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Acute stress impairs inhibitory control based on individual differences in parasympathetic nervous system activity

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ABSTRACT

Identifying environmental influences on inhibitory control (IC) may help promote positive behavioral and social adjustment. Although chronic stress is known to predict lower IC, the immediate effects of acute stress are unknown. The parasympathetic nervous system (PNS) may be a mechanism of the stress-IC link, given its psychophysiological regulatory role and connections to prefrontal brain regions critical to IC. We used a focused assessment of IC (the stop-signal task) to test whether an acute social stressor (the Trier Social Stress Test) affected participants' pre- to post-IC performance (n = 58), compared to a control manipulation (n = 31). High frequency heart-rate variability was used as an index of PNS activity in response to the manipulation. Results indicated that stress impaired IC performance, blocking the practice effects observed in control participants. We also investigated the associations between PNS activity and IC; higher resting PNS activity predicted better pre-manipulation IC, and greater PNS stressor reactivity protected against the negative effects of stress on IC. Together, these results are the first to document the immediate effects of acute stress on IC and a phenotypic marker (PNS reactivity to stressors) of susceptibility to stress-induced IC impairment. This study suggests a new way to identify situations in which individuals are likely to exhibit IC vulnerability and related consequences such as impulsivity and risk taking behavior. Targeting PNS regulation may represent a novel target for IC-focused interventions.

1. Introduction

Inhibitory control (IC), the ability to stop a prepotent response, allows individuals to flexibly meet environmental demands instead of relying on impulsive response tendencies (Diamond, 2013). Identifying environmental influences on IC is important because impairment is implicated in negative outcomes, such as substance use (Iacono, Malone, & McGue, 2008) and psychopathology (Wright, Lipsyc, Dupuis, Thayapararajah, & Schachar, 2014). Chronic stress has been associated with IC impairment and disruption of underlying neurobiology (Mani, Mullainathan, Shafir, & Zhao, 2013; Mika et al., 2012). However, the immediate effects of *acute stress* are poorly understood, despite the plausible link between IC impairment and accumulation of stressful experiences. Delineating impacts of acute stress could offer insight into the chronic stress–IC link and contexts in which individuals are susceptible to IC lapses and impulsive behavior.

The autonomic nervous system (ANS) may be a key underlying mechanism in the acute stress-IC link given its regulatory role across emotional, cognitive, and physiological domains (Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). The parasympathetic nervous system (PNS), one ANS branch, is of primary importance because of its fast-acting regulation of heart rate via the vagus nerve, allowing flexible psychophysiological responses (Thayer et al., 2012). Notably, PNS function is critical for modulating arousal demands in reaction to acute stress and is also linked to cognitive function (Thayer, Hansen Saus-Rose, & Johnsen, 2009; Yim, Quas, Rush, Granger, & Skoluda, 2015). Specifically, the prefrontal brain regions (e.g., medial prefrontal cortex; anterior cingulate cortex) critical for IC performance also control limbic regions that regulate PNS activity. Accordingly, we suggest that shared neural systems could help explain both a resting PNS-IC link and an acute stress-IC link (Graziano & Derefinko, 2013; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Thayer et al., 2012; Verbruggen & Logan, 2008).

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Specifically, preliminary evidence suggests associations between resting PNS activity and performance on some (Hovland et al., 2012; Beaumont et al., 2012), but not all (Capuana, Dywan, Tays, & Segalowitz, 2012) IC-related measures. However, extant paradigms have confounded multiple cognitive processes (e.g., emotion processing, working memory) with IC and are not tied to a specific neural system. Given that brain regions underlying "pure" IC are well established and linked to PNS regulation, an investigation of the link between baseline PNS activity and a focused measure of IC, such as stop signal reaction time (SSRT), is needed. The SSRT, assessed during the stop signal paradigm, is a particularly rigorous measure for examining individual differences in IC because of the adaptive nature of the allowable response window, which ensures that all participants maintain approximately 50% accuracy. Specifically, by 'holding' participants at 50% accuracy, speed/accuracy trade-off strategies become less relevant. The SSRT has been advocated for as the most suitable laboratory paradigm for IC research (Seli, Cheyne, Smilek, 2012; Verbruggen & Logan, 2008).

Top-down regulation of the limbic system by prefrontal regions is commonly used to explain the IC-PNS link, but evidence also suggests a bidirectional relationship, with limbic regions contributing bottom-up arousal demands (Blair & Ursache, 2011; Park & Thayer, 2014). Consistent with such bottom-up arousal demands, exposure to acute stress may lead to subsequent impairment of prefrontal cortex function, and consequently lower IC performance.

Although there has been no research to date examining the immediate consequences of acute stress on IC, two studies have examined delayed (10–30 min) effects of acute stress, with the goal of understanding possible effects of cortisol, which peaks approximately twenty minutes post-stressor (Dickerson & Kemeny, 2004). These studies document conflicting results, with one reporting higher post-stressor versus post-control IC (assessed via SSRT; Schwabe, Hoffken, Tegenthoff, & Wolf, 2013) while another (using a Go/No-Go task) reported slower Go Trial reaction time, and no effects on IC accuracy (Scholz et al., 2009). Neither study examined how individual differences in cortisol linked to IC, but Schwabe et al. (2013) found that pharmacologically blocking the effects of cortisol nulled the effects of acute stress. However, both of these studies had substantial limitations including small sample sizes (11–18 per group) and no assessment of pre-stressor IC. Related research in the working memory domain demonstrated that acute stress impaired n-back task reaction time and accuracy performance, with effects of acute stress diminishing over time (Schoofs, Preuss, & Wolf, 2008). Taken together, such results suggest that the immediate aftermath of an acute stressor may negatively impact performance on demanding executive function tasks. However, research using larger sample sizes that incorporates pre/post assessments and investigations of putative underlying neurobiological mechanisms is needed to further characterize this phenomenon.

In the present study, we specifically examined the extent to which lower PNS regulation in response to acute stress might result in subsequent IC impairment. Prior research using social-evaluative stressor tasks (i.e. the Trier Social Stressor Task; TSST) documents PNS engagement during the task, suggesting arousal regulation consistent with the demands of presenting a well-regulated speech to socially-threatening judges (Yim et al., 2015). Given such findings, it could be hypothesized that participants who demonstrate more flexible PNS engagement to the TSST (i.e. higher PNS) would be better able to manage increased post-stressor arousal demands during an IC task. However, because there has been no research to date examining the *immediate* effects of an arousal manipulation via acute stress on IC, the extent to which PNS activity could account for a relationship between acute stress and IC impairment is unknown.

Here, we conducted the first assessment of the immediate effects of acute stress on IC with the goal of examining the effects of PNS activity as an explanatory mechanism underlying an acute-stress and IC link. Specifically, a repeated measures design was used to assess the extent to which a social-evaluative stressor (the TSST) affected a rigorous index of IC (SSRT; Aron, Robbins, & Poldrack, 2014). We hypothesized that acute stress (vs. control) would impair IC. We further expected that (a) higher resting PNS activity would predict better pre-manipulation IC, and (b) for stressor participants, higher (vs. lower) PNS activity during acute stress would indicate resilience to the stressor and relatively better post-manipulation IC.

2. Method

2.1. Participants

Participants were 97 undergraduate students at the University of Oregon (50 [53.2%] female, age $M = 20.09$, $SD = 3.86$) recruited from the Department of Psychology human subjects pool who received course credit for participation.[1] Participants identified as 57.4% non-Hispanic Caucasian race/ethnicity, 14.9% Hispanic, 10.6% Asian, 3.2% Black, and 13.9% other. Three participants declined to report their age, gender, or ethnicity, and age data from an additional 17 were unavailable due to errors in data collection. We assigned participants to the stressor condition at a ratio of 2:1 to have greater statistical power to quantify individual differences in PNS activity within the stressor condition. A target sample size of 100 was linked to the recruitment goal of 70 participants in the stressor condition, based on previous individual difference research investigating PNS activity and cognitive performance (Beaumont et al., 2012). All participants provided informed consent in accordance with the University of Oregon Institutional Review Board.

2.2. Procedure

Before coming to the lab, participants provided informed consent and completed brief online questionnaires that included requests for demographic information. Upon arrival to the lab, participants provided additional consent for the lab portion of the study, which began between noon and 4:00 p.m. to control for diurnal variation in high-frequency heart rate variability (HF-HRV) and cortisol concentrations (Yamasaki et al., 1996). During consent, participants were told that in addition to playing games on a computer, they would be asked to either read magazines or speak in front of a panel of judges. Following consent, participants were asked to wear a Polar Heart Rate monitor for the duration of the experiment (Polar Electro Inc., Lake Success, NY). Participants then completed a brief in-lab questionnaire that is used to assess mental and physical health diagnoses and medication use. Baseline heart rate and HF-HRV were obtained from a four-minute period during which participants were seated. Participants then completed two ~5-min blocks of the stop-signal task, followed by either the stressor or control manipulation. Following this manipulation, participants completed two additional ~5-min blocks of the stop-signal task.

Cortisol was measured to confirm a successful stressor manipulation between groups. Saliva samples (2 mL) were collected

[1] Data from additional participants ($n = 23$) with self-reported mental health diagnoses, neurological conditions, and/or psychotropic medication use were not included in this study, given known associations between these diagnoses and medication use and PNS activity (Beauchaine, 2001; Kemp & Quintana, 2013; Lotufo, Valiengo, Benseñor, & Brunoni, 2012). Due to constraints associated with university human subjects pool recruitment guidelines, they could not be excluded from participation and thus were removed prior to data analysis.