60$) relative to the low reward condition ($M = 6.58$ μV, $SE = 0.46$). As expected, a significant interaction was observed between the reward valance and reward magnitude, $F(1, 72) = 50.61$, $p < 0.001$, $\eta p^2 = 0.41$. Post hoc comparisons revealed that the difference between reward delivery and reward omission was significantly higher in the high reward condition (12.80 μV *vs*. 6.17 μV, $p < 0.001$) than in the low reward condition (7.96 μV *vs*. 5.20 μV, $p < 0.001$).

For the P3, there were main effects of the valance, $F(1, 72) = 109.94$, $p < 0.001$, $\eta_p^2 = 0.60$, and reward magnitude, $F(1, 72) = 129.58$, $p < 0.001$, $\eta_p^2 = 0.64$. The P3 was significantly larger in the gain condition ($M = 11.66$ μV, $SE = 0.64$) compared with the non-gain condition ($M = 8.16$ μV, $SE = 0.56$), as well as in the high reward condition ($M = 11.68$ μV, $SE = 0.67$) relative to the low reward condition ($M = 8.14$ μV, $SE = 0.53$). The interaction between reward valance and reward magnitude was significant, $F(1, 72) = 15.47$, $p < 0.001$, $\eta_p^2 = 0.18$. A post hoc analysis indicated that the difference between reward delivery and reward omission was significantly higher in the high reward condition (13.95 μV *vs*. 9.40 μV, $p < 0.001$) than in the low reward condition (9.37 μV *vs*. 6.91 μV, $p < 0.001$).

No main or interaction effect with respect to the group was obtained ($ps > 0.05$) for the RewP and P3.

4. Discussion

In this study, we conducted pioneering research simultaneously investigating the effects of acute stress on both reward anticipation and consumption, utilizing a standard laboratory stressor and high temporal-resolution event-related potentials (ERPs). In accordance with previous studies (Denk et al., 2021; Narvaez Linares et al., 2020), the physiological (i.e., cortisol) and psychological responses (i.e., perceived stress, anxiety, and negative affect) to acute stress were enhanced immediately following the TSST administration and returned to baseline levels during the recovery phase, indicating that the TSST successfully induced an acute stress state and activated HPA axis responses. For the reward-related effects, a high reward magnitude elicited enhanced amplitudes of the cue-N2, SPN, and RewP, and reward delivery elicited high amplitudes of the RewP and P3 compared with reward omission; these results are consistent with those of previous studies (Glazer et al., 2018). More importantly, the cue-N2 was blunted, and the SPN was enhanced in the high reward condition compared with that in the low reward condition; however, this only occurred in the control group (not the stress group) during the reward anticipatory phase. During the reward consummatory phase, the RewP and P3 showed similar patterns in both groups. These results suggest that acute stress selectively undermines reward anticipation but not reward consumption.

During the anticipatory phase, N2 was enhanced in the low reward condition compared to the high reward condition, which is in line with enhanced N2 following loss or neutral cues compared to reward (Novak and Foti, 2015; N. Pornpattananangkul and Nusslock, 2015; Potts, 2011). The template mismatch theory has been used to interpret these results and proposes that the amplitude of N2 covaries with the deviation of real reward cues from pre-formed expectations (Folstein and Van Petten, 2008; Glazer et al., 2018). The results of the present study suggest that participants in the control group formed a positive bias towards upcoming reward cues, whereas low reward cues deviated from this preformed expectation and thus elicited a significantly higher N2 amplitude. However, acute stress might blunt reward sensitivity and dampen the positive bias toward reward cues, resulting in the disappearance of the reward magnitude effect during the anticipatory phase.

Furthermore, SPN was elevated during the reward anticipatory phase in the high reward condition compared with that in the low reward condition in the control group; however, this effect was not observed in stressed participants. Converging evidence has shown that SPN is enhanced when anticipating reward delivery compared with non-reward (Narun Pornpattananangkul, Nadig, Heidinger, Walden, & Nusslock, 2017) and loss (Angus et al., 2017; Brunia et al., 2011; Ohgami et al., 2006), which indicates that SPN reflects reward anticipation or motivation salience (Brunia et al., 2011; Ohgami et al., 2023). Therefore, our results suggest that the control participants were sensitive to reward magnitude and assigned more motivation salience to the higher magnitude of the upcoming reward. However, acute stress blunted sensitivity to the reward magnitude; therefore, the effects of reward magnitude disappeared during this anticipatory phase.

Pharmacological evidence assists in explaining our results of the cue-N2 and SPN. The VTA dopamine release plays a critical role in reward anticipation, especially in coding reward value (Chiew et al., 2016; Schultz, 2010; Tobler et al., 2005). Previous studies have suggested that acute stress exposure can suppress VTA dopamine activity toward subsequent stimulation (Douma and de Kloet, 2020; Holly and Miczek, 2016; Stanwood, 2019). In the current study, acute stress exposure may have blunted the activation of VTA dopamine in association with the reward value. Stressed participants, therefore, showed insensitivity towards the reward magnitude during the anticipatory phase.

During the reward consummatory phase, reward delivery elicited higher RewP and P3 amplitudes than reward omission, particularly under the high reward magnitude condition, regardless of stress. For the RewP, converging evidence demonstrates that it is enhanced in the reward condition with respect to a neutral or loss condition, which indicates that the amplitude of RewP amplitude mirrors reward responsiveness (Brown et al., 2022; Proudfit, 2015). Our results build on previous observations and show that acute stress does not influence reward responsiveness during the reward consummatory phase. For the P3, previous studies have suggested that it increases in amplitude as a function of attention based on motivation salience (Polich, 2007; San Martín, 2012). Our results suggested that participants assigned greater attention to high reward delivery for higher motivation salience, irrespective of where they were experiencing acute stress. Together, our results revealed that acute stress exerts no significant effect on reward processing during the consummatory phase.

Previous fMRI evidence has suggested that acute stress not only modulates reward anticipation but also influences reward consumption (Xin, 2020). As previously mentioned, few studies have examined the simultaneous effects of acute stress on reward anticipation and consumption, and these results could be confounded by the variations of stressors and reward tasks in different studies (e.g., Collins et al., 2017; Kumar et al., 2014; Porcelli et al., 2012). Furthermore, the effect of acute stress on reward consumption could be masked by that on reward anticipation owing to the relatively rapid reward processing but the low temporal resolution of the fMRI. Here, we applied a standard laboratory stressor and high temporal-resolution ERP technology to sophistically clarify how acute stress simultaneously modulates the dynamics of reward processing under different magnitudes of reward. Importantly, our results suggest that acute stress modulates reward anticipation and blunts sensitivity to the reward magnitude but not reward consumption.

A synthesis and integrated model proposed that stress induces anhedonia because it impairs the reward system, particularly reward anticipation (Pizzagalli, 2014). By simulating a stress situation with a standard laboratory stressor TSST, our results provide empirical evidence for this perspective and reveal that stress blunts sensitivity to the reward magnitude only during the anticipatory phase. Moreover, converging evidence reveals that stress often precedes the onset of depression and plays a crucial role in the development of depression (Beauchaine et al., 2019; Bylsma et al., 2008; Monroe et al., 2009). Previous studies have consistently linked depression to deficits of cognitive control and attempted to illustrate how stress impairs the

cognitive system (Clark and Beck, 2010; Kaser et al., 2017; Rutherford et al., 2023). However, Grahek et al. (2019) recently proposed a framework that incorporated cognitive control deficits and motivational impairments in depression, including reward processing. Considering the relationship between stress and depression, our results support this perspective and suggest that stress-induced desensitization of reward magnitude during the reward anticipatory phase is perhaps one of the key factors associated with the development of depression.

Our study has certain limitations. Firstly, all participants were undergraduates, and the sample is, therefore, not representative of the general population. Therefore, caution should be used in extrapolating our results (that show acute stress blunts reward anticipation but not consumption) to other populations. Secondly, individual differences might influence the effect of acute stress on the dynamics of reward processing. Previous studies have suggested that the effects of stress on reward processing could be buffered by certain personality traits, such as sensation seeking (McKay et al., 2018; Roth et al., 2019) and tolerance of uncertainty (Heereman and Walla, 2011; Iannello et al., 2017). Although we have controlled for individual differences in stress, depression, and reward sensitivity, future studies are required to illustrate the effect of individual differences on the relationship between stress and dynamics of reward processing. Thirdly, although the TSST has been proven to be an excellent stressor that causes robust HPA axis responses (Kirschbaum et al., 1993; Narvaez Linares et al., 2020), its effect is relatively mild compared with that of major life events. However, it is impossible to apply severe stressors to participants in laboratory situations owing to experiment ethics and mental health considerations. Future research is thus required to clarify the relationship between stress and reward in real-life situations. Furthermore, we did not include a loss/punishment condition in the current study. Previous studies suggested that inclusion of a loss/punishment condition could introduce break-even effects (i.e., prior losses make rewards especially appealing) (Huang and Chan, 2014; Suhonen and Saastamoinen, 2018; Suhonen et al., 2018; Thaler and Johnson, 1990), which could confound the effects of acute stress. However, the inclusion of a loss/punishment condition could provide a more realistic account of decision-making and enable to examine how acute stress affects loss/-punishment anticipation or consumption. Future research is necessary to investigate how acute stress affects the dynamics of reward processing under both reward and loss/punishment conditions, while carefully controlling for the potential confounds of break-even effects.

In conclusion, our study found that acute stress selectively dampens sensitivity to reward magnitude during the anticipatory phase but not the consummatory phase. Our findings provide insight into the potential mechanisms underlying stress-induced mental illness, and have implications for developing interventions aimed at mitigating the negative impact of stress on reward processing.

CRediT authorship contribution statement

Wei Yi: Writing – original draft, Writing – review & editing, Methodology, Formal analysis, Conceptualization. **Yantao Chen:** Investigation, Formal analysis. **Linlin Yan:** Formal analysis, Writing – review & editing. **Nils Kohn:** Conceptualization, Validation, Writing – review & editing. **Jianhui Wu:** Conceptualization, Validation, Writing – review & editing, Supervision.

Declaration of competing interest

The authors report no potential financial conflicts of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ynstr.2023.100583.

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