Acute Stress Impairs Error Monitoring and Post-Error Adjustment

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Abstract

In daily life, individuals constantly monitor the outcomes of their actions and make timely adjustments to adapt to environmental changes. However, whether individuals can effectively monitor their behavior and make adaptive adjustments under stress remains unknown. This study recruited 52 male college student participants, randomly assigned them to a stress group and a control group, used the Trier Social Stress Test (TSST) to induce stress responses, and combined it with the error awareness task (EAT) to explore error monitoring and post-error adjustment processes under acute stress. The stress indicator results showed that individuals in the stress group had significantly higher cortisol levels, heart rate, self-reported stress perception, and negative emotions than the control group, indicating successful induction of acute stress. Behavioral results showed that the error awareness accuracy rate in the stress group was significantly lower than that in the control group, and the error awareness reaction time was significantly shorter than that in the control group; furthermore, the accuracy rate on trials following error awareness in the stress group was significantly lower than that on trials following unaware errors, and the accuracy rate on trials following error awareness in the stress group was lower than that in the control group. The results indicate that acute stress reduces individuals' monitoring level of error responses; even when error responses are identified, individuals' behavioral monitoring and regulation are poorer. This study demonstrates that acute stress impairs the behavioral monitoring system, leading to decreased behavioral adaptability in individuals.

Full Text

Acute Stress Impairs Error Monitoring and Post-Error Adjustment

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Abstract

In daily life, individuals constantly monitor their behavioral outcomes and make timely adjustments to adapt to environmental changes. However, whether individuals can effectively monitor their behavior and make adaptive adjustments under stress remains unknown. This study recruited 52 male college students who were randomly assigned to either a stress group or a control group. We used the Trier Social Stress Test (TSST) to induce acute stress responses and combined it with an error awareness task (EAT) to explore error monitoring and post-error adjustment processes under acute stress. Stress index results showed that the stress group exhibited significantly higher cortisol levels, heart rate, self-reported stress perception, and negative affect compared to the control group, indicating successful acute stress induction. Behavioral results revealed that the stress group's error awareness accuracy was significantly lower than that of the control group, while their error awareness reaction time was significantly shorter. Furthermore, individuals in the stress group showed significantly lower accuracy on trials following aware errors compared to trials following unaware errors, and their accuracy after aware errors was lower than that of the control group. These results demonstrate that acute stress reduces individuals' monitoring of error responses, and even when errors are detected, behavioral monitoring and regulation become impaired. This study indicates that acute stress damages the behavioral monitoring system, leading to impaired behavioral adaptation.

Keywords: acute stress; error awareness; cortisol; post-error adjustment

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People inevitably make mistakes in daily life, and the ability to quickly and effectively monitor errors and regulate subsequent behavior to adapt to the environment is crucial. Error processing comprises two subprocesses: error monitoring and post-error response regulation (Gehring, Goss, Coles, Meyer, & Donchin, 2011). Previous research has shown that individuals make adaptive adjustments following errors, commonly manifested as post-error slowing (PES) and post-error improvement in accuracy (PIA) (Laming, 1979). Conflict mon-

itoring theory proposes that monitoring of error responses triggers enhanced cognitive control to prevent further mistakes (Botvinick, Braver, Barch, Carter, & Cohen, 2001). Neuroimaging studies have found that error monitoring and post-error regulation are primarily associated with the anterior cingulate cortex (ACC) and dorsolateral prefrontal cortex (dlPFC), where the ACC detects behavioral outcomes and the dlPFC is involved in task rule representation and behavioral control (Yeung, Botvinick, & Cohen, 2004). When individuals detect a discrepancy between actual and correct responses, the ACC signals the dlPFC, which implements cognitive control to regulate subsequent responses.

When facing stress, individuals experience mental tension and anxiety accompanied by rapid breathing, increased heart rate, and sweating—physiological and psychological responses collectively known as stress (Dickerson, Gruenewald, & Kemeny, 2004). Under stress, intense negative emotional experiences activate hypothalamic and brainstem stress pathways, triggering massive secretion of (nor)epinephrine and dopamine. These excess catecholamines enhance amygdala function while impairing prefrontal control processes (Vijayraghavan, Wang, Birnbaum, Williams, & Arnsten, 2007). Importantly, research indicates that core brain regions involved in error processing are negatively modulated by stress responses. For instance, the ACC shows deactivation patterns in individuals experiencing high stress (Pruessner et al., 2008; Dedovic et al., 2009), and the dlPFC exhibits reduced activation following stress (Arnsten, 2015). Additionally, studies have shown that acute stress impairs executive functions such as working memory (Bogdanov & Schwabe, 2016) and cognitive flexibility (Plessow, Fischer, Kirschbaum, & Goschke, 2011). Based on these findings, we can infer that stress may impair error processing.

Notably, existing research has primarily focused on error monitoring processes. Studies have found that individuals with post-traumatic stress disorder (PTSD) exhibit poorer error monitoring compared to healthy controls (Clemans, Elbaz, Hollifield, & Sokhadze, 2012), and studies using acute stress paradigms have also shown that stress impairs error monitoring system function (Cavanagh & Allen, 2008; Whitton et al., 2017). Only a few studies have reported post-error behavioral outcomes, but they failed to effectively reveal error processing under stress. For example, Cavanagh and Allen (2008) used a high-difficulty mathematical stress task to compare individual differences in post-error processing under stress but did not reveal typical stress effects on post-error processing. Furthermore, evidence suggests that post-error regulation only occurs when individuals become aware of their errors (Di Gregorio, Steinhauser, & Maier, 2016; Endrass, Reuter, & Kathmann, 2007), meaning that effective error monitoring is a prerequisite for post-error behavioral regulation. To date, the extent to which individuals can monitor their own error responses under stress and how error monitoring regulates post-error adjustment processes remain unclear.

To elucidate error processing under stress, this study employed the Trier Social Stress Test (TSST) and the error awareness task (EAT) (Hester et al., 2012; Hester, Foxe, Molholm, Shpaner, & Garavan, 2005) to investigate error monitor-

ing and post-error behavioral regulation under acute stress. The EAT, adapted from a go/no-go task, requires participants not only to perform the go/no-go task but also to explicitly mark their error responses with a key press. Trials in which participants correctly marked their errors after committing them were classified as "aware errors," while trials in which participants failed to mark their errors were classified as "unaware errors." The key-press marking of errors was used to reflect error monitoring levels under stress and non-stress conditions, while accuracy and reaction time on trials following error responses were used to reflect post-error regulation processing levels. During the experiment, we collected participants' heart rate, salivary cortisol, positive and negative affect, and self-reported stress perception to assess their physiological and psychological states and confirm successful stress induction. We compared differences between the stress and control groups in error awareness accuracy and post-error behavioral regulation to examine the effects of acute stress on error monitoring and post-error adjustment.

Methods

Participants

To exclude the influence of hormonal fluctuations from the female menstrual cycle on cortisol, this study recruited only male participants (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Laredo et al., 2015). Prior to recruitment, participants completed the Beck Depression Inventory (BDI) and Life Event Scale (LES) to exclude individuals affected by negative life events and depressive states. Fifty-six male participants were initially recruited, but four were excluded due to missing saliva samples and/or fewer than six error trials. The remaining 52 participants (26 in the stress group and 26 in the control group) ranged in age from 18 to 23 years, were all right-handed, non-smokers, had no color blindness/weakness, and were in good health. Participants had not taken medication, engaged in strenuous exercise, or consumed alcohol or stimulant/functional beverages within 24 hours before the experiment, and had not eaten within 2 hours prior. None had previously participated in similar stress tests. All participants provided informed consent before the experiment and received compensation afterward.

Measures

Beck Depression Inventory This 13-item scale uses a 4-point scoring system from 0 to 3. Higher total scores indicate stronger depressive symptoms, with scores above 8 indicating moderate or greater depression. This study excluded individuals with total scores exceeding 8 (Beck, 1967).

Stress Perception Self-Report Participants rated their perceived stress and tension levels on a 10-point scale, where 1 represented "very relaxed" and 10 represented "very tense."

State-Trait Anxiety Inventory (STAI) This scale uses a 4-point Likert format and includes two subscales: state anxiety and trait anxiety. The state anxiety subscale requires individuals to assess their anxiety level based on their "right now"state, while the trait anxiety subscale assesses their general or typical anxiety level (Spielberger, 1989).

Positive and Negative Affect Schedule (PANAS) This scale includes 20 adjectives reflecting individuals' mood states, with 10 items measuring positive affect and 10 measuring negative affect. Participants rated their mood states on a 5-point scale (Watson, Clark, & Tellegen, 1988).

Experimental Procedure

Human cortisol levels remain relatively stable between 12:00 and 18:30, so this study was conducted only during this time window. The experiment took place in a soundproof laboratory and lasted 90 minutes. The specific procedure is illustrated in Figure 1 [Figure 1: see original paper]. All participants were randomly assigned to either the stress or control group. After arriving at the laboratory, participants rested for 10 minutes, then wore heart rate monitoring equipment and completed the State-Trait Anxiety Inventory. The first saliva cortisol sample and baseline heart rate were collected, followed by completion of the stress perception self-report and PANAS. Participants then practiced the task. After practice, the stress group underwent the TSST while the control group underwent a control version of the stress test (control-TSST). Following the stress task, the second saliva cortisol sample and heart rate were collected, and participants completed the stress perception self-report and PANAS. After a 10-minute rest period to allow cortisol levels to peak, the third saliva cortisol sample and heart rate were collected, followed by the stress perception self-report, PANAS, and the formal task. After the experiment, the fourth saliva cortisol sample and heart rate were collected, and participants completed the final stress perception self-report and PANAS.

Stress Induction Procedure

The TSST consisted of a 5-minute interview speech and a 5-minute mental arithmetic task. In the speech task, participants faced two serious "experts" for a job interview. Participants had 5 minutes to prepare their speech, during which they had to explain why they were qualified for the position. The entire interview was video-recorded and evaluated by the two experts. Immediately following the interview, the mental arithmetic task began. Participants had to sequentially subtract 17 from 2093, maintaining both speed and accuracy. If participants reported an incorrect result, they had to restart from 2093. The control group completed a control version of the TSST, in which they gave a speech about a favorite movie without being video-recorded or evaluated by experts. The mental arithmetic task involved sequentially adding 15 starting from 0, without strict speed or accuracy requirements.

Error Awareness Task

This study employed the error awareness task, with detailed procedures shown in Figure 2 [Figure 2: see original paper]. In this task, participants responded to a series of color words presented on screen. The color words consisted of six color characters and colors: red (255,0,0), green (0,255,0), blue (0,0,255), purple (110,50,160), white (255,255,255), and yellow (255,255,0). Participants followed three response rules: (1) When the font color and meaning of the color word were inconsistent, participants made a key press (go trials); (2) When the font color and meaning were consistent, participants withheld their response (color no-go trials); (3) When the meaning of the current color word matched the meaning of the previous color word, participants withheld their response (repetition no-go trials). These two competing no-go conditions were designed to elicit unaware error responses. To mark "error awareness,"participants pressed a second "awareness key"when they made an erroneous key press (responding on either type of no-go trial). Participants were instructed to ignore the meaning and color of the immediately following go trial and complete the awareness key press on that color word. Given that error marking could affect processing of subsequent trials, the experimental program ensured that at least four go trials followed each no-go trial.

Each participant completed two practice blocks of 20 trials to familiarize themselves with the rules before entering the formal experiment. The EAT consisted of 6 blocks, each containing 210 trials (180 go trials and 30 no-go trials). The two types of no-go trials were equally distributed across the 6 blocks. Within each block, the three trial types were presented in pseudo-random order. Color words were presented for 800 ms and disappeared after a response, with an inter-trial interval of 700 ms. Half of the participants pressed "A" for the go/no-go task and "L" for error marking, while the other half used the opposite mapping.

Data Analysis

Stress Measurement In this study, salivary cortisol, heart rate, stress perception self-report, and positive/negative affect served as indices of stress levels. Saliva samples were collected using specialized saliva collection devices (salivette, SARSTEDT). After centrifugation, cortisol concentrations were measured using enzyme-linked immunosorbent assay (ELISA). Heart rate was recorded using Biopac' s MP150 multi-channel physiological signal recorder at a sampling frequency of 500 Hz, with heart rate calculations performed in AcqKnowledge 4.2 software. We conducted a time point (-40 min, 0 min, 10 min, 40 min) × group (stress, control) repeated measures ANOVA on these four indices.

Behavioral Data Analysis **Go/No-Go Task**: Independent samples t-tests were performed on go trial accuracy between the stress and control groups. A no-go type (repetition no-go, color no-go) × group (stress, control) repeated measures ANOVA was conducted on no-go trial accuracy. Additionally, independent samples t-tests were performed on go trial reaction times between

groups.

Error Awareness Task: Error awareness accuracy was calculated for each participant by dividing the number of aware error trials by the total number of error trials, with independent samples t-tests comparing groups. Independent samples t-tests were also performed on error awareness reaction times. Subsequently, no-go type (repetition no-go, color no-go) × group repeated measures ANOVAs were conducted on both error awareness accuracy and reaction times.

Error and Post-Error Responses: A trial type (aware error trials, unaware error trials) × group repeated measures ANOVA was performed on error trial reaction times. A no-go type (color no-go, repetition no-go) × group repeated measures ANOVA was conducted on error reaction times. Post-error adjustment in accuracy was calculated as the difference between post-error and post-correct trials: ACC_post-error adjustment = ACC_post-error trial -ACC_post-correct trial. Since post-error behavioral measures can be confounded by global performance shifts during the experiment (Dutilh et al., 2012), post-error reaction time adjustment was calculated by subtracting pre-error trial reaction times from post-error trial reaction times: RT_post-error adjustment = RT_post-error trial -RT_pre-error trial. We then conducted trial type (post-aware error trials, post-unaware error trials) × group repeated measures ANOVAs on accuracy and reaction time adjustments for both the first and second trials after errors. When participants were aware of an error and executed the error awareness key press, the first post-error trial was the trial immediately following the error awareness response; if participants were unaware of an error and did not execute the error awareness key press, the first post-error trial was the trial immediately following the error trial. The second post-error trial was the trial following the first post-error trial.

Statistical Analysis The significance level was set at 0.05 (two-tailed) with Bonferroni correction for multiple comparisons. For repeated measures ANOVAs with asymmetric samples, Greenhouse-Geisser correction was applied. When interactions were significant, post-hoc comparisons were performed using least significant difference (LSD) tests. With 52 participants, the study had 0.94 power (1 -) to detect a medium effect size (^{2} = 0.25) in two-factor repeated measures ANOVAs (trial type × group) on reaction time and accuracy at the 0.05 significance level.

Results

Stress Manipulation Check

Salivary Cortisol Concentration Cortisol changes in the stress and control groups are shown in Figure 3 Figure 3: see original paper. Repeated measures ANOVA revealed no significant main effects of group or time point (ps > 0.361), but a significant interaction between time point and group, F(3, 150) = 3.59, p = 0.022, ^{2} = 0.07, [0.01, 0.13]. Post-hoc analysis showed that cortisol concen-

tration in the stress group was significantly higher than in the control group at the 10-min time point ($p = 0.030$).

Heart Rate Heart rate changes are shown in Figure 3(2). Results showed a significant main effect of time point, $F(3, 150) = 54.62$, $p < 0.001$, $^2 = 0.52$, [0.42, 0.58]; a significant main effect of group, $F(1, 50) = 5.51$, $p = 0.023$, $^2 = 0.10$, [0.01, 0.24]; and a significant interaction between time point and group, $F(3, 150) = 21.18$, $p < 0.001$, $^2 = 0.52$. Post-hoc comparisons revealed significant group differences at 0 min, $F(1, 50) = 24.57$, $p < 0.001$, $^2 = 0.33$, [0.16, 0.47], and marginally significant differences at 40 min, $F(1, 50) = 3.91$, $p = 0.054$, $^2 = 0.07$, [0.00, 0.20].

Stress Perception Self-Report Stress perception self-report results are shown in Figure 3(3). Statistical analysis revealed a significant main effect of time point, $F(2.59, 129.64) = 26.80$, $p < 0.001$, $^2 = 0.35$, [0.24, 0.43]; a significant main effect of group, $F(1, 50) = 5.00$, $p = 0.030$, $^2 = 0.10$, [0.01, 0.23]; and a significant interaction between time point and group, $F(2.59, 129.64) = 11.58$, $p < 0.001$, $^2 = 0.19$, [0.09, 0.27]. Post-hoc comparisons showed that only the stress group exhibited significant differences across time points, $F(3, 48) = 32.38$, $p < 0.001$, $^2 = 0.67$, [0.51, 0.74]. Additionally, the stress group' s stress perception scores were significantly higher than the control group' s at the 0-min time point, $F(1, 50) = 34.06$, $p < 0.001$, $^2 = 0.41$, [0.23, 0.53].

Positive and Negative Affect Negative affect trends are shown in Figure 3(4). Repeated measures ANOVA on negative affect revealed a significant main effect of time point, $F(3, 150) = 14.62$, $p < 0.001$, $^2 = 0.23$, [0.12, 0.31]; a significant main effect of group, $F(1, 50) = 4.76$, $p = 0.034$, $^2 = 0.09$, [0.00, 0.22]; and a significant interaction between time point and group, $F(3, 150) = 11.58$, $p < 0.001$, $^2 = 0.16$, [0.09, 0.27]. Post-hoc analysis showed that only the stress group exhibited a significant main effect of time point, $F(3, 48) = 17.47$, $p < 0.001$, $^2 = 0.52$, [0.32, 0.61]. The stress group' s negative affect scores were significantly higher than the control group' s at 0 min, $F(1, 50) = 14.61$, $p < 0.001$, $^2 = 0.23$, [0.07, 0.37], and 10 min, $F(1, 50) = 6.48$, $p = 0.014$, $^2 = 0.12$, [0.01, 0.26].

Analysis of positive affect showed a significant main effect of time point, $F(3, 150) = 38.32$, $p < 0.001$, $^2 = 0.43$, [0.33, 0.51], with positive affect scores gradually decreasing across time points (M ± SD): $28.98 \pm 5.95 > 25.37 \pm 5.99 > 22.85 \pm 5.84 > 22.10 \pm 6.71$. Neither the main effect of group nor the interaction between time point and group was significant (ps > 0.65).

State-Trait Anxiety and Depression Levels Independent samples t-tests showed no significant differences between groups in state anxiety ($p = 0.85$; stress group: 36.31 ± 9.69, control group: 36.81 ± 8.57) or trait anxiety ($p =$

0.09; stress group: 39.77 ± 7.50, control group: 44.08 ± 10.22). Depression levels also did not differ significantly between groups (p = 0.97; stress group: 3.07 ± 2.46, control group: 3.40 ± 3.60).

Behavioral Results

Go/No-Go Task Performance Go/no-go task performance is presented in Table 1 . There was no significant difference between groups in go trial accuracy (p = 0.396). Repeated measures ANOVA on no-go trial accuracy revealed a significant main effect of trial type, F(1, 50) = 72.31, p < 0.001, ^{2} = 0.59, [0.43, 0.69], with color no-go trials showing significantly lower accuracy than repetition no-go trials. The main effect of group was significant, F(1, 50) = 8.91, p = 0.004, ^{2} = 0.15, [0.03, 0.30], with the control group showing significantly higher accuracy than the stress group. The interaction between trial type and group was not significant (p = 0.35). The stress group showed marginally faster go trial reaction times than the control group, t(50) = 1.90, p = 0.064, d = 0.54, [-2.30, 80.38].

Error Awareness Error awareness task performance is shown in Figure 4 [Figure 4: see original paper]. Results showed that the stress group' s error awareness accuracy was significantly lower than the control group' s, t(50) = 4.63, p < 0.001, d = 1.28, [0.15, 0.38], while their error awareness reaction time was significantly shorter, t(50) = 2.33, p = 0.024, d = 0.66, [0.09, 1.20]. Further repeated measures ANOVA on error awareness accuracy across different no-go trials revealed a significant main effect of no-go type, F(1, 50) = 14.45, p < 0.001, ^{2} = 0.22, [0.07, 0.37], with repetition no-go trials showing significantly lower error awareness accuracy than color no-go trials. The main effect of group was significant, F(1, 50) = 27.48, p < 0.001, ^{2} = 0.36, [0.18, 0.49], with the control group showing significantly higher accuracy than the stress group. The interaction between no-go type and group was not significant (p = 0.895). Analysis of reaction times showed a marginally significant main effect of group, F(1, 50) = 3.23, p = 0.079, ^{2} = 0.06, [0.00, 0.19]; neither the main effect of trial type nor the interaction was significant (ps > 0.373). The effect of trial type was consistent with previous research, indicating differences in trial difficulty (Hester et al., 2012), but these difficulty differences did not affect the between-group stress effects.

To explore whether individuals in the stress group were influenced by speed-accuracy trade-offs on the error awareness task, we compared the probability of becoming aware after making an error (P_awareness), the probability of remaining unaware after making an error (P_unawareness), the probability of false awareness after not making an error (P_false awareness), and the probability of correct judgment after not making an error (P_correct judgment). Independent samples t-tests revealed that the stress group' s P_awareness was significantly lower than the control group' s, t(50) = 4.63, p < 0.001, d = 1.28, [0.15, 0.38], while their P_unawareness was significantly higher, t(50) = -4.16, p

< 0.001, d = -1.16, [-0.36, -0.12]. No significant group differences were found for P_false awareness or P_correct judgment (ps > 0.861). These results indicate that stressed individuals did not execute more error awareness responses for erroneous no-go reactions, nor did they show better judgment for correct no-go responses compared to the control group. In other words, stressed individuals did not exhibit a processing pattern that sacrificed accuracy for impulsive and rapid error awareness responses.

Error and Post-Error Responses Repeated measures ANOVA on error trial reaction times showed a marginally significant main effect of group, $F(1, 50) = 3.74$, $p = 0.056$, $^2 = 0.07$, [0.00, 0.20]. Neither the main effect of trial type nor the interaction between trial type and group was significant (ps > 0.675). Analysis of error reaction times across the two no-go trial types revealed a significant main effect of no-go type, $F(1, 50) = 4.43$, $p = 0.040$, $^2 = 0.08$, [0.00, 0.21], with repetition no-go trials showing longer reaction times (490.02 ± 13.69 ms) than color no-go trials (475.57 ± 10.73 ms). The main effect of group was marginally significant, $F(1, 50) = 3.81$, $p = 0.056$, $^2 = 0.07$, [0.00, 0.20], and the interaction between trial type and group was not significant (ps > 0.613). See Table 1 for details.

Post-error adjustment results are presented in Figure 5 [Figure 5: see original paper]. For the first trial after errors, analysis of post-error accuracy adjustment revealed a significant main effect of trial type, $F(1, 50) = 10.43$, $p = 0.002$, $^2 = 0.17$, [0.04, 0.32], with accuracy after aware errors significantly lower than after unaware errors. The main effect of group was not significant ($p = 0.119$), but the interaction between trial type and group was significant, $F(1, 50) = 5.93$, $p = 0.019$, $^2 = 0.11$, [0.01, 0.25]. Post-hoc analysis showed that only the stress group exhibited significant differences between trial types, $F(1, 50) = 16.04$, $p < 0.001$, $^2 = 0.24$, [0.09, 0.90]. Additionally, the stress and control groups differed significantly in accuracy after aware errors, $F(1, 50) = 4.15$, $p = 0.047$, $^2 = 0.08$, [0.00, 0.21]. Analysis of post-error reaction time adjustment showed a significant main effect of trial type, $F(1, 50) = 52.67$, $p < 0.001$, $^2 = 0.51$, [0.34, 0.62], with reaction times after aware errors significantly shorter than after unaware errors. Neither the main effect of group nor the interaction between group and trial type was significant (ps > 0.094).

For the second trial after errors, analysis of post-error accuracy adjustment showed no significant effects (ps > 0.307). For post-error reaction time adjustment, the main effect of trial type was significant, $F(1, 50) = 8.02$, $p = 0.007$, $^2 = 0.14$, [0.02, 0.28], with reaction times after aware errors significantly shorter than after unaware errors. Neither the main effect of group nor the interaction between group and trial type was significant (ps > 0.130).

Correlation Analysis Correlation analyses between behavioral results and stress indices at the 0-min and 10-min time points revealed different relationships between negative affect at 0 min and error awareness accuracy in the stress and

control groups. In the stress group, negative affect at 0 min was significantly negatively correlated with error awareness accuracy, r = -0.42, p = 0.034, while this correlation was not significant in the control group, r = -0.09, p = 0.67. See Table 2 for details. We further used hierarchical regression to analyze the predictive power of stress indices on error awareness accuracy. Results showed that in the stress group, only negative affect at 0 min predicted error awareness accuracy, explaining 13.90% of the variance, = -0.42, F(1, 24) = 5.04, p = 0.034.

Discussion

This study used the Trier Social Stress Test (TSST) and error awareness task (EAT) to investigate the effects of acute stress on error monitoring and post-error adjustment. Results showed that compared to the control group, the stress group exhibited significant increases in salivary cortisol, heart rate, stress perception self-report, and negative affect during the stress procedure, indicating successful manipulation of participants' stress states. Behaviorally, the stress group showed lower no-go trial accuracy than the control group. The stress group' s error awareness accuracy was significantly lower than the control group' s, while their reaction time was significantly faster. Negative affect under acute stress negatively predicted error awareness accuracy in the stress group. For post-error trials, when individuals were aware of their errors, the stress group showed lower accuracy on subsequent trials compared to trials following unaware errors, and the stress group demonstrated poorer post-error performance than the control group.

The finding that the stress group exhibited lower accuracy on the response inhibition task than the control group indicates that acute stress impaired inhibitory control processing. This is consistent with research showing that acute stress impairs core executive functions including cognitive flexibility, working memory, and cognitive inhibition (Sänger, Bechtold, Schoofs, Blaszkewicz, & Wascher, 2014; Plessow et al., 2011; Qin, Hermans, van Marle, & Fernández, 2009). Furthermore, this study found that stressed individuals missed more error responses than the control group, and that negative affect under acute stress was significantly negatively correlated with error awareness accuracy. Negative affect serves as an immediate measure of stress state, revealing the intensity of individuals' stress experience. The stronger the negative affect during the stress test, the more error responses were missed, suggesting that acute stress leads to decreased error monitoring. Notably, errors are inherently aversive signals (Hajcak & Foti, 2008). According to the attentional competition theory, cognitive processing resources are limited, and emotional processing competes with executive control for these resources. When emotional states are intense, they occupy more cognitive resources and interfere with executive control processing (Pessoa, 2009). Multiple studies have also shown that negative emotional states lead to task disengagement (Luu, Collins, & Tucker, 2000; Weinberg, Riesel, & Hajcak, 2012), where attention shifts away from behavioral monitoring to other processes

such as one' s own emotional experience. Therefore, we speculate that the negative emotion induced by acute stress occupied substantial cognitive resources, leaving insufficient resources for response monitoring and consequently causing decreased error monitoring. In contrast, heart rate, stress perception self-report, and salivary cortisol were not significantly correlated with error awareness accuracy. According to previous research, these four indices are interrelated. Heart rate and cortisol are indicators of sympathetic-adrenal-medullary (SAM) system and hypothalamic-pituitary-adrenal (HPA) axis activity, respectively. Evidence suggests that catecholamines secreted by the SAM system enhance amygdala activation, and cortisol also potentiates catecholamine modulation (McReynolds et al., 2010; Roozendaal, Okuda, Van der Zee, & McGaugh, 2006), meaning that SAM and HPA axis activation also influence negative affective states under acute stress. We do not rule out the possibility that factors such as the specific error awareness paradigm and precise levels of cortisol and catecholamines may have influenced the correlation analysis, resulting in only negative affect showing a significant correlation with error awareness. The relationships between different physiological and psychological manifestations of acute stress and their effects on error monitoring require further investigation. Additionally, stressed individuals made faster responses on the error awareness task. Combined with the finding that stressed individuals showed faster go reaction times, we believe this relates to enhanced motor excitability under stress that facilitates response capability (Falconer et al., 2008; Wu et al., 2010). In other words, when detecting their own errors, the heightened motor readiness state in stressed individuals accelerated their error awareness key-press speed.

This study found that both stress and control groups showed response acceleration after detecting errors. Unlike the typical post-error slowing, post-error speeding often occurs in go/no-go tasks because the go response is a dominant response, and error awareness leads to automation of this dominant response (Hester et al., 2005; Hester, Simoes-Franklin, & Garavan, 2007). Importantly, only the stress group showed a significant decrease in accuracy after detecting errors; while the control group showed similar differences between conditions, they did not reach statistical significance. Interference accounts of post-error processing propose that attention to and evaluation of error signals cause temporary interference on subsequent trials because attention remains focused on the error trial, occupying resources needed for processing the next trial (Jentzsch & Dudschig, 2009; Ullsperger & Danielmeier, 2016). In the classic EAT paradigm, the interval between error trials, error awareness responses, and the first post-error trial is 700 ms, during which attention remains largely focused on the error response, limiting attention and regulation of subsequent trials. Indeed, most studies finding post-error accuracy improvements have used tasks with inter-trial intervals exceeding 900 ms (Danielmeier & Ullsperger, 2011; Steinhauser, Ernst, & Ibald, 2017). Furthermore, this study found that response speeding persisted on the second trial after errors, but accuracy returned to pre-error levels, suggesting that error monitoring-induced post-error adjustments can only improve behavioral accuracy within a limited time window. In the control group, the

difference in accuracy between trials following aware versus unaware errors did not reach significance, possibly due to flexible coping strategies under optimal cognitive processing conditions or ceiling effects; future research could address this by increasing task difficulty.

More importantly, this study found that individuals under acute stress could not effectively regulate their behavior after becoming aware of errors, and their performance was worse than the control group. We infer that post-error processing in stressed individuals experienced greater interference, likely caused by two factors: (1) Enhanced negative experience leads individuals to focus more on and evaluate the current error signal (Wu et al., 2014). Recently, Buzzell et al. (2017) showed that when the interval between error responses and subsequent trials is short, the amplitude of the error-related positivity (Pe)—which reflects error awareness and subjective evaluation—is negatively correlated with the P1 amplitude on the next trial, indicating that error evaluation and attention interfere with early perceptual processing of subsequent trials. Therefore, we can infer that under acute stress, error awareness and evaluation occupy more attentional resources, causing greater interference with processing of the next trial. In contrast, on trials following unaware errors, the error signal does not reach awareness and no subjective evaluation occurs, so acute stress does not affect processing of these subsequent trials. (2) Acute stress-induced dlPFC impairment leads to decreased executive control. Previous research shows that the dlPFC plays a crucial role in top-down attentional control (Banich, 2009; Fumi & Christos, 2012), and decreased cognitive control prevents individuals from quickly shifting attention back to task processing, making it difficult to timely regulate and improve current task performance. Compared to studies that did not find post-error behavioral regulation impairments (Whitton et al., 2017), the impairment found in this study may be due to characteristics of early (or intermediate) post-error processing stages. In this study, attention remained largely focused on the error response when processing post-error trials, preventing effective behavioral regulation to improve performance. In Whitton et al. (2017), the single-trial duration was 2050-2250 ms, providing individuals with longer regulation time after errors. This not only reduced the impact of error evaluation on post-error regulation but also allowed strategic compensation for dlPFC functional impairment, which may explain why that study did not find post-error behavioral regulation deficits.

Our results demonstrate that acute stress not only impairs error monitoring but also leads to poorer post-error adjustment, with negative affect playing an important role in mediating the effects of acute stress on error processing. On one hand, persistent negative emotional processing under acute stress occupies cognitive resources, leaving insufficient resources for response monitoring. On the other hand, the sustained negative state also causes excessive attention to detected error responses, leading to decreased post-error regulation. In fact, errors always elicit negative emotions, and Inzlicht et al. (2015) have proposed that negative emotions play an important role in driving cognitive regulation of error signals, as emotional responses facilitate adaptive preparation for aver-

sive signals. The biphasic-reciprocal model states that under non-stress conditions, the frontoparietal control network centered on the dlPFC efficiently regulates cognitive processing in other brain regions. However, under stress, stress-induced hormones such as catecholamines and cortisol enhance amygdala function, which increases vigilance for negative signals but at the cost of compromising frontoparietal control network function (Hermans, Henckens, Joëls, & Fernández, 2014). We propose that under acute stress, processing of error responses is susceptible to distraction and interference from one' s own negative emotions. This reduces cognitive resources available for behavioral monitoring, decreasing the efficiency of response monitoring, while also preventing appropriate evaluation of one' s own errors and subsequent generation and execution of adaptive regulatory strategies. In summary, the negative emotional processing bias induced by acute stress is detrimental not only to error monitoring but also to implementing adaptive behavioral adjustments.

This study demonstrates that acute stress impairs both error monitoring and post-error behavioral adjustment processes. Consistent with the biphasic-reciprocal model, enhanced amygdala activation under acute stress leads to dysfunctional frontoparietal networks that support cognitive control. This study shows that brain functional changes under acute stress damage the error monitoring system, resulting in decreased error monitoring efficiency and insufficient adaptive regulation.

Arnsten, A. F. (2015). Stress weakens prefrontal networks: molecular insults to higher cognition. *Nature neuroscience*, 18(10), 1376–1385.

Banich, M. T. (2009). Executive Function: The Search for an Integrated Account. *Current Directions in Psychological Science*, 18(2), 89–94.

Beck, A. T. (1967). *Depression: Clinical, experimental and theoretical aspects*. New York: Hoeber.

Bogdanov, M., & Schwabe, L. (2016). Transcranial stimulation of the dorsolateral prefrontal cortex prevents stress-induced working memory deficits. *Journal of Neuroscience*, 36(4), 1429–1437.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, 108(3), 624–652.

Buzzell, G. A., Beatty, P. J., Paquette, N. A., Roberts, D. M., & McDonald, C. G. (2017). Error-Induced Blindness: Error Detection Leads to Impaired Sensory Processing and Lower Accuracy at Short Response-Stimulus Intervals. *The Journal of Neuroscience*, 37(11), 2895–2903.

Cavanagh, J. F., & Allen, J. J. (2008). Multiple aspects of the stress response under social evaluative threat: an electrophysiological investigation. *Psychoneuroendocrinology*, 33(1), 41–53.

Clemans, Z. A., Elbaz, A. S., Hollifield, M., & Sokhadze, E. M. (2012). Single trial time-frequency domain analysis of error processing in post-traumatic stress disorder. *Neuroscience Letters*, 525(2), 105–110.

Danielmeier, C., & Ullsperger, M. (2011). Post-Error Adjustments. *Frontiers in Psychology*, 2, 233.

Dedovic, K., Rexroth, M., Wolff, E., Duchesne, A., Scherling, C., Beaudry, T.,···Pruessner, J. C. (2009). Neural correlates of processing stressful information: an event-related fMRI study. *Brain Research*, 1293, 49–60.

Di Gregorio, F., Steinhauser, M., & Maier, M. E. (2016). Error-related brain activity and error awareness in an error classification paradigm. *NeuroImage*, 139, 202–210.

Dickerson, S. S., Gruenewald, T. L., & Kemeny, M. E. (2004). When the social self is threatened: shame, physiology, and health. *Journal of Personality*, 72(6), 1191–1216.

Dutilh, G., Ravenzwaaij, D. V., Nieuwenhuis, S., Han, L. J. V. D., Forstmann, B. U., & Wagenmakers, E. J. (2012). How to measure post-error slowing: A confound and a simple solution. *Journal of Mathematical Psychology*, 56(3), 208–216.

Endrass, T., Reuter, B., & Kathmann, N. (2007). ERP correlates of conscious error recognition: aware and unaware errors in an antisaccade task. *European Journal of Neuroscience*, 26(6), 1714–1720.

Falconer, E., Bryant, R., Felmingham, K. L., Kemp, A. H., Gordon, E., & Peduto, A., et al. (2008). The neural networks of inhibitory control in posttraumatic stress disorder. *Journal of psychiatry & neuroscience: JPN*, 33(5), 413–422.

Fumi, K., & Christos, C. (2012). Early involvement of prefrontal cortex in visual bottom-up attention. *Nature Neuroscience*, 15(8), 1160–1166.

Gehring, W. J., Goss, B., Coles, M., Meyer, D. E., & Donchin, E. (2011). The Error-Related Negativity. *Perspectives on Psychological Science*, 13(2), 200–204.

Hajcak, G., & Foti, D. (2008). Errors Are Aversive: Defensive Motivation and the Error-Related Negativity. *Psychological Science*, 19(2), 103–108.

Hermans, E. J., Henckens, M. J., Joëls, M., & Fernández, G. (2014). Dynamic adaptation of large-scale brain networks in response to acute stressors. *Trends in neurosciences*, 37(6), 304–314.

Hester, R., Foxe, J. J., Molholm, S., Shpaner, M., & Garavan, H. (2005). Neural mechanisms involved in error processing: A comparison of errors made with and without awareness. *Neuroimage*, 27(3), 602–608.

Hester, R., Simoes-Franklin, C., & Garavan, H. (2007). Post-error behavior in active cocaine users: poor awareness of errors in the presence of intact performance adjustments. *Neuropsychopharmacology*, 32(9), 1974-1978.

Hester, R., Nandam, L. S., O' Connell, R. G., Wagner, J., Strudwick, M., Nathan, P. J.,···Nandam, L. S. (2012). Neurochemical Enhancement of Conscious Error Awareness. *Journal of Neuroscience*, 32(8), 2619-2627.

Inzlicht, M., Bartholow, B. D., & Hirsh, J. B. (2015). Emotional foundations of cognitive control. *Trends in cognitive sciences*, 19(3), 126-132.

Jentzsch, I., & Dudschig, C. (2009). Why do we slow down after an error? Mechanisms underlying the effects of posterror slowing. *The Quarterly Journal of Experimental Psychology*, 62(2), 209-218.

Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine*, 61(2), 154-162.

Laming, D. (1979). Choice reaction performance following an error. *Acta Psychologica*, 43(3), 199-224.

Laredo, S. A., Steinman, M. Q., Robles, C. F., Ferrer, E., Ragen, B. J.,···Trainor, B. C. (2015). Effects of defeat stress on behavioral flexibility in males and females: modulation by the mu-opioid receptor. *European Journal of Neuroscience*, 41(4), 434-441.

Luu, P., Collins, P., & Tucker, D. M. (2000). Mood, personality, and self-monitoring: negative affect and emotionality in relation to frontal lobe mechanisms of error monitoring. *Journal of Experimental Psychology: General*, 129(1), 43-60.

McReynolds, J. R., Donowho, K., Abdi, A., McGaugh, J. L., Roozendaal, B., & McIntyre, C. K. (2010). Memory-enhancing corticosterone treatment increases amygdala norepinephrine and Arc protein expression in hippocampal synaptic fractions. *Neurobiology of learning and memory*, 93(3), 312-321.

Pessoa, L. (2009). How do emotion and motivation direct executive control? *Trends in Cognitive Sciences*, 13(4), 160-166.

Plessow, F., Fischer, R., Kirschbaum, C., & Goschke, T. (2011). Inflexibly Focused under Stress: Acute Psychosocial Stress Increases Shielding of Action Goals at the Expense of Reduced Cognitive Flexibility with Increasing Time Lag to the Stressor. *Journal of Cognitive Neuroscience*, 23(11), 3218-3227.

Pruessner, J. C., Dedovic, K., Khalili-Mahani, N., Engert, V., Pruessner, M., Buss, C.,···Lupien, S. (2008). Deactivation of the limbic system during acute psychosocial stress: evidence from positron emission tomography

and functional magnetic resonance imaging studies. *Biological Psychiatry*, 63(2), 234–240.

Qin, S., Hermans, E. J., Van Marle, H. J. F., Luo, J., & Fernández, G. (2009). Acute Psychological Stress Reduces Working Memory-Related Activity in the Dorsolateral Prefrontal Cortex. *Biological Psychiatry*, 66(1), 25–32.

Roozendaal, B., Okuda, S., Van der Zee, E. A., & McGaugh, J. L. (2006). Glucocorticoid enhancement of memory requires arousal-induced noradrenergic activation in the basolateral amygdala. *Proceedings of the National Academy of Sciences*, 103(17), 6741–6746.

Sänger, J., Bechtold, L., Schoofs, D., Blaszkewicz, M., & Wascher, E. (2014). The influence of acute stress on attention mechanisms and its electrophysiological correlates. *Frontiers in Behavioral Neuroscience*, 8, 353.

Spielberger, C. D. (1989). *State-Trait Anxiety Inventory: A comprehensive bibliography*. Palo Alto, CA: Consulting Psychologists Press.

Steinhauser, M., Ernst, B., & Ibald, K. W. (2017). Isolating Component Processes of Posterror Slowing With the Psychological Refractory Period Paradigm. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 43(4), 653–659.

Ullsperger, M., & Danielmeier, C. (2016). Reducing Speed and Sight: How Adaptive Is Post-Error Slowing? *Neuron*, 89(3), 430–432.

Vijayraghavan, S., Wang, M., Birnbaum, S. G., Williams, G. V., & Arnsten, A. F. (2007). Inverted-U dopamine D1 receptor actions on prefrontal neurons engaged in working memory. *Nature Neuroscience*, 10(3), 376–384.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology*, 54(6), 1063–1070.

Weinberg, A., Riesel, A., & Hajcak, G. (2012). Integrating multiple perspectives on error-related brain activity: The ERN as a neural indicator of trait defensive reactivity. *Motivation & Emotion*, 36(1), 84–100.

Whitton, A. E., Veer, A. V. T., Kakani, P., Dillon, D. G., Ironside, M. L., & Haile, A., et al. (2017). Acute stress impairs frontocingulate activation during error monitoring in remitted depression. *Psychoneuroendocrinology*, 75, 164–172.

Wu, J., Ge, Y., Shi, Z., Duan, X., Wang, L., & Sun, X., et al. (2010). Response inhibition in adolescent earthquake survivors with and without post-traumatic stress disorder: A combined behavioral and ERP study. *Neuroscience Letters*, 486(3), 117–121.

Wu, J., Yuan, Y., Duan, H., Qin, S., Buchanan, T. W., & Zhang, K., et al. (2014). Long-term academic stress increases the late component of error processing: An ERP study. *Biological psychology*, 99, 77–82.

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: conflict monitoring and the error-related negativity. *Psychological Review*, 111(4), 931–959.

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