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Effect of acute psychological stress on response inhibition: An event-related potential study



Mingming Qi^{a,1}, Heming Gao^{c,1}, Guangyuan Liu^{a,b,*}

^a Faculty of Psychology, Southwest University, Chongqing, 400715, China

^b College of Electronic and Information Engineering, Southwest University, Chongqing, 400715, China

^c School of Psychology, Liaoning Normal University, Dalian, 116029, China

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ABSTRACT

This study aimed to investigate the effect of acute psychological stress on response inhibition and its electrophysiological correlates using a dual-task paradigm. Acute stress was induced by a primary task (mental arithmetic task), which consisted of a stress block and a control block. Response inhibition was measured using a secondary task (Go/NoGo task). In each trial, a Go/NoGo stimulus was presented immediately after the mental arithmetic task. The results revealed increased subjective stress and negative affect for the stress relative to control block, suggesting that the mental arithmetic task triggered a reliable stress response. ERPs locked to the Go/NoGo stimuli revealed that decreased P2 and increased N2 components were evoked for the stress block compared to the control block. These results demonstrated that acute psychological stress alters the response inhibition process by reducing the early selective attention process and enhancing the cognitive control process.

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1. Introduction

Stress is ubiquitous in our everyday lives. It can modulate behavioral and hormonal reactions [1,2], and fundamentally alter neural responses to incoming information [3,4]. Acute stress exerts a great impact on a variety of cognitive processes, ranging from attention and cognitive control to memory and social cognition [5,6].

Increasing evidence indicates that stress tends to facilitate cognitive function, particularly in well-rehearsed or simple tasks, or when the cognitive load is not excessive. However, it impairs tasks requiring complex, flexible reasoning [7,8]. Some studies that investigated the effect of stress on executive control processes argued that stress reallocates executive control resources from working memory and cognitive flexibility to selective attention, in order to focus processing on current stress-relevant information [9–11]. Stress modulates executive control and shifts cognition to a state of habitual action to facilitate adaptation to the current circumstances [12,13].

At the neural level, stress can influence the prefrontal cortexdependent cognition by increasing noradrenergic, dopaminergic, or glucocorticoid mediated signaling [7,14,15]. Some studies have suggested that stress impairs tasks that require prefrontal cortex (PFC) operations, whereas engrained habits that rely on basal ganglia circuits are spared or enhanced [7,16]. However, other studies have also found enhancing effects of stress on PFC functions. The activation of a fronto-parietal executive function network, including the dorsolateral prefrontal cortex, has been found to be enhanced under stress [17,18].

Executive control is engaged in situations requiring decisionmaking, conflict resolution, error correction, and response inhibition [19]. As one of the core components of executive function, response inhibition is often measured using the Go/NoGo task, in which participants respond to a designated stimulus (Go) and withhold responding to another stimulus (NoGo). Numerous fMRI studies have demonstrated that activation of the PFC and the anterior cingulate cortex is associated with response inhibition [20,21]. The right inferior PFC seems to be specialized for inhibiting inappropriate motor responses [22]. Therefore, response inhibition might be modulated by psychological stress which could influence the PFC function.

Few studies have addressed the link between acute psychological stress and response inhibition. Scholz et al. have suggested that acute psychological stress significantly impairs Go/NoGo performance, and executive functioning is reduced under acute stress [23]. Similarly, participants with post-traumatic stress disorder showed more inhibition-related errors [24] and longer P3 latency

^{*} Corresponding author at: Faculty of Psychology, Southwest University, Chongqing, 400715, China.

E-mail address: liugy@swu.edu.cn (G. Liu).

¹ Mingming Qi and Heming Gao contributed equally to this work.

relative to controls [25]. However, a recent meta-analysis of acute stress effect on core executive function found that stress significantly enhanced response inhibition [11]. Some studies have also found that acute stress enhanced response inhibition [15,26]. Stop signal reaction times were shorter for stressed participants relative to controls, suggesting that response inhibition was improved [15]. Similarly, some studies adopted threat of shock as a stressor and found decreased errors of commission on the NoGo trials for the stress relative to the safe condition [27,28]. Thus, the effect of acute psychological stress on response inhibition is still uncertain. Additionally, no studies have examined acute psychological stress effects on neural activity related to response inhibition [11].

In the present study, we aimed to investigate the effect of acute psychological stress on response inhibition using a dual-task paradigm. A state of psychological stress was induced using a mental arithmetic task [4], i.e., the primary task. Response inhibition was measured using a Go/NoGo task, i.e., the secondary task. In each trial, a Go/NoGo stimulus was presented immediately after the mental arithmetic task. A mental arithmetic task characterized by uncontrollability and social-evaluative threat has been used to evoke psychological stress in previous well-established studies [1,29–31]. This task has been shown to generate a reliable stress response with a significant increase in self-reported stress and sali-vary cortisol levels [4,29–31].

We investigated the ERPs evoked by the stimuli in the Go/NoGo task. Numerous electrophysiological studies have demonstrated that NoGo stimuli evoke more negative frontal N2 and positive frontal P3 components compared to Go stimuli, indicating more intensive conflict monitoring (N2) and response evaluation/decision (P3) processes in the NoGo trials [25,32–34]. We hypothesized that if response inhibition was enhanced under acute psychological stress, a larger frontal N2 component would be expected for the stress relative to control (non-stress) block. On the contrary, if response inhibition was impaired under stress, a reduced frontal N2 component would be found for the stress relative to control block.

2. Materials and methods

2.1. Participants

Twenty volunteer college students were recruited through online advertisements, and were pre-screened using the Beck Depression Inventory (BDI) [35]. Three participants were found to experience depressive symptoms (BDI scores \geq 14) and did not take part in the formal experiment. Seventeen participants (BDI scores <14) completed the formal experiment. Three participants were excluded from the analysis due to excessive artifacts (more than 50% of their trials were invalid). Therefore, data from fourteen participants were finally included in the analysis (6 males and 8 females; mean age = 21.8 years; *SD* = 1.6). All of the female participants were in the follicular phase (Kirschbaum et al., 1999) [36]. All participants were right-handed, and had normal or correctedto-normal vision. All participants signed an informed consent form and were paid for their participation. This study was approved by the Research Ethics Committee of the Southwest University of China and was performed in accordance with the ethical guidelines of the Declaration of Helsinki.

2.2. Design and materials

A dual-task was used in this study. A Go/NoGo task was followed by the mental arithmetic task [4] in each trial. A set of multiplication formulas containing 240 arithmetic expressions (e.g., 4.94×2.01) was adopted. Stimuli in the Go/NoGo task were letters ("O" or "X").

For the mental arithmetic task, participants were asked to estimate whether multiplying two two-decimal numbers would result in a product that was above 10 or not, within a time limit by pressing "f" or "j" key. In the stress block, participants were given 1500 ms to perform the mental arithmetic. The time limit was enforced for each trial, and the elapsed time was indicated by red dots that progressed from left to right on the computer screen. As soon as a response was submitted, the formula disappeared, and a blank screen was presented. Then, meaningful feedback was presented, which consisted of a comparison between the participant's individual reaction times (RTs) and average RTs, and the correctness of the response, with the RT comparison on top and the correctness on the bottom (Fig. 1A). In the control block, participants were given 6000 ms to perform the same mental arithmetic. To ensure consistency between the stress and control blocks regarding formatting of the stimuli, red dots were also presented below the multiplication formula in the control block but in a pseudo-random order. The formula disappeared once a response was submitted. Next, a black rectangle without meaningful feedback was presented (Fig. 1B). Participants were told that their movements and performances were being monitored during



Fig. 1. Experimental procedure and sample materials for the stress block (A) and the control block (B).

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