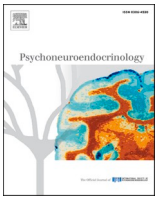
Psychoneuroendocrinology 133 (2021) 105388

Contents lists available at ScienceDirect

Psychoneuroendocrinology

journal homepage: www.elsevier.com/locate/psyneuen



Neural signature of affective but not cognitive self-regulation predicts cortisol response to psychosocial stress☆

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ARTICLE INFO

Keywords:
Cortisol response
HPA axis
Psychosocial stress
Cognitive control
Emotion regulation

ABSTRACT

Self-regulation is theoretically closely related to coping with stressful events, yet whether self-regulation capacities can predict individual stress responses is largely unknown. Cognitive control and emotion regulation are two major aspects involved in self-regulation, both of which are mechanisms to support goal-directed behaviors. Here, we aimed to elucidate whether the neural processes involved in emotion regulation and cognitive control could predict the cortisol response to stress. Therefore, we recorded first electroencephalography (EEG) during a cognitive conflict task (Simon task) and an emotion regulation task (cognitive reappraisal and expressive suppression) before healthy participants (n = 72) underwent a psychosocial stressor. Our results showed that late positive potentials (LPPs) during the emotion regulation task predicted both cortisol reactivity to and recovery from stress. Cognitive control and its neural underpinning, however, did not predict the individual stress response. These findings indicate that neural emotion regulation processes can predict HPA axis response to stress, and suggest a differential involvement of cognitive and affective components of self-regulation in the adaptation to stressful events.

1. Introduction

When threatened by physical and psychological stressors, organisms strive to reach a dynamic equilibrium that is known as homeostasis. In response to stress, the rapid activation of the sympathetic nervous system (SNS) leads to the release of noradrenaline and adrenaline, which lead, for instance, to increases in heart rate and blood pressure. In addition to SNS activity, the hypothalamus-pituitary-adrenocortical (HPA) axis is also activated is response to stress, resulting in the rise of glucocorticoids (mainly cortisol in humans) (de Kloet et al., 2005). The up-regulation of the stress hormone cortisol, however, comes at the cost of inhibiting other essential body mechanisms, such as the inflammatory/immune response (Tsigos and Chrousos, 2002). Thus, effective coping includes both a rapid activation of the stress response and an efficient termination afterwards (de Kloet et al., 2005).

HPA axis activity to stress is a crucial mechanism to adaptation: it up-regulates cortisol when needed and down-regulates it when the threat has been overcome (Erickson et al., 2003). It should be noted that the adaptive system HPA axis is featured with considerable individual variability (Gunnar and Quevedo, 2007; Kudielka and Wust, 2010). Accumulated evidence has shown that malfunction of HPA axis regulation is related to various mental and physical disorders, including depression, anxiety, and cardiovascular diseases (Foley and Kirschbaum, 2010; Lupien et al., 2009). Therefore, the ability to predict the stress response, and the responding of the HPA axis in particular, is important.

In the face of stressors, when the dynamic balance is broken, individuals need to cope with or adjust to challenges in the environment (Rothbaum et al., 1982). Such adjustments of may require the investment of self-regulation resources (Muraven and Baumeister, 2000). Self-regulation refers to the dynamic adjustment of an individual's internal state (emotion or cognition) or behavior to cope with a changing environment (Nigg, 2017). Previous studies suggested that poor self-regulation is associated with many stress-related mental disorders, such as depression, eating disorders, and generalized anxiety disorder

☆ We have no conflict of interest to disclose.

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https://doi.org/10.1016/j.psyneuen.2021.105388
Received 14 April 2021; Received in revised form 6 August 2021; Accepted 6 August 2021
Available online 9 August 2021

(Nigg, 2017; Wang et al., 2015). In addition, the dysregulation of stress hormone levels is related to difficulty in self-regulation (Blair et al., 2005). Cognitive control and emotion regulation are two classic components involved in self-regulation, both of which are underlying mechanisms that support goal-directed behaviors (Egner, 2017; Gross, 1998a). Interestingly, both cognitive control and emotion regulation have been reported to be related to the stress response. Regarding the relationship between cognitive control and the stress response, previous studies have mainly focused on error processing (Cavanagh and Allen, 2008; Compton et al., 2013; Tops et al., 2006; Wu et al., 2017). Most of those studies showed that the Error-related Negativity (ERN) component (Cavanagh and Allen, 2008; Compton et al., 2013; Tops et al., 2006) is associated with the cortisol response to acute stress, yet a recent study using psychosocial stress to induce cortisol did not detect the predictive effect of ERN, but reported that the error positivity (Pe) component (Wu et al., 2017) is predictive of a cortisol increase after stress manipulation. Whether ERN or Pe, they are both the neural activity reflected by error processing, and especially ERN is suggested to be closely related to anxiety (Moser et al., 2013; Saunders and Inzlicht, 2020). However, it remains unclear whether cognitive control in general can predict the stress response. The dynamic adjustment of cognitive control characterizes adaptive behavior: it matches the processing modes (the narrow or wide focus of attention) to changing environmental demands, and it monitors signals such as conflict (Braem et al., 2019). A widely used indicator to measure this adaptive control is the *conflict adaptation* effect (Egner, 2017). According to conflict monitoring theory, in the cognitive control tasks, the conflict caused by previous incongruent trials triggers the upregulation of attention towards the target and thus promotes more efficient attention selection in subsequent trials (Botvinick et al., 2004). Importantly, a previous study administrating a Simon task after stress/control manipulation found that the cognitive flexibility (indexed by sequential modulation of interference effects, also called conflict adaptation effects) was reduced in the stressful situation compared with the control group (Plessow et al., 2011). Based on the theoretical association between adaptive control and stress response, as well as the evidence from Plessow et al. (2011), we are particularly interested in whether the conflict adaptation measured under baseline status is able to predict stress response. Two event-related potentials (ERPs) are related to conflict adaptation: N2 and P3. N2 is a negative component elicited about 250–350 ms after the onset of stimuli that distributes in fronto-central areas (van Veen and Carter, 2006), and reflects conflict monitoring (Clayson and Larson, 2011a; Larson et al., 2014). P3 is a positive component observed on centro-parietal electrodes 300–600 after stimulus onset, and may be representative of response inhibition (Albert et al., 2013) and attention distribution (for review see Polich, 2007). Taking the advantage of ERP technique to dig into neural mechanisms, we aimed at employing conflict adaptation effects of N2 and P3 components as cognitive self-regulation predictors to the stress-induced cortisol response.

Another key mechanism of self-regulation is emotion regulation, which is defined as "how individuals influence which emotions they have, when they have them, and how they experience and express them" (Gross, 1998a). Two emotion regulation strategies have been investigated and compared in several previous studies. Accumulated evidence has shown that the cognitive reappraisal strategy is more emotionally adaptive than expressive suppression (Gross, 1998b). Emotion regulation is believed to be closely related to stress coping (Gross, 1999), and a previous study based on questionnaires found that the higher trait reappraisal was associated with greater cortisol recovery in healthy participants (Lewis et al., 2017), but their induced cortisol response was pretty weak. A more recent study also reported that habitual maladaptive emotion regulation strategies predicted blunted cortisol response to psychosocial stress (Krkovic et al., 2018). However, there is no neural evidence supporting the relationship between emotion regulation strategy and stress response. ERP research in the field of cognitive neuroscience found that emotional stimuli can induce a continuous late positive potential (LPP). The LPP appears approximately 300 ms after the onset of emotional stimuli and distributes over the centro-parietal electrodes (Hajcak, Macnamara, and Olvet, 2010). Compared to neutral stimuli, emotional stimuli elicited a larger LPP (Cuthbert et al., 2000; Schupp et al., 2004). More importantly, LPP is sensitive to the emotion regulation strategy and reflects the dynamic changes in the arousal level induced by emotional stimuli (Hajcak and Nieuwenhuis, 2006). Previous studies have shown that emotion regulation strategies (such as reappraisal, suppression, and distraction) reduced the LPP to negative stimuli (Moser et al., 2006, 2014; Thiruchselvam et al., 2011). Thus, we attempted to use this classic LPP component to measure the neural activity during two types of emotion regulation strategies (cognitive reappraisal and expressive suppression) and use this neural marker to predict the cortisol response to stress.

The present study aimed to investigate how cognitive control and emotion regulation predict stress response as indexed by HPA axis function. We measured the conflict adaptation with a classic cognitive control task (Simon task) by considering the indices in N2, and P3 components. We also extracted the LPP component from the same sample of participants who performed an emotion regulation task. Electrophysiological measures were used as predictors in the present study to investigate neural mechanisms and accumulate evidence on the association between baseline neural markers and stress response. Based on the findings of Krkovic et al. (2018), we hypothesized that the reduced LPP magnitude by emotion regulation predicts cortisol reactivity to and recovery from stress, and the effect of reappraisal and suppression strategies might predict in a different or even opposite direction. Meanwhile, according to the preliminary evidence of association between cognitive control and cortisol response to stress (e.g., Plessow et al., 2011), we assumed that a conflict adaptation effect on N2 and P3 might also be predictive of stress-induced cortisol change.

2. Material and methods

2.1. Participants

We recruited a total of 75 volunteers (38 males and 37 females; Mean age ± SD: 20.39 ± 1.26 years old; Range of age: 18–24 years old; Mean educated year: 14.43 ± 1.30) through an online advertisement. Based on an a-priori sample size calculation using the software G*power 3 (Faul et al., 2007), a sample of 56 participants is needed in a linear multiple regression with $\rho^2 = 0.3$ (Slattery et al., 2013), predictors = 3, power = 0.95, $\alpha = 0.05$. To obtain a sample size that was a multiple of 18 (the total number of combinations of picture sets assignment and conditions sequence in the Emotion regulation task), we aimed to achieve a sample of 72 valid datasets. The following exclusion criteria were checked during the recruitment procedure: (1) History of any mental or neurological disorder; (2) History of any endocrine disorders (such as Cushing syndrome); (3) History of other major chronic physiological diseases, such as diabetes, cardiovascular disease, meningitis, severe kidney disease, malignant tumors, etc.; (4) History of any neurological disorders (such as brain surgery, cerebral hemorrhage, severe head trauma, etc.); (5) Long-term use of antipsychotic drugs, neurological drugs or adrenal cortex hormone drugs; (6) Long-term use of any illicit drugs; (7) Major operations in the past six months; (8) Excessive alcohol consumption (more than two drinks a day) or excessive smoking (more than five cigarettes a day). Three participants were excluded from the analysis: one male due to being extremely sleepy while collecting EEG; one male due to the experimenter error while collecting EEG; one female due to current usage of oral contraceptive, thus resulting in a final sample of 72 participants (36 males and 36 females; Mean age ± SD: 20.40 ± 1.26 years old; Range of age: 18–24 years old; Mean educated year: 14.72 ± 1.31). All participants had normal or corrected to normal vision and were right-handed. They gave their informed consent to participate in the study and received monetary compensation. This study was approved by the Ethics Committee of Human Experimentation at the

Institute of Psychology, Chinese Academy of Sciences.

2.2. Procedure

The procedure is illustrated in Fig. 1. All participants came to the laboratory twice, within an interval of one week.

2.2.1. Day 1

Participants completed a Simon task (see Section 2.3.) and an Emotion regulation task (see Section 2.4.) while an electroencephalogram (EEG) was recorded.

2.2.2. Day 2 (within one week after Day1)

The second session took place in the afternoon between 1:30 and 4:00 pm to control for the cortisol circadian rhythm (Edwards et al., 2001). Within the two hours before the second session, participants were required to refrain from strenuous exercise, eating, or drinking anything except water. Upon arrival, participants were seated in a quiet room for rest. The participants completed a batch of questionnaires (the results of questionnaires will not be reported here since they were not the focus of the current study). After the rest phase, the first salivary sample was collected and heart rate (HR) was measured as baseline. Then the participants completed a stress manipulation task (see 2.6. Stress manipulation). The saliva samples were collected at 0 min, 5 min, 10 min, 15 min, 20 min, 30 min, 40 min, 50 min, and 60 min after the end of stress manipulation. The HR was recorded continuously during the experiment and was marked at each time point of saliva sample collection. Participants were seated in a quiet room during the recovery phase and were allowed to read scientific magazines.

2.3. Conflict task

In the numerical Simon task (see Fig. 1), eight numbers from 1 to 9 (except 5) were presented as target stimuli on the left or right side of the screen (Fischer et al., 2018). Participants sat in front of the screen with a viewing distance of 60 cm. The visual angle of the digit was $0.67° \times 0.95°$. The target digits were presented in white color on a black 17-inch screen, 10 cm randomly to the left or right side of fixation. Participants were instructed to decide whether the target number was smaller (press left key) or larger (press right key) than 5. The E-Prime 2.0 program was used for stimulus presentation and data collection (Schneider et al., 2002).

There were three blocks in this task. Participants were allowed to take a short break between blocks. Each block had a total of 65 trials (the first trial was not analyzed). In each trial, a fixation cross was presented first for 1000 ms, followed by the target stimulus for 250 ms. Then, the fixation cross remained until the subject responded (maximum:1600 ms). After response or timeout, a random blank screen interval between 200 and 800 ms was presented before the next trial started. Before the formal experiment, participants completed a practice block of 20 trials.

The sequence of the eight numerical stimuli was pseudo-random to ensure that identical stimuli did not appear on two consecutive trials to reduce the influence of low-level feature repetition and increase the degree of involvement in cognitive control (Egner, 2017). Except for the first trial of each block, all eight numbers in the remaining 64 trials were presented equally often on the left and right side (each number appeared four times on the left and four times on the right side). Thus, half of the trials were consistent and half were inconsistent to avoid the potential biased association between response and stimuli or position (Schmidt, 2013).

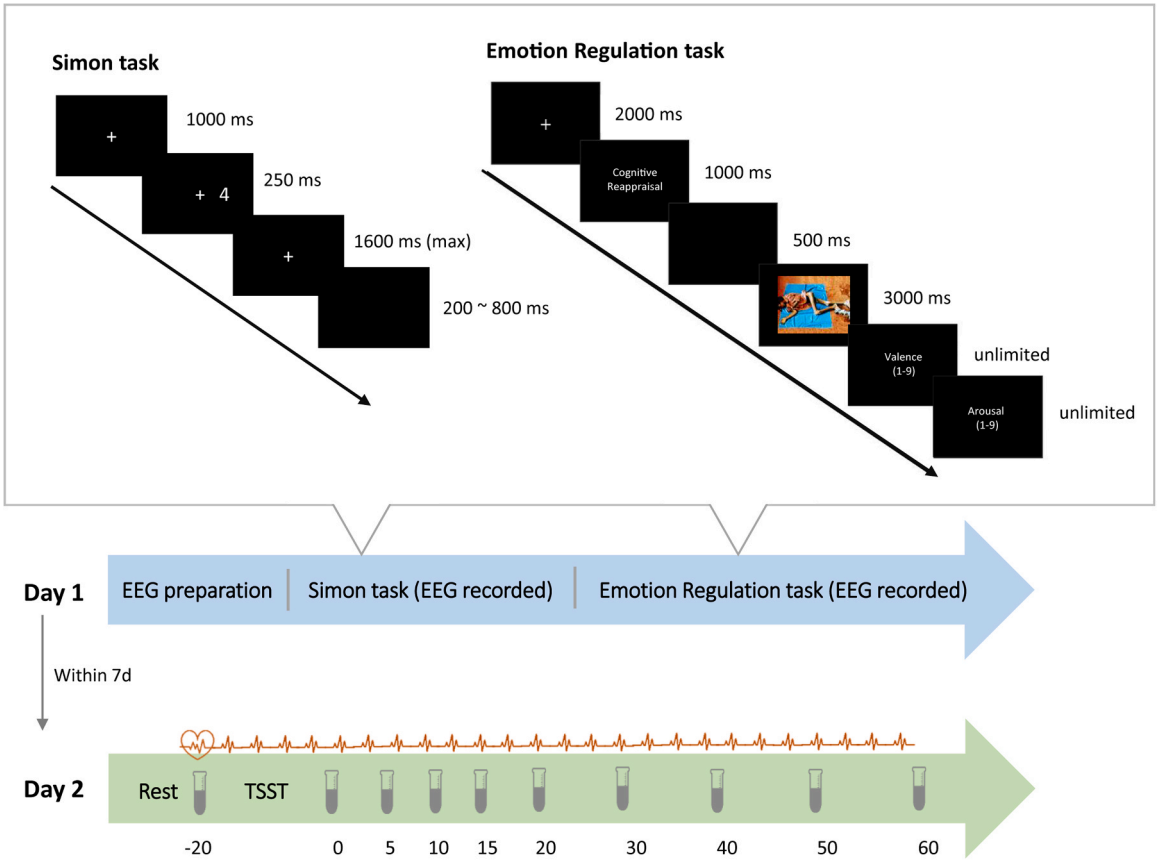

Fig. 1. General procedure. Day 1: we measured the neural activities of cognitive control and emotion regulation. Day 2 (within one week after Day 1): we measured the stress response by administrating the Trier Social Stress Test (TSST) protocol. 10 salivary samples in total were collected before and after the TSST to analyze cortisol response. Heart rate were measured continuously through the Day 2 session.

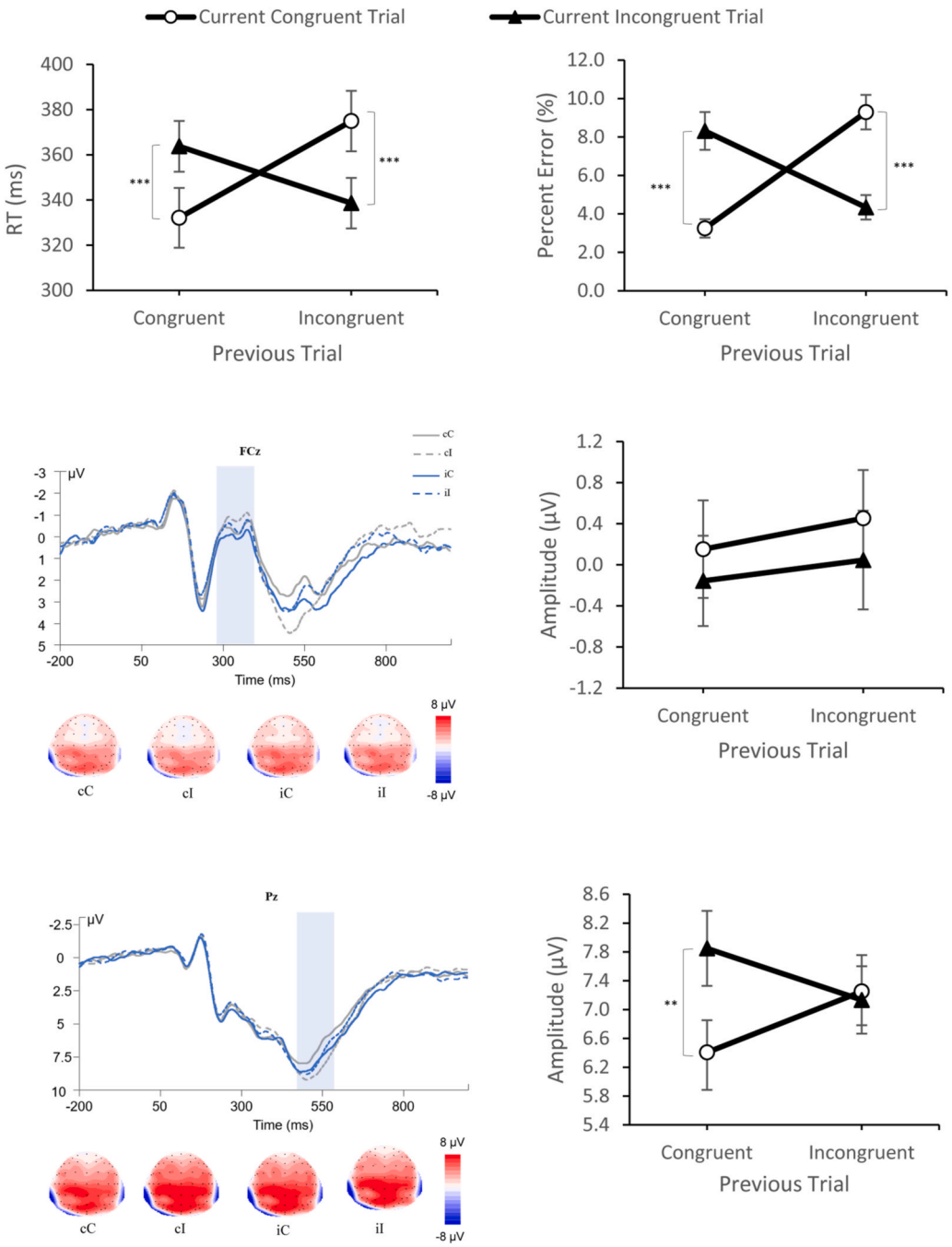

Fig. 2. (A) Response time (RT) and Percent Error (%) of Simon task; (B) N2 component on FCz at time window from 280 to 400 ms; (C) P3 component on Pz at time window from 480 to 600 ms. [cC: congruent precedes congruent; cI: congruent precedes incongruent, iC; incongruent precedes congruent; iI: incongruent precedes incongruent]. Error bars show standard error (same as below). **: $p < 0.01$, ***:$p < 0.001$.

Response conflict occurred when the side that required a response was incongruent with the position of the stimuli on the screen. Conflict adaptation was indicated by the reduction of the incongruent-congruent difference following incongruent trials compared to the incongruent-congruent difference following congruent trials (Clayson and Larson, 2011a; Fischer et al., 2018).

2.4. Emotion regulation task

The stimulus materials for the emotion regulation task included 120 pictures (90 negative, 30 neutral) selected from the International Affective Picture System (IAPS; Lang et al., 2008). Most of these selected pictures had been used in previous studies to investigate the neural mechanisms of emotion regulation (Thiruchselvam et al., 2011). According to the ratings provided for the IAPS (Lang et al., 2008), negative pictures and neutral pictures differed in valence (negative: M = 2.37, SD = 0.63; neutral: M = 5.13, SD = 0.51) and arousal (negative: M = 5.97, SD = 0.77; neutral: M = 3.23, SD = 0.69). These valence and arousal levels of these pictures were similar to previous LPP studies (Hajcak and Nieuwenhuis, 2006; Schupp et al., 2004; Thiruchselvam et al., 2011). The 90 negative pictures were divided into three picture sets A, B, and C, with 30 pictures each. The valence and arousal levels of these three picture sets were comparable (*ps* > 0.7).

Participants completed the emotion regulation task (Moser et al., 2014; Thiruchselvam et al., 2011) while EEG was recorded. This task included four conditions: view (neutral pictures), view (negative

pictures), cognitive reappraisal (negative pictures), and expressive suppression (negative pictures). In the viewing task, subjects were instructed to watch neutral and negative pictures, respectively, and allowed their natural emotional reactions. The instructions of cognitive reappraisal and expression suppression were adapted from Gross (1998b).

2.4.1. Cognitive reappraisal

While you are viewing the pictures, please use the cognitive reappraisal strategy to regulate your emotions so that you can be more emotionally neutral. Emotional regulation strategy reference: reinterpret the meaning of the picture, give it a more positive meaning (e.g., imagine that the picture depicts a scene that will evolve for the better over time), or see it from a detached perspective (e.g., imagine that the picture depicts a movie scene or a scene made by Photoshop, which is not real, and just appreciate the level of production of the picture).

2.4.2. Expression suppression

While you are viewing the pictures, please use the expression suppression strategy to regulate your emotions so that you can be more emotionally neutral.

Expression inhibition strategy reference: consciously control your emotional reaction to the picture without showing it, so that the person next to you cannot perceive your emotional experience.

All trials started with a white fixation in the center of the black screen for 2000 ms, followed by a 1000 ms cue (view, cognitive reappraisal, expressive suppression), a 500 ms blank screen, and then a picture for 3000 ms (see Fig. 1). After the picture disappeared, participants were required to rate the valence and arousal on a scale from 1 to 9, using the Self-Assessment Manikin (SAM) scale (Bradley and Lang, 1994; Lang, 1980). Participants' visual distance to a 17-inch computer screen was approximately 60 cm. E-Prime 2.0 was used for stimulus presentation and data collection (Schneider et al., 2002).

We used a block design with four blocks. Only one condition was in each block and 30 trials were in each condition. All participants completed the view (neutral) condition first to avoid the influence of emotional conditions on subsequent tasks (Gross, 2002; Moser et al., 2006). Participants rested for 2 min between blocks to return to a basic state (Cai et al., 2016). The three conditions of view (negative), cognitive reappraisal (negative), and expressive suppression (negative) were counterbalanced among the participants. The pictures in each block were presented in a random sequence. In addition, the assignment of the three negative picture sets A, B, C to the three negative conditions was counterbalanced across participants.

2.5. EEG recording

EEG signals were recorded from 64 scalp sites according to the international 10–20 system using Ag/AgCl electrodes (Neuroscan Inc., USA). The EEG recording was amplified with a 0.05–100 Hz band pass filter and continuously digitized at a sampling rate of 1000 Hz, with an online reference to the left mastoid. The vertical electrooculograms (VEOG) were recorded by electrodes placed above and below the left eye. The horizontal electrooculograms (HEOG) were recorded from electrodes placed 1 cm from the outer canthi of each eye. The impedance of all electrodes was maintained below 5 kΩ.

The EEG data were processed offline with Scan 4.3 (Neuroscan Inc., USA). The data were re-referenced to the average of the left and right mastoids. Eye blinks were identified and corrected using an Ocular Artifact Reduction (OAR) transform algorithm built in the Neuroscan software (Semlitsch et al., 1986). Data were digitally lowpass-filtered at 30 Hz (FIR filter, half-amplitude cut-off) and extracted into epochs of 1200 ms (baseline-corrected with the 200 ms window pre-stimulus) time-locked to the onset of the stimuli (numbers in the Simon task; pictures in the Emotion regulation task). Trials with artifacts exceeding ±100 μV were automatically detected and rejected. For the conflict task, we included only correct trials in the subsequent analyses to prevent ERPs from potential contamination of error-related negativity (Miller et al., 2012).

For the EEG data of the Simon task, after excluding the first trial in each block, error trials (6.32% in total), and trials with artifacts exceeding ±100 μV (7.12% in total), the number of average accepted trials for condition cC, iC, cI, and iI (see Section 2.8 for description of each condition) was 41.57, 41.57, 41.65, and 41.40 trials, respectively. Previous studies identified maximal amplitudes of N2 in the centro-frontal regions (Clayson and Larson, 2011a; Larson et al., 2014; van Veen and Carter, 2002, 2006) and distribution of P3 in the centro-parietal areas (Clayson and Larson, 2011a; Polich, 2007). According to the above evidence and visual inspection of our data, N2 was extracted in a time window of 280–400 ms over electrodes of F1, Fz, F2, FC1, FCz, FC2, and P3 was extracted in a 480–600 ms time window over sites of CP1, CPz, CP2, P1, Pz, P2. The average mean amplitudes across those respective electrodes were used for subsequent analysis regarding N2 and P3.

For the EEG data of the Emotion regulation task, after excluding trials with artifacts exceeding ± 100 μV (1.74% in total), the number of average accepted trials for condition View neutral, View negative, Cognitive reappraisal, and Expression suppression was 29.49, 29.54, 29.40, and 29.49 trials, respectively. LPP was extracted over centro-parietal brain areas (CP1, CPz, CP2, P1, Pz, P2, PO3, POz, PO4) in the time window of 300–1000 ms based on visual inspection and the previous studies (Hajcak et al., 2010; Hajcak and Nieuwenhuis, 2006; Moser et al., 2014).

2.6. Stress manipulation

Kirschbaum et al. (1993) developed an effective and widely used protocol to induce psychosocial stress in the laboratory, the Trier Social Stress Test (TSST). In the current study, we implemented a modified version of the TSST (Buchanan et al., 2012), which has been reported to result in a robust cortisol increase (Buchanan et al., 2012; Wu et al., 2017). The TSST had three periods: a 5-min preparation, a 5-min speech, and a 5-min mental arithmetic task. Participants were told to prepare a speech to defend themselves in a scenario in which they were accused of shoplifting, and that their performance would be recorded with a camera. After preparation, participants were taken to another room, where three experimenters, dressed in white lab coats, were seated who acted as two store managers and a police officer. Participants had to give a defending speech in front of those three experimenters who did not provide any reinforcing feedback. After the speech, the participants were then required to perform a mental arithmetic task in which they counted backwards from 1022 in steps of 13. Once participants made a mistake, they had to start from 1022 again. After TSST, participants were escorted back to the previous room for a rest.

2.7. Assessment of stress response

Saliva samples were collected from each participant ten times using Salivette collection tubes (Sarstedt, Rommelsdorf, Germany) to measure their cortisol level. The samples were stored at −22 °C within 2 h after collection until they were analyzed for cortisol concentration. The samples were later analyzed using electrochemiluminescence immunoassay (ECLIA, Cobas e 601, Roche Diagnostics). The lower sensitivity for cortisol was 0.500 nmol/L. Intra- and inter-assay variations were below 10%. One participant had one cortisol missing value at 0 min post stress manipulation, and it was imputed based on the mean of the rest cortisol values from this participant (Booij et al., 2013).

HR was measured using a Polar watch combined with a wireless chest HR sensor (Polar Vantage M, Polar Electro, Finland). The HR was continuously recorded during the experiment, and markers were made at each time point when the saliva sample was collected and between TSST periods. The HR was averaged across the segments based on those

markers: baseline, 5-min preparation, 5-min speech, 5-min arithmetic, and eight segments (0–5 min, 5–10 min, 10–15 min, 15–20 min, 20–30 min, 30–40 min, 40–50 min, 50–60 min) post the stress manipulation. One participant had missing HR data after 5 min post stress due to technical failure.

2.8. Data analysis

For the Response time (RT) analysis in Simon task, the first trials in each block, post-error trials (6.3%), trials exceeding 2.5 SD from the individual condition mean (2.2%), and error trials (5.6%) were excluded from subsequent analysis (Fischer et al., 2018). For EEG data analysis, the first trials in each block and error trials were excluded. Conflict adaptation (CA) values of N2 and P3 were all calculated using the following formula: CA = (cI-cC)-(iI-iC) [CA: conflict adaptation; cC: congruent precedes congruent trials; cI: congruent precedes incongruent trials, iC; incongruent precedes congruent trials; iI: incongruent precedes incongruent trials] (Fischer et al., 2018). This conflict adaption score reflects the degree of adjustment in cognitive control following trials with conflict (Clayson and Larson, 2011a).

For the Emotion regulation task, both subjective (ratings on arousal and valence) and EEG data were calculated for each condition. Difference scores [ΔLPP_{neu_neg} = LPP (view neutral) – LPP (view negative); ΔLPP_{reapp_neg} = LPP (cognitive reappraisal) – LPP (view negative); ΔLPP_{suppr_neg} = LPP (expressive suppression) – LPP (view negative)] were calculated to denote emotional sensitivity, cognitive reappraisal magnitude, and expressive suppression magnitude, respectively.

To check the effect of stress manipulation, we conducted repeated-measures ANOVAs for salivary cortisol and HR, with Time as a within-subject variable to evaluate stress responses. In addition, we calculated three scores of cortisol change to respectively measure the reactivity (up-regulation) magnitude, the recovery magnitude, and the recovery efficiency/slope of the stress responses [here we presented two measures of recovery, for a detailed review on the advantages and disadvantages among various measures of recovery, see Linden et al. (1997)]. The reasons for adopting those measures were that 1) the reactivity magnitude has been a widely used measure to indicate "cortisol increase" (e.g. Compton et al., 2013; Wu et al., 2017) and thus could be compared with previous studies; 2) the recovery magnitude was also a main interest of our study and has been reported before (e.g., Roy et al., 2001); 3) the recovery efficiency was indicative of fast return to equilibrium and better adaption, and we also had intensive measurements to enable the curve-fitting estimate.

Reactivity magnitude: $\Delta Up = CORT_{peak}\text{-}CORT_{baseline}$; $CORT_{peak}$ was individually identified for the maximum value after stress.

Recovery magnitude post stress: $\Delta Rec = CORT_{peak}\text{-}CORT_{post30}$; We chose the time point of 30 min post stress because the results showed that the most significant decrease was within 30 min after the stress (see Fig. 4).

Recovery efficiency/slope: Rec_slope was obtained with curve-fitting estimates. We explored both linear and logarithmic curve models to fit the cortisol value points (from the maximum post-stress point to the post60 point) for each participant. Since the logarithmic curve (Mean ± SD of R^2: 0.951 ± 0.049) fitted better than the linear (Mean ± SD of R^2: 0. 0.898 ± 0.055), $t(63) = 6.968$, $p < 0.001$, we used the estimates of the logarithmic curve in the present study. Rec_slope was the β value in the following formula: $y = \beta * \ln(x) + b$. More negative β suggested more efficient recovery. The cortisol data of eight participants were unable to be fitted into a curve due to the fluctuation of values without an apparent trend, which was excluded regarding the analysis of this measure.

To investigate the possibility of using cognitive control and emotion regulation to predict stress response pattern, we performed a series of multivariate regression models: (1) CA scores of N2 and P3 as predictors; (2) LPP indices as predictors. The outcome variables are the three stress response scores we calculated above. For significant regression results, we also tested whether the results remain stable after controlling for age, gender, and BMI.

The data analyses were performed using jamovi 1.6(The jamovi project, 2021), R 4.0(R Core Team, 2020), with packages afex (0.28–1; Singmann, 2018) and emmeans (1.6.1; Lenth, 2020), and IBM SPSS 21.0. The significance level was set at 0.05 and all reported *p*-values were two-tailed. Greenhouse–Geisser correction was applied in the case of violations of sphericity. Pairwise comparisons with Bonferroni adjustment were conducted for multiple comparisons. Effect sizes were reported using partial eta-squared (η_p^2).

3. Results

3.1. Conflict task: behavioral and neural data

3.1.1. Behavioral data

3.1.1.1. RT (see Fig. 2A). A repeated measures ANOVA with Previous-trial Congruency (congruent vs. incongruent) and Current-trial Congruency (congruent vs. incongruent) as within-subject factors yielded a significant main effect of Previous-trial Congruency, $F(1, 71) = 14.108$, $p < 0.001$, $\eta_p^2 = 0.166$, indicating that the responses were slowed by previous conflict (357 ms) in comparison with no previous conflict (348 ms), which was denoted as a post-conflict slowing (Verguts et al., 2011). A significant sequential modulation effect of the Simon effect was found in the interaction between Previous-trial Congruency and Current-trial Congruency, $F(1, 71) = 236.367$, $p < 0.001$, $\eta_p^2 = 0.769$, showing that the Simon effect was greatly reduced following conflict trials ($\Delta = -36$ ms, $t(71) = -8.414$, $p < 0.001$) compared to after non-conflict trials ($\Delta = 32$ ms, $t(71) = 8.260$, $p < 0.001$). This interaction effect reflects the predicted conflict adaptation.

3.1.1.2. Percent Error (see Fig. 2A). The repeated measures ANOVA on error rates generated a same trend as the RT, thus excluding potential speed-accuracy trade-offs. A significant main effect of Previous-trial Congruency was found, $F(1, 71) = 6.168$, $p = 0.015$, $\eta_p^2 = 0.080$, indicating that the previous conflict led to more errors. There was also a significant Previous-trial Congruency × Current-trial Congruency interaction effect, $F(1, 71) = 71.335$, $p < 0.001$, $\eta_p^2 = 0.501$, showing a sequential modulation of the Simon effect.

3.1.2. Electrophysiological data

3.1.2.1. N2 (see Fig. 2B). The same repeated measures ANOVA on N2 only observed a significant overall Simon effect: $F(1, 71) = 4.459$, $p = 0.038$, $\eta_p^2 = 0.059$, which indicated the incongruent trials had a significantly larger N2 (N2 is a negative component, so less positive amplitudes indicates larger N2) than congruent trials, while no sequential modulation effect was found (Previous-trial Congruency × Current-trial Congruency interaction: $p = 0.794$).

3.1.2.2. P3 (see Fig. 2C). A Simon effect was shown in larger P3 amplitudes in incongruent trials (7.49 μV) compared to congruent trials (6.83 μV), $F(1, 71) = 16.963$, $p < 0.001$, $\eta_p^2 = 0.193$. Further, a strong Previous-trial Congruency × Current-trial Congruency interaction was observed, $F(1, 71) = 17.365$, $p < 0.001$, $\eta_p^2 = 0.197$, indicating the sequential modulation effect. The P3 of current incongruent trials was significantly reduced when following a conflict, $t(71) = -2.775$, $p = 0.042$.

3.2. Emotion regulation task: subjective and neural data

3.2.1. Subjective data

The repeated measures ANOVA on subjective ratings with Condition as a within-subject factor revealed a significant effect of Condition on

valence (see Fig. 3), $F(2.722, 193.283) = 123.935$, $p < 0.001$, $\eta_p^2 = 0.636$. Participants' valence ratings were lower in the View negative condition compared to the View neutral condition, $t(71) = -16.609$, $p < 0.001$. The Cognitive reappraisal and Expressive suppression conditions both showed less negative affect compared to the View negative condition ($t(71) = 11.386$, $p < 0.001$; $t(71) = 8.433$, $p < 0.001$). Also, participants rated the valence higher, i.e. less negative, in the Cognitive reappraisal condition compared to the Expressive suppression conditions, $t(71) = 5.986$, $p < 0.001$, indicating a more successful emotion regulation effect on valence by Cognitive reappraisal than by Expressive suppression.

The one-way ANOVA also found a significant effect of Condition on arousal, $F(2.408, 170.955) = 61.444$, $p < 0.001$, $\eta_p^2 = 0.464$. The View negative condition induced significantly higher arousal than the View neutral condition, $t(71) = 12.465$, $p < 0.001$. The Cognitive reappraisal and Expressive suppression reduced the arousal compared to the View negative condition ($t(71) = -8.011$, $p < 0.001$; $t(71) = -6.758$, $p < 0.001$). However, no significant difference in arousal between those two emotion regulation strategies was found, $t(71) = -0.736$, $p > 0.9$.

3.2.2. Electrophysiological data

The emotion regulation effect was also reflected in the LPP ($F(3, 213) = 19.761$, $p < 0.001$, $\eta_p^2 = 0.218$). The post-hoc comparisons revealed that the View negative condition elicited significantly larger LPP amplitudes than the View neutral condition, $t(71) = 6.746$, $p < 0.001$. The Cognitive reappraisal and Expressive suppression conditions both lowered the LPP amplitudes compared to the View negative condition ($t(71) = -3.691$, $p = 0.003$; $t(71) = -4.502$, $p < 0.001$), in absence of a significant difference between the two regulation conditions, $t(71) = 0.743$, $p > 0.9$.

3.3. Physiological stress response

As expected, the repeated-measures ANOVAs for salivary cortisol revealed a significant effect of Time, $F(1.642, 116.589) = 75.239$, $p < 0.001$, $\eta_p^2 = 0.514$, see Fig. 4. Post-hoc analysis revealed that cortisol concentrations were significantly elevated from baseline to 0 min after the stressor ($t(71) = 7.924$, $p < 0.001$), from 0 to 5 min post stress ($t(71) = 8.165$, $p < 0.001$), from 5 to 10 min post stress ($t(71) = 4.032$, $p = 0.006$). Cortisol concentrations significantly declined from 10 to 15 min post stress ($t(71) = -7.558$, $p < 0.001$), from 15 to 20 min post stress ($t(71) = -6.200$, $p < 0.001$), from 20 to 30 min post stress ($t(71) = -7.139$, $p < 0.001$), from 30 to 40 min post stress ($t(71) = -8.302$, $p < 0.001$), and from 40 to 50 min post stress ($t(71) = -11.078$, $p < 0.001$), and stabilized from 50 to 60 min post stress ($t(71) = -2.492$, $p = 0.676$). Compared with baseline, 30 min post stress was still significantly higher than baseline ($t(71) = 5.744$, $p < 0.001$), but 40 min post stress was not significantly different from baseline ($t(71) = 3.257$, $p = 0.078$).

For sympathetic response, we also observed a significant effect of

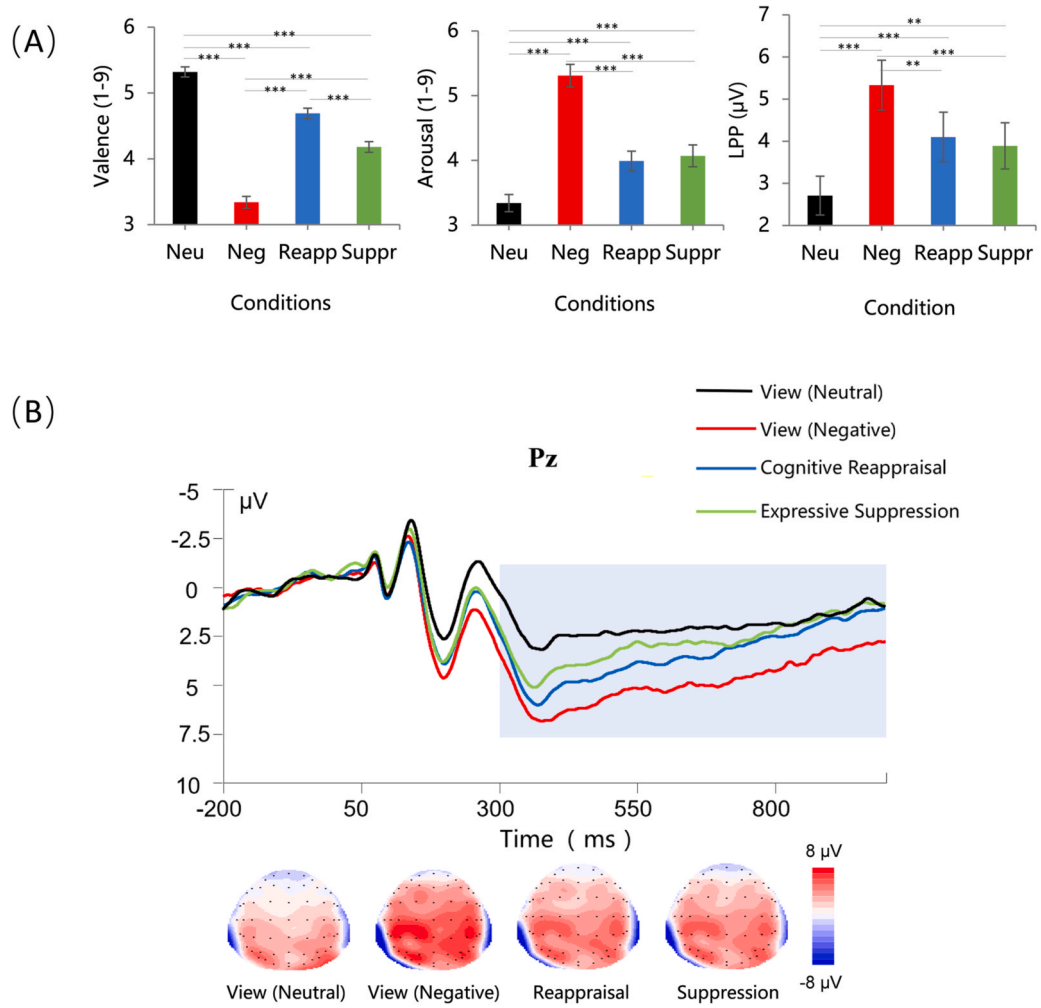

Fig. 3. (A) Difference in arousal, valence, and LPP among four task conditions [Neu: View (Neutral); Neg: View (Negative); Reapp: Cognitive Reappraisal; Suppr: Expressive Supression]. Lower values in valence indicate more negative affect. (B) Grand average LPP on Pz at the time window of 300–1000 ms. **: $p < 0.01$, ***: $p < 0.001$.

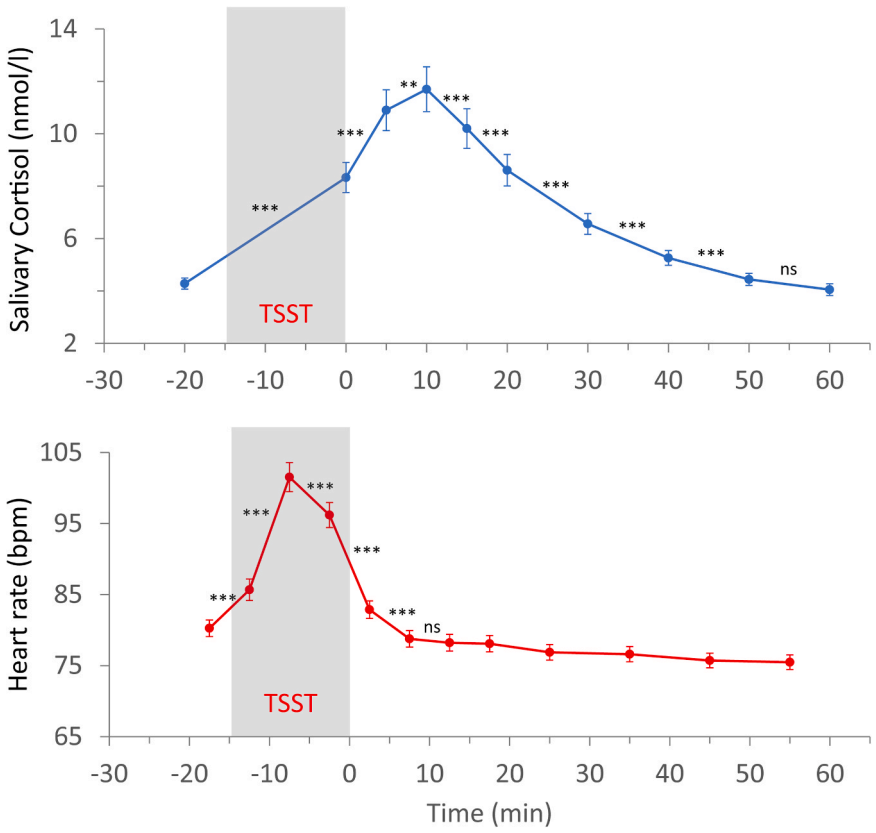

Fig. 4. Cortisol (above panel) and Heart Rate (bottom panel) change over time. The asterisks marked the significant difference between two consecutive time points regarding cortisol or two consecutive time segments regarding heart rate. ns: not significant, **: $p < 0.01$, ***: $p < 0.001$.

Time on HR, $F(2.238, 159.629) = 206.277$, $p < 0.001$, $\eta_p^2 = 0.747$, see Fig. 4. Pairwise post-hoc comparisons showed that HR significantly increased from baseline to the preparation phase of TSST ($t(70) = 6.986$, $p < 0.001$) and from the preparation to the speech phase of TSST ($t(70) = 11.789$, $p < 0.001$). Then HR started to decrease after the speech phase of TSST ($t(70) = -6.446$, $p < 0.001$), and quickly reduced within the first 0–5 min segment after stress ($t(70) = -14.035$, $p < 0.001$), but still higher compared with baseline ($t(70) = 5.664$, $p < 0.001$). Then HR slightly decreased in the next 5–10 min segment ($t(70) = -11.357$, $p < 0.001$), and was already lower than the baseline ($t(70) = -4.434$, $p = 0.002$). The 10–15 min segment was not significantly different from the 5–10 min segment ($t(70) = -2.245$, $p > 0.9$).

3.4. Regression analysis

Six multivariate regression models were performed to respectively test the main hypotheses of the current study, i.e., the possibility of using cognitive control or/and emotion regulation to predict the cortisol response pattern to psychosocial stress. The results of the multivariate regression analysis are shown in Table 1. Cognitive control (as indicated by CA scores) had no significant predictive effect on the cortisol response to the TSST ($ps > 0.3$). In contrast, the difference scores in LPP during the emotion regulation task significantly predicted all indices of the stress response, i.e., reactivity magnitude (ΔUp), recovery magnitude (ΔRec), and recovery efficiency (Rec_slope) [Note that supplementary analyses suggested those reactivity and recovery indices were highly correlated ($|rs > 0.80$, $ps < 0.001$)]. With a close inspection, we observed that larger reduced LPP magnitude by expressive suppression strategy ($LPP_{\Delta suppr\_neg}$) predicted stronger cortisol reactivity ($\beta = -0.413$, $t = -2.64$, $p = 0.010$) and recovery magnitude ($\beta = -0.499$, $t = -3.24$, $p = 0.002$), as well as greater recovery efficiency ($\beta = 0.507$, $t = 3.04$, $p = 0.004$), while the LPP reduced by cognitive reappraisal ($LPP_{\Delta reapp\_neg}$) predicted a trend of opposite pattern. The results for recovery magnitude and recovery efficiency remained significant after controlling for age, gender, and BMI. The results for reactivity magnitude were reduced to a non-significant trend ($p = 0.075$).

Table 1
Multivariate regression on indicators of cortisol reactivity and recovery.

	Reactivity magnitude (ΔUp)			Recovery magnitude (ΔRec)			Recovery efficiency (Rec_slope)		
Predictors	β	t	p	β	t	p	β	t	p
CA_N2	-0.306	-1.02	0.313	-0.245	-0.92	0.361	0.144	0.85	0.402
CA_P3	0.222	0.74	0.462	0.177	0.67	0.508	-0.119	-0.71	0.482
$LPP_{\Delta neu\_neg}$	0.278	1.98	0.052	0.262	1.89	0.063	-0.357	-2.51	**0.015**
$LPP_{\Delta reapp\_neg}$	0.239	1.62	0.111	0.247	1.69	0.096	-0.324	-2.06	**0.044**
$LPP_{\Delta suppr\_neg}$	-0.413	-2.64	**0.010**	-0.499	-3.24	**0.002**	0.507	3.04	**0.004**

CA: Conflict adaptation. **Bold:** $p < 0.05$. Note: Greater negativity in Rec_slope indicated better recovery efficiency; greater negativity in the ΔLPP indicated larger difference compared to the View negative condition.

3.5. Explorative predictive analysis on sympathetic stress response

We also conducted an explorative analysis of the predictive value of cognitive and affective self-regulation on the sympathetic stress response, i.e, heart rate change. The maximum increase value in HR was used as a dependent variable in the regression analysis. However, we did not observe any significant regression results either with cognitive components ($p = 0.748$) or affective components ($p = 0.091$).

4. Discussion

The current study aimed at investigating whether the neural activity of cognitive control and/or emotion regulation predicts the reactivity to and recovery from psychosocial stress. Our results show that the neural emotion regulation processing predicted subsequent cortisol response to stress. We further distinguished between the role of cognitive vs. affective components of self-regulation and found an effect specially for emotion regulation but not for the cognitive component.

In the emotion regulation task, we found that both cognitive reappraisal and expressive suppression reduced the LPP amplitudes to negative stimuli, which is consistent with previous emotion regulation studies (e.g., Moser et al., 2006, 2014; Thiruchselvam et al., 2011). More importantly, our results found that the LPP of the emotion regulation task is predictive of cortisol response indices regarding both reactivity to and recovery from an acute stressor. Particularly, we identified that the more successful reduction in LPP with suppression strategy predicted stronger cortisol response, while reduction in LPP with reappraisal predicted in the opposite trend. This pattern is inconsistent with Krkovic et al. (2018), which found that a relationship between habitual use of maladaptive emotion regulation strategies and blunted cortisol response. One possible explanation is that the habitual emotion regulation strategy score is not equalized with the LPPs regulated by emotion regulation. Alternatively, the difference in stress levels could also contribute to the distinct findings. Although we both used TSST to induce stress, participants in our study showed generally much higher cortisol increase (from baseline 4.28 ± 1.80 nmol/l to peak 11.68 ± 7.24 nmol/l).

As suggested by studies that combined ERP and functional magnetic resonance imaging (fMRI), the LPP evoked by emotional stimuli was related to the activation of the occipital, parietal and infratemporal area (Sabatinelli et al., 2007). The enhanced activation of visual cortex has been suggested to reflect motivational engagement to survival-related stimuli processing, and this motivated attention may have been the basis of the interconnections between the amygdala and the visual system (Bradley et al., 2003). During emotion regulation, the reduction in LPP is associated with reduced self-reported emotional experience (Hajcak and Nieuwenhuis, 2006), as well as the reduced activity of bilateral amygdala (Bunford et al., 2018). This decreased amygdala activity is regulated by the PFC that supports cognitive reappraisal of environmental stimuli (Hajcak et al., 2010), and two prefrontal-subcortical pathways (from ventrolateral PFC to nucleus accumbens/ventral amygdala) have been reported to play a major role in emotion regulation (Wager et al., 2008).

Importantly, the limbic pathways, which are connected to both PFC and hypothalamus, are also the underlying central nervous mechanisms for HPA response to stress (Feldman et al., 1995). Firstly, the PFC integrates the sensory information and appraises the meaning (e.g., whether it signals a threat). These appraisals further generate emotional responses through the pathway from the PFC to the limbic system. The limbic system is connected to the hypothalamus, and mediates the cortical response to stress with hypothalamic neurotransmitters (Ulrich-Lai and Herman, 2009). Thus, our findings that LPP predicted cortisol response might result from the overlapping neural network shared by stress response and emotion regulation.

Moreover, participants differed in the LPP evoked by emotion regulation strategies (cognitive reappraisal vs. expressive suppression) showed a distinct trend in cortisol change. This might be explained by the process model of emotion regulation proposed by Gross (1998b): cognitive reappraisal is an antecedent-focused strategy that alters emotional responses before their activation, while expressive suppression is a response-focused strategy that alters the expression of emotional responses afterwards. These results suggest that the neural signatures that predict cortisol response are specific to the regulation strategy.

As to the adaptation of cognitive control, we observed the conflict adaptation in behavior, which was reflected by the interaction between the congruency of previous trials and current trials. It's worth noting that the behavioral result showed a complete reversal after previous conflict. This reversed interference effect has also been reported in Fischer et al. (2015). They suggested that the reversal found in action-oriented individuals indicated an initiation of inhibitory control by the conflict signal. The inhibitory tagging prepares the individuals to use a diagonal response strategy after the conflict, which benefits incompatible trials after the conflict at the cost of slowing down compatible trials (Fischer et al., 2015). Therefore, we assume that the salient reversal observed in the present study might be due to the extremely strong suppression/inhibitory of the automatic response after conflict, which leads to the adoption of a conflict-triggered diagonal response strategy, i.e., the tendency to respond in a diagonal direction after previous conflict. Physiologically, the EEG results showed that the prefrontal N2 and centro-parietal P3 were sensitive to conflicts. For the neural activities during conflict task, N2 has been suggested to originate from the anterior cingulate cortex (ACC), which is responsible for conflict-monitoring (Larson et al., 2014; vanVeen and Carter, 2002) and the adaptation of cognitive control, as shown by larger N2 amplitudes on incongruent trials following congruent trials compared to following incongruent trials (Clayson and Larson, 2011a; Larson et al., 2012). Consistent with previous studies (Clayson and Larson, 2011a; Fischer et al., 2018), we also found a larger P3 amplitude for conflict trials and the sequential modulation effect. P3 in conflict tasks was suggested to reflect the allocation of attentional resources and is similar to the classic P3a (Clayson and Larson, 2011b). However, these components did not predict the cortisol response to the psychosocial stressor. It is inconsistent with previous studies in which they found associations between error processing and stress response (Cavanagh and Allen, 2008; Compton et al., 2013; Tops et al., 2006; Wu et al., 2017). The major difference between the studies is that error processing (mostly measured by ERN) reflects the neurocognitive indicator that ongoing events are evaluated as "worse" than expected (Hobson et al., 2014). Thus, it might get emotionally involved rather than a mere cognitive control processing (Moser et al., 2013).

It should be noted that self-regulation is an umbrella concept that incorporates many aspects. Regarding cognitive control, we only investigated conflict adaptation. While cognitive control is a general concept that describes a set of higher-order cognitive processes underlying goal-directed behaviors, adaptive control (or the adjustment of cognitive control) refers to dynamic regulation of the control processes in response to the changing context, which reflects an adaptive behavior (Braem et al., 2019). In the present study, we are particularly interested in how control dynamically adjusts to the changing environment. Conflict adaptation is the most widely used measure of adaptive control, which refers to an adaptive process that is triggered after a conflict. Although we hypothesized that the conflict adaption would be conceptually related to stress response, our findings did not support this hypothesis. As for emotion regulation, many other strategies (e.g., distraction) are also common in daily life. More systematic research is needed to clarify the relationship between self-regulation and the physiological system of stress. In addition, the nature of this predictive research makes it impossible to make causal inferences, so further work could make a factor design with manipulation of regulation strategies to investigate their different influences on the stress response.

In conclusion, we show here that the neural signature of emotion

regulation (as indexed by LPP) is predictive of the cortisol response to acute psychosocial stress. This effect might be due to overlapping neural circuits, i.e., the PFC-limbic system pathways shared by emotion regulation and HPA axis response to stress. To the best of our knowledge, this is the first study to measure the neural processing of both cognitive control and emotion regulation in the same sample to predict physiological stress response. We show that it is the affective component of self-regulation and its neural signature that predicts subsequent stress regulation. These outcomes might be applied in the preventive screening and intervention of stress-related disorders.

Acknowledgements

This work was supported by National Key Research and Development Plan under Grant Nos. 2018YFC0831101 and Shenzhen-Hong Kong Institute of Brain Science – Shenzhen Fundamental Research Institutions (NYKFKT2020003).

The Authors thank Shiwei Dai for his work during data collection. Author Lin thanks CAS-DAAD Joint Fellowship Programme for supporting her visiting research in Germany.

Conflict of interest

The authors have no conflict of interest to disclose.

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