Acute Stress Impairs Attentional Disengagement from Threat Stimuli

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Abstract

Acute stress enhances attentional bias toward threat stimuli, but it remains unclear whether acute stress enhances attentional orienting toward threat stimuli or impairs attentional disengagement from threat stimuli. The present study employed the Socially Evaluated Cold Pressor Task and dot-probe task, combined with event-related potential (ERP) technique, to investigate the cognitive mechanisms underlying the effects of acute stress on attentional bias toward threat stimuli. Following the Socially Evaluated Cold Pressor Task, state anxiety and cortisol levels increased significantly in individuals in the stress group. Regarding attentional bias, the stress group exhibited slower attentional disengagement from threat stimuli compared to the control group, with no significant difference in attentional orienting toward threat stimuli between the stress and control groups. ERP results revealed that threat stimuli elicited a more negative SPCN in the stress group than in the control group, with no significant difference in N2pc. The difference in cortisol increase between the stress and control groups was significantly positively correlated with the between-group differences in both N2pc and SPCN. These results indicate that acute stress enhances attentional bias toward threat stimuli because it impairs individuals' attentional disengagement from threat stimuli, which may be due to acute stress impairing the function of the frontoparietal network associated with attentional disengagement.

Full Text

Acute Stress Impairs Attentional Disengagement from Threat Stimuli*

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Abstract

Acute stress enhances attentional bias toward threat stimuli, but it remains unclear whether this effect reflects enhanced attentional orienting to threat or impaired attentional disengagement from threat. The present study employed the Socially Evaluated Cold Pressor Task (SECPT) and dot-probe task, combined with event-related potential (ERP) technology, to investigate the cognitive mechanisms underlying the effect of acute stress on attentional bias to threat. Following the SECPT, individuals in the stress group exhibited significantly elevated state anxiety and cortisol concentrations. In terms of attentional bias, the stress group showed slower attentional disengagement from threat compared to the control group, with no significant group differences in attentional orienting to threat. ERP results revealed that threat stimuli elicited more negative SPCN amplitudes in the stress group than in the control group, while no significant differences were observed for N2pc. The difference in cortisol increments between groups showed significant positive correlations with the group differences in both N2pc and SPCN. These findings suggest that acute stress enhances attentional bias to threat by impairing attentional disengagement from threat, possibly due to acute stress-induced dysfunction of the frontoparietal network associated with attentional disengagement.

Keywords: Acute stress; Attentional orienting; Attentional disengagement; N2pc; SPCN

1 Introduction

Acute stress influences attentional bias toward threat stimuli (Jiang et al., 2017; Sanger, Bechtold, Schoofs, Blaszkewicz, & Wascher, 2014; Weymar, Schwabe, Low, & Hamm, 2012). However, the mechanism underlying this effect remains unclear—specifically, whether acute stress enhances early attentional orienting to threat or impairs later attentional disengagement from threat. Therefore, the present study investigates the cognitive mechanisms through which acute stress affects attentional bias to threat.

Attentional bias to threat refers to differential attentional allocation between threat and neutral stimuli, representing a preferential processing of threat. This bias manifests primarily as facilitated attentional orienting to threat and difficulty in attentional disengagement from threat (Koster, Crombez, Verschuere, Van Damme, & Wiersema, 2006; Zhang, Luo, Zhao, Chen, & Li, 2014). Facilitated attentional orienting to threat denotes that individuals can shift attention to threat stimuli or threatening locations more rapidly than to neutral stimuli (Carlson & Reinke, 2008). Difficulty in attentional disengagement from threat means that, compared with neutral stimuli, individuals have greater difficulty

terminating attentional processing of threat stimuli or threatening locations (Fox, Russo, Bowles, & Dutton, 2001).

The dot-probe task is frequently used to measure attentional orienting and disengagement to threat. In this task, attention allocation conditions (threat-neutral stimulus pairs) and non-attention allocation conditions (threat-threat pairs, neutral-neutral pairs) serve as cues, followed by a neutral target appearing randomly at either the threat or neutral cue location, to which participants respond. In attention allocation conditions, trials where the target appears at the threat cue location are termed congruent conditions, while those where it appears at the neutral cue location are termed incongruent conditions. In non-attention allocation conditions, participants show no attentional bias because the cue attributes are identical (Koster, Crombez, Verschuere, & De Houwer, 2004). Facilitated attentional orienting is evidenced by slower responses to targets in non-attention allocation conditions compared to congruent conditions, whereas difficulty in attentional disengagement is indicated by slower responses in incongruent conditions compared to non-attention allocation conditions (Salemink, van den Hout, & Kindt, 2007).

Previous research using event-related potential (ERP) technology has found that attentional orienting and disengagement to threat are associated with N2pc (N2-posterior-contralateral) and SPCN (sustained posterior contralateral negativity), respectively (Holmes, Bradley, Kragh Nielsen, & Mogg, 2009; Holmes, Mogg, de Fockert, Nielsen, & Bradley, 2014; Kappenman, Farrens, Luck, & Proudfit, 2014; Kappenman, MacNamara, & Proudfit, 2015). N2pc is a difference wave representing the amplitude discrepancy between electrodes contralateral and ipsilateral to a visual target (e.g., if a threat stimulus appears on the right, left hemisphere electrodes are contralateral and right hemisphere electrodes are ipsilateral), distributed over posterior brain regions with maximal amplitude around P7/P8 and PO7/PO8 electrodes, typically emerging approximately 180–300 ms after target onset. Its amplitude reflects attentional selection of target stimuli (Holmes et al., 2009; Holmes et al., 2014; Kappenman et al., 2014; Kappenman et al., 2015). Research has shown that threat stimuli elicit more negative N2pc amplitudes, indicating that threat stimuli capture attention more rapidly (Holmes et al., 2009; Holmes et al., 2014; Kappenman et al., 2014; Kappenman et al., 2015). This attentional orienting to threat may be closely linked to the amygdala-anterior cingulate network centered on the amygdala (Carlson et al., 2012; Carlson & Reinke, 2008).

SPCN, like N2pc, is a lateralized posterior ERP component that is more negative contralaterally, typically appearing after 300 ms post-stimulus. SPCN amplitude reflects the process of attentional maintenance toward target stimuli (Holmes et al., 2009; Holmes et al., 2014). Studies have also found that threat stimuli evoke larger SPCN amplitudes, suggesting attentional maintenance to threat—meaning individuals have greater difficulty disengaging attention from threat stimuli (Holmes et al., 2009; Holmes et al., 2014; Meconi, Luria, & Sessa, 2014; Sessa, Luria, Gotler, Jolicoeur, & Dell' acqua, 2011). This attentional

disengagement from threat may be associated with the frontoparietal network, including the dorsolateral prefrontal cortex and lateral posterior parietal cortex (Fecteau & Munoz, 2006; Gottlieb, 2007).

Acute stress refers to a nonspecific response that occurs when unpredictable and uncontrollable environmental demands exceed an individual' s natural regulatory capacity (Koolhaas et al., 2011). Subjectively, acute stress significantly increases negative emotions such as anxiety. Physiologically, acute stress activates the autonomic nervous system, triggering catecholamine secretion (e.g., norepinephrine), and interacts with glucocorticoid release (e.g., cortisol) resulting from Hypothalamic-Pituitary-Adrenal (HPA) axis activation, thereby influencing neural and physiological activity (Ulrich-Lai & Herman, 2009). These neurophysiological changes induced by acute stress exert specific effects on brain regions including the amygdala-anterior cingulate network and the frontoparietal network, both of which are closely associated with attentional bias to threat (Arnsten, 2009; Hermans et al., 2011).

On one hand, acute stress enhances activity in the amygdala-anterior cingulate network (Cousijn et al., 2010), facilitating threat detection (Debiec & LeDoux, 2006). Cousijn et al. found that acute stress amplifies phasic amygdala responses (Cousijn et al., 2010). Behavioral research by Rued et al. demonstrated that individuals in an acute stress state detect threat stimuli faster than non-threat stimuli but with lower accuracy, showing a behavioral response advantage for threat (Rued, Hilmert, Strahm, & Thomas, 2019). ERP studies have also revealed that acute stress enhances early sensory input to threat stimuli, manifested as increased amplitude of the early attention-related N1 component (Löw, Weymar, & Hamm, 2015; Shackman, Maxwell, McMenamin, Greischar, & Davidson, 2011). Attentional orienting to threat is closely associated with the amygdala-anterior cingulate network (Carlson et al., 2012; Carlson & Reinke, 2008), and acute stress increases activity in this network. Based on this evidence, we hypothesize that acute stress will enhance attentional orienting to threat.

On the other hand, acute stress reduces activity in the frontoparietal network (Arnsten, 2009; Qin, Hermans, van Marle, Luo, & Fernandez, 2009), impairing the allocation of attentional control resources (Meconi et al., 2014). Qin et al. found that stress significantly decreased activation in prefrontal and parietal cortical regions (Qin et al., 2009). Similar results have been observed in ERP studies of stress-related disorders such as anxiety and depression. Meconi et al. found that individuals' anxiety levels increased their difficulty in disengaging attention from untrustworthy faces (threat stimuli), with higher anxiety individuals showing larger SPCN amplitudes to untrustworthy faces compared to low-anxiety individuals (Meconi et al., 2014). Attentional disengagement from threat is associated with the frontoparietal network (Fecteau & Munoz, 2006; Gottlieb, 2007), and acute stress impairs activity in this network. Therefore, we hypothesize that acute stress will impair attentional disengagement from threat.

In summary, acute stress may differentially affect distinct manifestations of at-

tentional bias. Specifically, acute stress may enhance attentional orienting to threat while impairing attentional disengagement from threat. Based on this framework, the present study employed the Socially Evaluated Cold Pressor Task to induce stress responses, followed by a dot-probe task combined with ERP technology to investigate the cognitive mechanisms through which acute stress influences attentional bias to threat. We hypothesized that acute stress would enhance attentional orienting to threat and impair attentional disengagement from threat. If acute stress enhances attentional orienting to threat, the stress group should show faster attentional orienting than the control group behaviorally, manifested as a larger difference in reaction times between non-attention allocation and congruent conditions; ERP results should show more negative N2pc amplitudes elicited by threat stimuli in the stress group. If acute stress impairs attentional disengagement from threat, the stress group should show slower disengagement than the control group behaviorally, reflected as a larger difference in reaction times between incongruent and non-attention allocation conditions; ERP results should reveal more negative SPCN amplitudes elicited by threat stimuli in the stress group.

2.1 Subjects

Considering the influence of female menstrual cycles on stress responses (Fernández et al., 2003) and the inability to recruit a sufficient number of female participants across different menstrual cycle phases to meet experimental conditions, the present study conveniently sampled 44 male adult participants (24 in the stress group and 20 in the control group), aged 18–21 years, right-handed, self-reported free of physical illness and mental disorders, and with no steroid medication use within one month prior to the experiment. All participants had normal or corrected-to-normal vision. The study was approved by the local ethics committee. Participation was voluntary, and informed consent was obtained before the experiment.

For stable and reliable ERP results, averaging over 100 trials per condition is necessary (Boudewyn, Luck, Farrens, & Kappenman, 2018). Therefore, during data analysis, 8 participants were excluded because they had fewer than 100 artifact-free trials available for averaging in each condition after EEG artifact detection. The final sample comprised 36 participants (18 in each group).

2.2.1 Experimental Stimuli and Questionnaires

All experimental stimuli were presented on a 17-inch display (resolution: 1024×768, refresh rate: 60 Hz) with a black background. Emotional faces were selected from the Chinese Affective Face Picture System (CAFPS) (Wang & Luo, 2005), including neutral and fearful faces in grayscale. Each image subtended 7°×9° of visual angle. The experiment comprised a total of 100 emotional faces, including 50 fearful faces and 50 neutral faces, with equal numbers of male and female faces, and each face identity appearing only once.

The State-Trait Anxiety Inventory (STAI), developed by Spielberger et al. (1970), comprises two subscales: State Anxiety (S-AI) and Trait Anxiety (T-AI), each containing 20 items rated on a 4-point scale ranging from "not at all" to "very much so." Half of the items assess positive emotions and half assess negative emotions, with positive emotion items reverse-scored. Higher total scores indicate greater anxiety severity (Spielberger, 1970). In the present study, the Cronbach' s coefficient for the full scale was 0.89, and for the state and trait anxiety subscales were 0.93 and 0.79, respectively.

2.2.2 Experimental Design and Procedure

The experiment employed a mixed design of 2 (Group: stress/control) × 2 (Cue-target location: congruent/incongruent), with group as a between-subjects factor. To avoid the influence of individual cortisol circadian rhythms on the results, all sessions were scheduled between 13:30 and 18:30 (Luo et al., 2018). Four days before the experiment, participants were contacted by phone and instructed to abstain from alcohol for 72 hours, refrain from smoking for 24 hours, avoid vigorous exercise on the experimental day, and consume no food (except water) for 2 hours prior to the experiment.

The experimental procedure (illustrated in Figure 1 Figure 1: see original paper) was as follows: Upon arrival at the laboratory, participants rested quietly for 5 minutes after reading and signing the informed consent form in a private room. They then completed the first State-Trait Anxiety Inventory and provided saliva sample S1 (-85 min) using German SARSTEDT saliva collection tubes, which required placing a cotton swab in the mouth and rotating it for 2 minutes without chewing. After an additional 20-minute rest, participants completed the second state anxiety questionnaire and provided saliva sample S2 (-55 min). Participants were then randomly assigned to either the stress or control group and underwent the SECPT or corresponding control task (-5 min). Immediately after the task, they completed the third state anxiety questionnaire and provided saliva sample S3 (0 min). Subsequently, participants performed the dot-probe task while behavioral and EEG data were recorded simultaneously. Following the dot-probe task, participants completed the fourth state anxiety questionnaire and provided saliva sample S4 (40 min). After a 20-minute break, they completed the fifth state anxiety questionnaire and provided saliva sample S5 (70 min) before concluding the session.

Socially Evaluated Cold Pressor Task (SECPT): This task was developed by Schwabe et al. (Schwabe, Haddad, & Schachinger, 2008; Schwabe & Schachinger, 2018). In the stress condition, participants were required to maintain gaze on a video camera while two experimenters in medical attire stood before them with serious expressions, continuously evaluating their performance. The experimenters positioned themselves at a distance that formed an equilateral triangle with the participant and camera, ensuring participants could see the experimenters peripherally while looking at the camera. Participants then immersed their foot (including the ankle) in ice water (0–2°C). Feet rather

than hands were immersed to prevent excessive cooling that might affect button-press responses. Participants were unaware of the exact immersion duration but could remove their foot at any time, with a maximum duration of 3 minutes, at which point the experimenter prompted removal. During the stress task, experimenters remained silent and avoided any form of positive reinforcement (e.g., smiling, nodding). In the control condition, experimenters wore casual clothing, maintained neutral expressions, and no camera was present. Participants immersed their foot (including ankle) in warm water (36–38°C) for at least 3 minutes.

Dot-probe task: A modified version of the dot-probe task was used to measure attentional bias. In each trial (see Figure 1(b)), a fixation cross "+"was presented at the center of the screen for 750–1250 ms, followed by an emotional face pair comprising randomly combined fearful and neutral faces presented for 400–600 ms. The face pairs included three types: mixed pairs (fearful-neutral/neutral-fearful), neutral pairs (neutral-neutral), and fearful pairs (fearful-fearful). All face pairs were gender-matched, with each face appearing in random positions no more than nine times. A target stimulus then appeared for 400 ms to the left or right of the fixation point. The target consisted of two dots arranged horizontally or vertically, each subtending 0.5°×0.5°. After the target disappeared, participants were required to indicate the dot orientation (vertical or horizontal) by pressing the 'k' or 'l' key within 1100 ms while maintaining accuracy. Response keys were counterbalanced across participants. No response within 1100 ms was counted as an error, and the next trial began automatically. There were 360 trials with mixed pairs and 60 trials each for neutral and fearful pairs, totaling 480 trials. Among the 360 mixed-pair trials, 180 were congruent conditions (target replacing the fearful face location) and 180 were incongruent conditions (target replacing the neutral face location). Participants' eyes were level with the screen center at a viewing distance of approximately 60 cm. Stimulus presentation and behavioral data collection were controlled using E-Prime software.

2.3.1 Saliva Storage and Assay

Saliva samples were stored frozen at -20°C. Before assay, samples were thawed and centrifuged at 3000 rpm for 10 minutes. Salivary cortisol concentrations were determined using electrochemiluminescence immunoassay (Cobas e 601, Roche Diagnostics, Numbrecht, Germany). The assay sensitivity was 1.5 nmol/L (lower limit).

2.3.2 Behavioral Data Analysis

For the dot-probe task, behavioral data for each participant were processed as follows: (1) trials with incorrect responses were excluded; (2) trials with reaction times less than 200 ms or greater than 1000 ms were excluded; (3) trials with reaction times exceeding 3 standard deviations from the mean were

excluded. Following these procedures, the exclusion rate ranged from 0.63% to 13.75% across participants, with an average of 4.91%. For participants included in the analysis, attentional orienting and disengagement scores were calculated and submitted to a 2 (Group: stress/control) × 2 (Attentional bias: orienting/disengagement) repeated-measures ANOVA. Attentional orienting was computed as the difference between mean reaction times in non-attention allocation and congruent conditions; attentional disengagement was computed as the difference between mean reaction times in incongruent and non-attention allocation conditions (Salemink et al., 2007).

2.3.3 EEG Recording and Data Analysis

EEG was recorded using the NeuroScan ERP recording and analysis system with a 64-channel electrode cap based on the extended international 10-20 system. The left mastoid served as the online reference. Vertical electrooculogram (VEOG) and horizontal electrooculogram (HEOG) were recorded bipolarly, with VEOG electrodes placed above and below the left eye and HEOG electrodes placed lateral to the outer canthi of both eyes. The ground electrode was located at the midpoint between FPz and Fz on the anterior scalp. The sampling rate was 1000 Hz with a bandpass filter of 0.05–100 Hz, and electrode impedances were maintained below 5 kΩ.

Continuous EEG data were analyzed offline using MATLAB (R2013b), EEGLAB (v14.1.2), and ERPLAB (v7.0.0). Data were re-referenced to the average of bilateral mastoids and filtered with a high-pass filter (0.1 Hz, 24 dB/octave) and low-pass filter (30 Hz, 48 dB/octave). The data were segmented into epochs from 200 ms before to 600 ms after the onset of emotional face pairs, with the -200 to 0 ms interval serving as the baseline. Blink artifacts were corrected automatically using the Moving Window Peak-to-Peak method (Width: 200 ms; Step Size: 100 ms) and Simple Voltage Threshold in ERPLAB (v7.0.0). Movement artifacts were rejected with a threshold of ±100 V, and the rejection threshold for horizontal EOG (HEOG) was ±80 V.

For all ERP analyses, only EEG data from correct trials in mixed pairs were included in the averaging. Based on previous research, N2pc and SPCN were selected as indices of attentional orienting and disengagement (Holmes et al., 2009; Holmes et al., 2014; Kappenman et al., 2014; Kappenman et al., 2015; Meconi et al., 2014). N2pc and SPCN amplitudes were calculated as the mean difference between contralateral and ipsilateral electrodes at posterior scalp sites P7/P8 and PO7/PO8. Because subjective selection of time windows for computing mean amplitudes may introduce substantial bias, we used component area measures (Sawaki, Geng, & Luck, 2012). Specifically, N2pc amplitude was defined as the area of negative amplitude in the contralateral-minus-ipsilateral difference wave between 180–300 ms, while SPCN was the area of negative amplitude in the same difference wave between 300–600 ms. As area measures are always positive and thus violate normality assumptions, traditional parametric tests were inappropriate. Therefore, we employed more suitable non-parametric

permutation tests (Gaspelin & Luck, 2018; Sawaki et al., 2012; Sun et al., 2018; Wang et al., 2016).

The main steps of non-parametric permutation testing are as follows: First, the laterality of threat stimulus trials is randomly recoded, such that the ipsilateral side of threat stimuli is randomly assigned as contralateral or ipsilateral. Second, the recoded EEG data are averaged, and contralateral-minus-ipsilateral difference waves are computed. Finally, the area of negative amplitude within the corresponding time window is calculated from the difference wave. This procedure is repeated 1000 times to generate a null distribution of the test statistic. If the observed amplitude value exceeds the 95th percentile of the null distribution amplitude values, the observation is considered to reflect genuine physiological responses rather than noise in the data. Additionally, non-parametric permutation tests can assess group differences using the same principles and procedures (Gaspelin & Luck, 2018; Sun et al., 2018; Wang et al., 2016). For group difference permutation tests, group membership is randomly recoded (i.e., stress group randomly assigned as stress or control group), and group differences are computed. Repeating this process 1000 times generates a null distribution for group differences. If the observed group difference amplitude value exceeds or falls below the 95th percentile of the two-tailed null distribution, the group difference is considered statistically significant.

3.1 Questionnaire

For trait anxiety scores, no significant difference was found between the stress group (42.72 ± 4.40) and control group (42.27 ± 5.54), $t(2, 34) = 0.27$, $p = 0.79$. For state anxiety scores, a 2 (Group: stress/control) × 5 (Measurement time: -85 min/-55 min/0 min/40 min/70 min) repeated-measures ANOVA revealed (see Figure 2 Figure 2: see original paper) a significant main effect of measurement time, $F(4, 24) = 3.27$, $p = 0.046$, $^{2}p = 0.35$, but no significant main effect of group, $F(1, 27) = 0.83$, $p = 0.37$, and no significant Group × Time interaction, $F(4, 108) = 0.56$, $p = 0.60$. Based on our specific hypothesis that state anxiety would increase significantly after stress induction, we conducted separate analyses for the 0-min time point (immediately after SECPT). State anxiety was significantly higher in the stress group (43.93 ± 1.12) than in the control group (40.21 ± 1.16), $F(1, 27) = 5.33$, $p = 0.03$, $^{2}p = 0.16$. Additionally, no significant group differences in state anxiety were observed at -85 min ($F(1, 27) = 0.83$, $p = 0.37$) or -55 min ($F(1, 27) = 0.39$, $p = 0.54$) before the SECPT. These results indicate that the SECPT successfully increased state anxiety.

3.2 Cortisol

For cortisol concentrations, a 2 (Group: stress/control) × 5 (Measurement time: -85 min/-55 min/0 min/40 min/70 min) repeated-measures ANOVA revealed (see Figure 2(b)) a significant main effect of measurement time, $F(4, 31) = 5.71$, $p = 0.006$, $^{2}p = 0.42$, but no significant main effect of group, $F(1, 34) = 0.29$,

$p = 0.60$. The Group × Time interaction was significant, $F(4, 136) = 7.50$, $p = 0.001$, $^2p = 0.18$. Further analysis showed that at 40 min, cortisol levels were significantly higher in the stress group (9.74 ± 1.36) than in the control group (5.25 ± 1.36), $F(1, 34) = 5.44$, $p = 0.03$, $^2p = 0.14$. At -85 min, cortisol levels were significantly lower in the stress group (4.10 ± 0.75) than in the control group (6.47 ± 0.75), $F(1, 34) = 5.01$, $p = 0.03$, $^2p = 0.13$.

To further confirm the effectiveness of the stress induction, paired t-tests were conducted on cortisol concentrations at 0 min and 40 min for each group. The stress group showed significantly higher cortisol at 40 min (9.74 ± 7.81) than at 0 min (8.01 ± 6.96), $t(1, 17) = -2.45$, $p = 0.03$, Cohen' s $d = 0.23$. The control group showed no significant difference between cortisol levels at 40 min (5.25 ± 2.42) and 0 min (5.63 ± 2.19), $t(1, 17) = 1.088$, $p = 0.292$. These results demonstrate that the SECPT significantly increased cortisol concentrations in the stress group.

3.3 Behavioral Results

Figure 2. State anxiety scores (a) and cortisol concentrations (b). * indicates $p < 0.05$.

For behavioral results, analysis of attentional bias scores (see Figure 3 Figure 3: see original paper) revealed no significant main effect of group, $F(1, 34) = 0.85$, $p = 0.36$, no significant main effect of attentional bias type, $F(1, 34) = 0.49$, $p = 0.49$, and a marginally significant Group × Attentional bias interaction, $F(1, 34) = 2.87$, $p = 0.099$. Therefore, we conducted separate comparisons of attentional orienting and disengagement scores between groups. The stress group showed marginally slower attentional disengagement (5.72 ± 3.69 ms) than the control group (-4.17 ± 3.69 ms), $F(1, 34) = 3.59$, $p = 0.07$, $^2p = 0.10$. No significant group difference was found for attentional orienting, $F(1, 34) = 1.44$, $p = 0.24$.

3.4.1 N2pc

Independent samples t-tests on N2pc amplitudes (see Figure 3(b)) revealed no significant difference between the stress group (0.14 ± 0.15) and control group (0.13 ± 0.11), $t(2, 34) = 0.17$, $p = 0.87$. This indicates that the stress and control groups did not differ in attentional orienting to threat.

Non-parametric permutation tests also indicated that the observed N2pc values for both stress and control groups (see Figure 4 Figure 4: see original paper, red lines) were statistically significant (exceeding the 95th percentile of the null distribution, yellow area), demonstrating that threat stimuli effectively elicited N2pc in both groups. However, the group difference in N2pc between stress and control groups was not statistically significant (see Figure 4(b)).

3.4.2 SPCN

Independent samples t-tests on SPCN amplitudes (see Figure 3(b)) revealed that the stress group (0.42 ± 0.44) was significantly larger than the control group (0.19 ± 0.13), t(2, 34) = 2.13, p = 0.04, Cohen' s d = 0.71. This indicates a significant group difference in attentional disengagement from threat.

Non-parametric permutation tests also showed that the observed SPCN values for both stress and control groups (see Figure 4(a), red lines) were statistically significant (exceeding the 95th percentile of the null distribution, yellow area), indicating that threat stimuli effectively elicited SPCN in both groups. Moreover, the group difference in SPCN (stress group minus control group, see Figure 4(b), red line) was also statistically significant (exceeding the 95th percentile of the null distribution, yellow area), demonstrating that the stress group elicited larger SPCN than the control group.

3.5 Correlation

To further investigate the effects of acute stress on attentional orienting and disengagement, we examined correlations between the group difference in cortisol increments and the group differences in N2pc and SPCN. The group difference in cortisol increment was computed as the stress group' s increment minus the control group' s increment, with cortisol increment defined as the difference between cortisol concentrations at 40 min and 0 min. Group differences in N2pc and SPCN were computed as differences in component area between stress and control groups. Results revealed significant positive correlations between the group difference in cortisol increment and group differences in both N2pc (r = 0.49, p = 0.04) and SPCN (r = 0.47, p = 0.05).

3.6 Time Effects

Acute stress-induced activation of the sympathetic nervous system and HPA axis exhibits important time-dependent characteristics in modulating brain cognitive functions (de Kloet, Joels, & Holsboer, 2005; Hermans, Henckens, Joels, & Fernandez, 2014; Luo, Lin, Wu, & Qin, 2013). Therefore, to further examine the differential time-dependent effects of acute stress-induced sympathetic and HPA axis activation on attentional orienting and disengagement, we divided the 480 experimental trials into two equal halves based on temporal progression, with 240 trials in each half, including 90 congruent and 90 incongruent trials, and 30 neutral and 30 mixed pairs in each half. For behavioral data, a 2 (Group: stress/control) × 2 (Trial time: first half/second half) × 2 (Attentional bias: orienting/disengagement) repeated-measures ANOVA revealed no significant main effects of group, F(1, 68) = 0.69, p = 0.41, trial time, F(1, 68) = 0.01, p = 0.94, or attentional bias, F(1, 34) = 0.46, p = 0.50, and no significant three-way interaction, F(1, 34) = 0.37, p = 0.55.

For ERP data, separate 2 (Group: stress/control) × 2 (Trial time: first

half/second half) repeated-measures ANOVAs were conducted for N2pc and SPCN. For N2pc, neither the main effect of group, $F(1, 34) = 0.005$, $p = 0.94$, nor trial time, $F(1, 34) = 0.02$, $p = 0.90$, was significant, but the Group × Trial time interaction was significant, $F(1, 34) = 9.23$, $p = 0.01$, $^{2}p = 0.21$. However, follow-up analyses revealed no significant group differences in N2pc for either the first half, $F(1, 34) = 2.63$, $p = 0.11$, or second half of trials, $F(1, 34) = 2.06$, $p = 0.16$. For SPCN, neither the main effect of group, $F(1, 34) = 2.59$, $p = 0.12$, nor trial time, $F(1, 34) = 0.01$, $p = 0.91$, was significant, but the Group × Trial time interaction was marginally significant, $F(1, 34) = 3.94$, $p = 0.06$, $^{2}p = 0.10$. Follow-up analyses showed no significant group difference in SPCN during the first half of trials, $F(1, 34) = 0.02$, $p = 0.90$, but significantly larger SPCN in the stress group (0.27 ± 0.06) than in the control group (0.09 ± 0.06) during the second half of trials, $F(1, 34) = 4.92$, $p = 0.03$, $^{2}p = 0.13$. These results suggest that acute stress effects on attentional bias occurred primarily during the second half of the dot-probe task, which aligns temporally with HPA axis activation. This indicates that acute stress effects on attentional bias are mainly driven by HPA axis activation, which impairs attentional disengagement from threat, consistent with our earlier findings.

Discussion

The present study investigated the effects of acute stress on attentional orienting and disengagement from threat stimuli. The findings demonstrate that the SECPT successfully induced stress responses, with the stress group showing significantly higher state anxiety scores and cortisol concentrations than the control group after the task. Regarding attentional bias, no significant group differences were observed in attentional orienting, while the stress group exhibited slower attentional disengagement than the control group. ERP results showed no group differences in N2pc, but the stress group generated more negative SPCN amplitudes than the control group. Correlational analyses revealed significant positive relationships between the group difference in cortisol increment and group differences in both N2pc and SPCN. In terms of time effects, SPCN was more negative in the stress group than in the control group during the second half of trials. These results indicate that acute stress affects attentional bias to threat, primarily by impairing attentional disengagement from threat, making it more difficult for individuals to disengage from threat stimuli and resulting in longer attentional maintenance on threat.

Cortisol concentration and subjective state measures confirmed that the SECPT effectively induced stress responses. Cortisol concentrations were significantly higher in the stress group than in the control group. Previous research has shown that salivary cortisol concentrations peak approximately 25 minutes after SECPT and that stress effects can persist for 35–60 minutes (Luo et al., 2018; Schwabe et al., 2008; Schwabe, Hoffken, Tegenthoff, & Wolf, 2013; Schwabe, Joels, Roozendaal, Wolf, & Oitzl, 2012; Schwabe & Schachinger, 2018), which aligns with our cortisol measurements at 40 minutes post-SECPT. However, at

-85 min before the SECPT, cortisol concentrations were significantly lower in the stress group than in the control group, possibly due to individual differences in adaptation to the laboratory environment. Further analysis of cortisol concentrations after a 20-minute rest period (-55 min) showed no significant group differences, suggesting that cortisol levels returned to baseline after participants adapted to the laboratory setting. Therefore, the cortisol results at -85 min did not affect the stress induction outcomes. Regarding subjective state measures, anxiety levels were significantly higher in the stress group than in the control group. Garrett et al. used the state anxiety questionnaire to assess emotional experience in stressful environments and found that state anxiety scores were significantly correlated with stress experience (Garrett, Gonzalez-Garzon, Foulkes, Levita, & Sharot, 2018), consistent with our finding of significantly higher state anxiety scores in the stress group immediately after SECPT (0 min). Unlike previous studies, we did not observe a significant Group × Measurement time interaction, possibly because state anxiety scores in the stress group were higher than in the control group before the SECPT, but these differences were not statistically significant.

Attentional bias and ERP findings revealed that acute stress does not enhance attentional orienting to threat but rather makes disengagement from threat more difficult and prolongs attentional maintenance on threat stimuli. Behaviorally, no group differences were observed in attentional orienting, while the stress group showed slower attentional disengagement than the control group. These control group findings are consistent with Armstrong and Olatunji (2012), who found that individuals maintain attention longer on threat stimuli in visual search tasks, leading to slower attentional disengagement. In the present study, the SECPT effectively induced an anxious state that impaired attentional disengagement from threat. Research on stress-related disorders such as anxiety and depression has similarly found that high-anxiety individuals have greater difficulty disengaging attention from threat stimuli (Taylor, Cross, & Amir, 2016). Additionally, depressed individuals do not show faster attentional orienting to threat stimuli such as sad faces (Cisler & Koster, 2010), but they do concentrate attention on threat stimuli shortly after onset (Peckham, McHugh, & Otto, 2010). Furthermore, previous research has shown that threat stimulus duration influences attentional bias. When threat stimuli are presented very briefly (e.g., 100 ms), individuals typically show facilitated attentional orienting; when presentation durations are relatively long (e.g., 500 ms), difficulty in attentional disengagement is typically observed (Cooper & Langton, 2006). In the present study, with threat stimuli presented randomly for 400-600 ms, individuals showed difficulty in attentional disengagement but no difference in attentional orienting, consistent with previous research.

ERP results showed no significant group differences in N2pc, but more negative SPCN in the stress group than in the control group. Additionally, significant positive correlations were found between the group difference in cortisol increment and group differences in both N2pc and SPCN. Previous research has demonstrated that N2pc reflects attentional shifting to stimuli, while SPCN re-

flects attentional maintenance of information (Holmes et al., 2009; Holmes et al., 2014; Meconi et al., 2014). The absence of significant N2pc differences between stress and control groups in our study may be explained as follows: During early processing stages, threat stimuli were presented for relatively long durations, and acute stress may have suppressed early attentional orienting to threat. The amygdala is a key brain structure for processing fear-related information and expressing fear-related behaviors (LeDoux, 2003). Under acute stress, excessive cortisol secretion alters functional connectivity between the amygdala and frontoparietal network, weakening the amygdala' s specific responses to threat and changing neural mechanisms such that early processing of threat shifts from specific to sensitive responding, with non-discriminative selective responses to stimuli. Consequently, threat and neutral stimuli undergo similar cognitive processing and elicit similar ERP components (Grant, Judah, White, & Mills, 2015; Sanger et al., 2014; van Marle, Hermans, Qin, & Fernandez, 2009). This interpretation aligns with the significant positive correlation between the group difference in cortisol increment and the group difference in N2pc. Thus, acute stress influenced responses to threat stimuli without significantly enhancing attentional orienting to threat.

However, the stress group exhibited more negative SPCN than the control group, indicating that after threat identification, the stress group allocated more attentional resources to maintaining attention on threat stimuli. Weymar et al. found that under acute stress, threat stimuli (e.g., unpleasant images) elicited larger and longer-lasting LPP components, suggesting that acute stress strengthens attentional maintenance of threat (Weymar et al., 2012). Meconi et al. also found that, compared to non-anxious individuals, threat stimuli (e.g., untrustworthy faces) elicited more negative SPCN in anxious individuals, indicating sustained attentional processing of threat in anxiety (Meconi et al., 2014). This is consistent with our finding that acute stress-induced HPA axis activation impairs attentional disengagement from threat. Taylor et al. found that attentional control moderates the relationship between anxiety and attentional disengagement from threat, with weaker attentional control associated with slower disengagement (Taylor et al., 2016). Gindt et al. reported similar findings, showing that individuals with post-traumatic stress disorder have greater difficulty disengaging from threat than those with anxiety disorders (Gindt, Nachon, Chanquoy, Faure, & Garcia, 2017). The frontoparietal network is primarily responsible for attentional resource allocation (Fecteau & Munoz, 2006; Gottlieb, 2007). The neural mechanism by which acute stress impairs attentional disengagement from threat may involve acute stress-induced dysfunction of brain regions in the frontoparietal network associated with attentional disengagement, leading to reduced functional connectivity between the dorsolateral prefrontal cortex and other brain regions. This impairs effective attentional control, causing reductions or elimination of top-down attentional control resource reallocation (Luo et al., 2018; Qin et al., 2009; Sanger et al., 2014; Weerda, Muehlhan, Wolf, & Thiel, 2010; Wu et al., 2014). This interpretation is consistent with the significant positive correlation between the group difference in cortisol increment

and the group difference in SPCN. Therefore, acute stress impairs attentional disengagement from threat, producing more negative SPCN during later stages of threat processing.

5 Conclusion

Acute stress does not significantly affect attentional orienting to threat stimuli but impairs attentional disengagement from threat. This effect may be due to acute stress-induced dysfunction of the frontoparietal network associated with attentional disengagement.

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Note: Figure translations are in progress. See original paper for figures.