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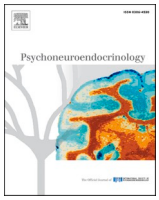

Acute stress influences the emotional foundations of executive control: Distinct effects on control-related affective and cognitive processes☆

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ABSTRACT

Acute stress is known to influence performance on various task outcomes indicative of executive functioning (i.e., the top-down, goal-directed control of cognition and behavior). The most common interpretation of these effects is that stress influences control processes themselves. Another possibility, though, is that stress does not impair control per se, but instead alters the affective dynamics underlying the recruitment of control (e.g., reducing the extent to which making an error is aversive), resulting in less recruitment of control and thus poor performance. To date, however, no work has examined whether stress effects on executive function outcomes are driven by affective dynamics related to the recruitment of control. In the current study, we found that acute stress influenced—and cortisol responses related to—both executive control-related performance outcomes (e.g., post-error slowing) and control-related affective dynamics (e.g., negative affect following recruitment of control) in a modified Stroop task, but that these effects appeared to be independent of each other: The effects of stress on, and associations of cortisol with, control-related cognitive outcomes were not statistically mediated by task- or control-related affective dynamics. These results thus suggest that although stress influences affective dynamics underlying executive function, the effects of stress on executive function outcomes appear to be at least partially dependent on nonaffective processes, such as control processes themselves.

1. Introduction

We have all had the experience of saying or doing something while stressed that we later regret. Perhaps because of its commonality, much work has focused on determining why this experience occurs. Acute stress is known to impair performance on many outcomes indicative of *executive function* (i.e., top-down ability to control our own thoughts and behavior; Geißler et al., 2023; Plieger and Reuter, 2020; Quinn and Shields, 2023; Shields et al., 2016; Tsai et al., 2019). However, the mechanisms underlying these effects are still relatively unclear (though see Chang et al., 2020; Geißler et al., 2023; Shields et al., 2016; Tsai et al., 2019). Although much progress has been made in understanding the biological underpinnings of the effects of stress on executive functions (e.g., Geißler et al., 2023), less work to date has examined the psychological mechanisms that may underlie these effects (though see, for example, Shields et al., 2019b; Tsai et al., 2019). The current study attempts to address that gap.

Acute stress appears to exert complex effects on executive functioning via multiple biological pathways. Although executive functioning is not a monolithic entity, prior work has suggested the existence of a "common" executive function that supports performance across all executive function tasks (Friedman and Miyake, 2017; Karr et al., 2018; Miyake and Friedman, 2012). This common executive function is often referred to as "executive control" or "cognitive control" more generally (e.g., Quinn and Shields, 2023). Acute stress appears to impair performance across a broad range of executive function tasks, supporting the idea that it modulates the recruitment or efficacy of the common executive function (Shields et al., 2016). However, there are nuances to these effects, as acute stress—via a glucocorticoid- or androgenic-dependent mechanism blocked by the drug spironolactone—enhances performance on response inhibition outcomes (Schwabe et al., 2013). Cortisol in particular seems to be important to these response inhibition effects, because although acute stress and hydrocortisone (i.e., synthetic cortisol) administration exert distinct effects on outcomes reflecting

☆ R syntax and data for this manuscript can be viewed at the following link: https://osf.io/pfa7v/?view_only=6d82374dec474a05808ed26667a282b6

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most facets of executive function, they exert indistinguishable effects on response inhibition (Shields et al., 2016, 2015). Therefore, acute stress appears to impair performance on outcomes reflecting executive control (i.e., the common executive function) at least in part via a cortisol-*independent* pathway, whereas it appears to enhance performance on outcomes reflecting response inhibition via a cortisol-*dependent* pathway.

At the psychological level of analysis, however, the mechanisms underlying the effects of stress on outcomes reflecting executive functioning are less clear. Many theories of control posit that we engage executive control to resolve task conflict or prevent or reduce errors (e.g., Diamond, 2013). Importantly, though, as Inzlicht et al. (2015) have noted, the dynamic of recruiting control to resolve conflict between one's present state and one's desired state—such as doing well on a task and presenting oneself as intelligent—is a prevailing view of self- and/or emotion regulation. Drawing on this similarity, Inzlicht and colleagues have proposed that exerting executive control is a type of emotion regulation (Frömer et al., 2021; Inzlicht et al., 2015; Saunders et al., 2015). For example, making errors on trials is frustrating, and the primary or perhaps most common mechanism through which that frustration can be prevented or resolved is by exerting control to resolve conflicts between multiple responses and provide a correct response; however, this frustration can also be prevented by changing the goal state, such that performing well is viewed as less important (i.e., control avoidance; Saunders et al., 2015). The consequences of poor control and "control avoidance" are the same—poor performance on outcomes reflecting executive functioning—indicating that outcomes reflecting executive functioning can reflect both control and affective dynamics underlying the exertion of control. It is thus possible that stress may influence performance on executive function outcomes via affective dynamics underpinning recruitment of control. This idea is attractive due to the known, strong effects of stress on affect and emotion (Burani et al., 2021; Li et al., 2014, 2013; Preston et al., 2022; Sandner et al., 2020). To date, however, the possibility that acute stress influences performance on outcomes reflecting executive functioning via modulation of affective dynamics underlying recruitment of control has not yet been considered.

If recruiting executive control is a form of emotion regulation, then it is possible that performance decrements on executive function tasks that are observed under stress are not due to impairments in control per se, but instead to emotion regulation mechanics that result in poor task performance in a way that mimics impaired control. As shown in Fig. 1, task conflict can be dealt with either by changing the current state or by changing the goal, just like within the cybernetic model of self-regulation. Put differently, stress can impair performance by reducing the ability to resolve the conflict or by increasing the likelihood that someone changes the goal to perform well. If one decides, for example, that one is stressed and does not feel like trying, one will choose to exert less control—even though one could exert control just fine if they chose to do so. The result of this dynamic is a behavioral metric of poor executive function task performance identical to that which would be observed if control itself was impaired (e.g., more errors of commission, a greater enhancement by congruent trials relative to incongruent trials, etc.). To date, however, no study has tested this hypothesis.

1.1. Current research

The current study tested whether the effects of acute stress on, or associations between cortisol and, executive-function-related outcomes were driven by affective dynamics related to exerting control or making errors. To this end, we examined executive function or cognitive control-related cognitive indices and affective dynamics during an executive control task in 155 participants randomly assigned to either an acute stressor (a Zoom-adapted Trier Social Stress Test) or a control condition. Participants provided saliva samples immediately before and 10 min after manipulation offset (28 min after manipulation onset), from which we assayed cortisol. Because cortisol may exert both linear and threshold effects, we examined each outcome in relation to both linear associations with cortisol and cortisol responder group differences. Approximately 13 min post-manipulation offset (31 min post-manipulation onset), participants then completed a modified Stroop task. The Stroop task was modified in that after each response it probabilistically and repeatedly asked participants how they felt in the moment, from which two affective dynamics predicted by the theory of emotional foundations of cognitive control (Inzlicht et al., 2015)—namely, conflict-induced decrease in affect and error-induced decrease in affect—were calculated. Drawing on the work described above, we hypothesized (1) that acute stress would influence performance outcomes within the Stroop task indicative of executive function or cognitive control (i.e., RT interference effects and post-error slowing; described in Method, below), (2) that cortisol responses to the manipulation would predict differential performance on those same outcomes, (3) acute stress and/or cortisol responses would influence affective dynamics related to executive control (i.e., conflict-induced change in affect and error-induced change in affect; described in Method, below), and (4) acute stress effects and/or cortisol links with control-based outcomes would be partially mediated by differences in task-related affective dynamics.

2. Method

2.1. Participants

We recruited 170 young adults (randomly assigned to conditions; 87 control, 83 stress) from a large public university in the South Central United States for this study.[1] Individuals were only invited to participate if they did not take psychotropic medication (e.g., antidepressants, stimulants) or medication(s) that can influence stress responses (e.g., immunosuppressants, beta-adrenergic inhalers, corticosteroids), consume excessive amounts of caffeine (e.g., $>$ 8 cups of coffee per day), have severe sleep disturbances within the prior month (e.g., shift work, chronic insomnia), have an autoimmune or major health disorder (unless a particular disorder is a focus of the study), or take hormonal contraceptives. Additional exclusion criteria were being currently sick or sick over the past week, or pregnancy. We verified compliance with these inclusion and exclusion criteria at the beginning of the study. Of the 170, eight individuals (five control, three stress) were excluded for participating without meeting the above inclusion criteria, and the data from seven more individuals (three control, four stress) were unusable for other reasons (e.g., the participant started the task immediately post-manipulation and without instructions, the participant got up and left the lab to go to the bathroom in the middle of the Stroop task without being between blocks on break, etc.). These issues resulted in a sample of 155 participants with usable Stroop task data (79 control, 76 stress). Of this sample (M_{age}=19.03, SD_{age}=1.75; 57.4% assigned female at birth, one transgender male), 80.0% identified primarily as White, 6.5% as Hispanic or Latino/a/e, 5.8% as Asian or Asian American, 3.2% as Black or African American, 0.6% as Native Hawaiian or Pacific Islander, and 3.9% declined to state or did not provide data. Of this sample, only 0.6% of participants reported smoking more than 1 cigarette per month, and no participant reported smoking more than three cigarettes per month (i.e., none smoked as frequently as once per week). No participant smoked on their day of participation. Female participants also reported the first day of their last menstrual period, which was used to infer their phase of the menstrual cycle (follicular: 0–13 days; luteal: 14–40 days; nonmenstruating or other: 41 + days). The percent in each phase was

[1] This was part of a larger study recruiting a total of 187 participants. Of the 187 participants, 17 were unable to run the task due to miscommunications via Zoom (e.g., clicking on the wrong icon on the desktop and skipping the Stroop task entirely). Only 170 participants opened the Stroop task.

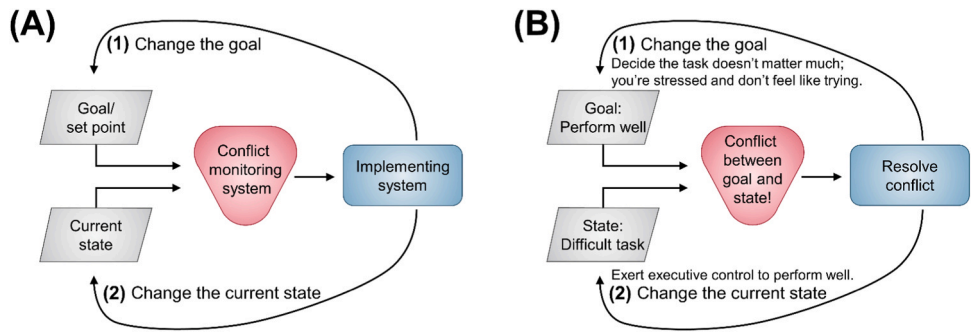

Fig. 1. Depiction of a prominent model of self- and emotion regulation (A) applied to executive function task performance under stress (B). According to this model, negative affect, which is induced by conflict between one's goal and one's present state, can be resolved by either changing the goal (path 1) or changing the current state (path 2). The effects of stress on executive function have typically been understood to be a result of an influence on path 2 or its efficacy, but an influence on path 1 could produce equivalently impairing effects on task performance.

44.9% (follicular), 47.2% (luteal), and 7.9% (nonmenstruating or other).

2.2. Materials

2.2.1. Stress manipulation

The stress manipulation was a Trier Social Stress Test (TSST) (Kirschbaum et al., 1993) that was conducted via Zoom within the lab (with separate room for the participant) due to COVID-related precautions. Prior work has examined Zoom-based TSST manipulations, and the general finding is that these manipulations are successful, albeit weaker than the standard TSST (Gunnar et al., 2021; Meier et al., 2022).

Participants in the stress condition were first introduced to the stressor (anticipation phase) by being told that in three minutes they would give a speech to a committee who were trained in the analysis of nonverbal behavior. Participants were given three minutes to prepare a speech on their actual qualifications for the next step in their career towards their dream job. After three minutes had elapsed, the experimenter left the Zoom call and evaluators joined. Evaluators were trained to look directly at their computers' webcams so that the participant's view was such that the evaluators appeared to be looking directly at them. Evaluators wore white lab coats and were positioned against egg-white solid backgrounds. The evaluators then instructed the participant to begin their speech (speech phase). If at any time the participant stopped speaking or paused for more than 10 s, the evaluators told the participant that they still had some time, and to continue speaking. The speech phase lasted 10 min. After the speech phase had ended, participants were asked to count backwards in steps of 13 from 2934 (mathematics phase). If at any point the participant made an error, they were told to start back at 2934. Further, at prespecified times (90 s, 180 s, and 240 s), participants were told to count faster. Once five minutes had elapsed, the evaluators told the participant to stop and instructed the experimenter to rejoin the Zoom call via text to continue the protocol.

Participants in the control condition were first introduced to the control task (anticipation phase) by being told that in three minutes they would talk quietly to themselves about the topic of their choosing for 10 min, and after that, that they should count up from 1 to 30 as many times as they could until the experimenter rejoined. At the end of the anticipation phase, the experimenter left to Zoom call and started a timer to rejoin after 15 min. After 15 min had elapsed, the experimenter rejoined the Zoom call.

2.2.2. Manipulation checks

2.2.2.1. Negative affect. Negative affect was assessed pre- and post-manipulation using the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988). Participants were asked to report the extent to which they currently felt 10 negative and 10 positive emotions (20 items total). Responses to each item were provided on a 1 (Very slightly or not at all) to 5 (Extremely) scale, and responses to the 10 questions assessing negative affect were then averaged to create an overall index of negative affect, with higher scores indicating more negative affect. Internal consistency for the scale was acceptable both pre- and post-manipulation, $\alpha s \geq .77$.

2.2.2.2. Cortisol. Participants provided two saliva samples (baseline and post-manipulation) via passive drool. Immediately after collection, the saliva vials were placed in a − 20ºC freezer until assayed in duplicate using high-sensitivity Salivary Cortisol ELISA Kits (Salimetrics LLC, State College, PA) according to manufacturer instructions. Eight of the 155 participants did not provide enough saliva for the assay at one or both timepoints and were thus unable to be included in cortisol analyses. Inter-and average intra-assay CVs were 6.79% and 7.34%, respectively. Assay sensitivity was 0.007 μg/dL. All controls were in the expected ranges. Cortisol concentrations were converted from μg/dL to nmol/L.

2.2.3. Modified Stroop task

The Stroop task was used to assess executive functioning and related affective dynamics, as acute stress could potentially produce poor task performance by impairing executive function directly or by altering affective dynamics related to the recruitment of control. This paragraph describes the parameters of the Stroop task itself. Subsequent paragraphs will describe key outcomes created from the task in detail. Participants completed a two-color Stroop task that included probabilistic affect prompts (see Fig. 2). Each trial began with a fixation cross displayed for 0.40 s, followed by the target stimulus. Targets were either the words RED or BLUE (60 pt font, 1920 ×1080 display) and were written in either the same font color as the target's semantic word content (e.g., BLUE written in blue font; *congruent* trials) or the opposite (e.g., BLUE written in red font; *incongruent* trials). Participants were told to indicate the color of the font that the word was written in and to ignore the meaning of the word; participants were instructed to use the "r" key to indicate red font and the "b" key to indicate blue font; no other response keys were accepted. Each target was displayed for the minimum of 2.50 s or until participant response. Following each response, participants were given feedback on their response for 0.75 s (i.e., "Correct!!" or "Incorrect" displayed in 24 pt black font where the target had been). Following feedback, affect prompts were probabilistically displayed depending upon response accuracy. In particular, following targets to which the participant responded correctly, the affect prompt

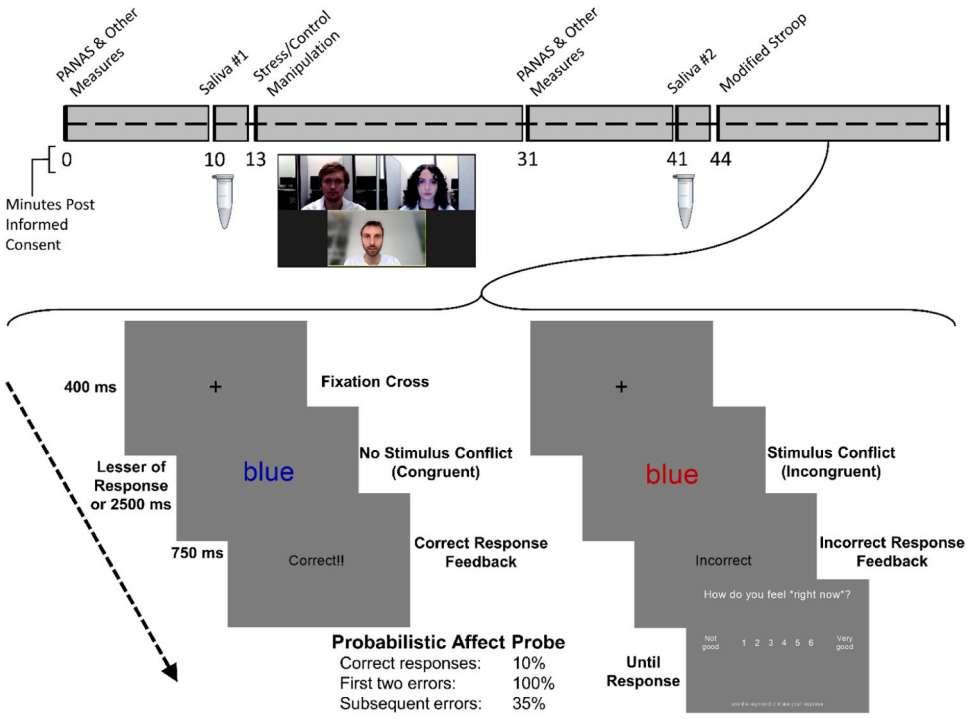

Fig. 2. Depiction of study procedure and modified Stroop task trial types, feedback, and probabilistic affect probes. The stress/control manipulation was conducted via Zoom; the depicted stressor serves to illustrate that it was a TSST, not that it was in-person.

occurred with a 10% probability. Following the first two errors, the affect prompt occurred with 100% probability[2]; following additional errors, the affect prompt occurred with 35% probability. These probabilities were chosen as a result of extensive piloting to produce stable estimates of affect on both congruent and incongruent correct trials, and on both errors and correct trials. The affect prompt consisted of a screen that displayed text asking participants, "How do you feel *right now* ?" and a response scale of 1–6, anchored at 1(*Not good*) and 6(*Very good*). Participants were given unlimited time to make their response, after which the scale was removed from the screen. Of the trials, 66.7% were congruent (split evenly between RED and BLUE), and 33.3% were incongruent (split evenly between RED and BLUE). There were 54 rials (36 congruent, 18 incongruent) in each of six blocks, for a total of 324 trials (216 congruent, 108 incongruent) across the entire task. Between each block of trials, participants were how many of six total blocks they had completed, and they were told to take as long of a break as they would like before continuing. Prior to beginning the task, participants received extensive instructions on both the Stroop task and responding to the affect prompt, and they completed twelve practice trials before completing the primary task. Task performance was used to create two executive control outcomes (i.e., Stroop interference effect and post-error slowing) and two outcomes measuring affective dynamics related to recruiting control. These four dependent variables are described in greater detail below.

2.2.3.1. *Executive functioning*

2.2.3.1.1. Stroop interference effect. The most common executive function-related outcome of interest in this task is the Stroop RT interference effect (or, *Stroop effect*), which is the difference in reaction time between correct incongruent trials and correct congruent trials. Larger Stroop effects therefore indicate stronger biasing of processing by goal-irrelevant information (i.e., semantic word content). Stroop effects may be driven by response inhibition, common executive control, or both control processes, but are nonetheless driven by at least one form of executive functioning (Friedman and Miyake, 2004; Karr et al., 2018; Roos et al., 2017; Shields, 2017; Shields and Yonelinas, 2018).

2.2.3.1.2. Post-error slowing. Following an error, most people respond more slowly on subsequent trials. This phenomenon, known as *post-error slowing*, has previously been related to cortisol (Tops and Boksem, 2011) and is thought to reflect (at least) two aspects of cognitive control. The first aspect is error detection, or the ability to recognize that one indeed made an error; individuals with ADHD, for example, show poorer error detection on conflict tasks due to greater mind wandering or lapses in goal maintenance, and subsequently adapt their behavior less—that is, have less post-error slowing—as a result (Arnett et al., 2021; van Meel et al., 2007). However, because the task used in this study has extensively presented trial feedback following each response, any observed post-error slowing in this study is unlikely to reflect individual differences in error detection: All individuals are explicitly told whether they made an error or not in each response, there was no need to remember the task rules to know whether responses were in error. The second aspect of cognitive control that post-error slowing reflects is a resource bottleneck: The extent to which errors slow responses is indicative of fewer attentional and control-related resources that can be deployed to simultaneously attend to an error and the current stimulus, or to simultaneously reconfigure control deployment and attend to the current stimulus (Houtman and Notebaert, 2013; Lavro et al., 2018; Notebaert et al., 2009). Greater post-error slowing has been related to real-world outcomes indicative of poorer cognitive control, such as a greater likelihood of future relapse in abstinent individuals with alcohol use disorder (Wang et al., 2023). Thus, greater post-error slowing, within the context of this task (i.e., with error detection removed from influence), reflects poorer cognitive control.

Post-error slowing was calculated from mean post-error minus pre-

[2] The condition triggering the affect prompt with 100% probability on the first two errors was added on the second day of running the study, when two participants made so few errors that they did not see the prompt; for the first four participants, their error prompt occurred with 35% probability in every error trial. This was the only modification made to the task during the study, and excluding the participants who participated prior to this task change did not influence the results.

error RT (Dutilh et al., 2012). We excluded trials on which participants had provided affect responses at the end of the prior trial, as the time permitted to provide an affect response nullifies prior-trial-related resource bottlenecks (affect response time was unlimited).

2.2.3.2. Affective indices relevant to control. Consistent with the theory of emotional foundations of cognitive control, we expected affect to be lower on trials requiring exertion of control (i.e., correct incongruent trials relative to correct congruent trials; *conflict-induced Δ affect*), and lower following errors (i.e., errors relative to correct responses; *error-induced Δ affect*). These two Δ affect scores thus served as the primary control-related affective outcomes in this task. We used Δ affect in these measures so that a change from zero was intuitively interpretable, but using residualized change scores (as we did with cortisol; see below) led to equivalent results (see syntax and data).

2.3. Procedure

The study procedure is depicted in Fig. 2. Upon arrival at the lab, participants were taken to an isolated room, wherein they completed all measures. The experimenter joined a Zoom call on a second computer in a separate room to guide the participants through the procedures, starting with informed consent. After providing informed consent, participants completed acclimation measures for approximately 10 min. Participants then provided the baseline measure of negative affect and baseline saliva sample before completing the stress or control task (depending upon randomly assigned condition). The stress manipulation lasted a total of 18 min. Following the manipulation, participants immediately completed the post-manipulation negative affect assessment and subsequently completed filler questionnaires until exactly 10 min had elapsed since the stress manipulation had ended. At 10 min post-manipulation offset, participants provided the post-manipulation saliva sample. Next, participants then completed the modified Stroop task, which lasted approximately 15 min. Participants then completed tasks unrelated to the present study before finally being debriefed and dismissed.

2.4. Data analysis

All analyses were conducted in R, version 4.3.1. Bayesian parameter estimation formed the basis of all between-group, between-condition, between/within-condition, and regression analyses using Stan and JAGS, via R, with the packages rstanarm (v2.21.4), brms (v2.19.0), BANOVA (v1.2.1), BEST (v0.5.4), and bayestestR (v0.13.1) (Bürkner, 2017; Dong and Wedel, 2017; Kruschke, 2013; Makowski et al., 2019; Muth et al., 2018). Bayesian parameter estimation is robust against unbalanced designs (e.g., Kruschke, 2013). In addition, Bayesian parameter estimation goes further than providing sample summary statistics in that it estimates population values. Therefore, keeping with convention (Kruschke, 2013), we report Bayesian estimates with their respective Greek symbols—denoting population value estimates. All chains converged according to model diagnostics. Provided Bayesian *p* values indicate the one minus probability of direction, multiplied by two, denoted $p_{d\times2}$, thus being interpretable similarly to a two-tailed *p* value in frequentist statistics. Results with frequentist statistics were similar; the primary condition differences were that, without removing outliers, the effects on post-error slowing were slightly stronger but the effects on conflict-induced Δaffect were slightly weaker, but with outliers removed, the frequentist results were equivalent to those reported below (see syntax and data). The cortisol responder group analyses were also stronger with frequentist statistics; these analyses are shown within a figure in the results.

As we have done previously (e.g., Shields et al., 2019a), we calculated changes in cortisol by regressing post-manipulation cortisol on pre-manipulation cortisol. Residualized changes were calculated for negative affect in the same way. Residualized changes were used instead of simple change scores because residual change scores are more reliable than simple change scores by better accounting for the influence that basal values have on change scores (Cronbach and Furby, 1970). Using simple change scores (i.e., Δ cortisol) did not alter the results. Residualized change scores were centered at their mean Δ change scores for interpretability.

Cluster analyses were conducted using the mclust package (v6.0.0) in R. We characterized cortisol response groups using cluster analyses in these data rather than using the recommended optimally sensitive and specific 1.5 nmol/L cutoff, or the more highly specific 2.5 nmol/L cutoff, described elsewhere (Miller et al., 2013) for two reasons. First, we expected that our Zoom-based stressor might produce different response profiles than other stress manipulations, and we wanted to account for this possibility. Second, we wanted to compare negative affect stress responders to cortisol stress responders in order to ensure that our cortisol responder group analyses reflected cortisol specifically, rather than a more general greater stress response, and we thus used the same method to derive response profile clusters for both cortisol and negative affect. Using cortisol responder cutoffs of 2.5 nmol/L produced similar, albeit slightly weaker, results compared to those described below (see Supplement).

We did not cluster changes in affect and cortisol together in our primary analyses because these cluster analyses were not intended to produce stress clusters—the experimental manipulation provided those—but to produce particular response clusters. This approach allows us to explore categorical effects, and perhaps even causal (though this cannot be addressed by our data), effects of potentially important variables in relation to our outcomes of interest. In other words, the clustering approach allowed us to examine how profiles of cortisol responses and profiles of affect responses independently–across conditions–related to our primary outcomes of interest. We report within Supplemental Material cluster analyses that concurrently clustered both changes in negative affect and changes in cortisol.

We derived clusters across conditions, rather than clustering within the stress condition alone. This is a nonstandard approach to clustering particular responses, but it was intentional: It is the appropriate approach if cortisol (or, negative affect) itself causally influences the outcome. That is, if an increase in cortisol (or negative affect) itself affects task performance, it would affect task performance in the same way regardless of the condition to which participants were assigned. Our stress manipulation effects are described in the experimental condition analysis, and our cortisol cluster (and negative affect cluster) analyses thus intentionally cut across experimental conditions—these analyses examine additional potential influences on task performance, rather than potential mediators. We note that "potential" is an important word here, because the cortisol (and PANAS negative affect) change data are correlational, but our analyses of them across experimental conditions are at least consistent with the idea that cortisol (or negative affect) itself exerts an effect on our outcomes independent of experimental condition. Cluster analyses separating control participants from stress clusters are presented within Supplemental Material.

3. Results

3.1. Stress responses

Consistent with hypotheses, the Zoom-based stressor increased both negative affect and cortisol relative to control conditions. In particular, we observed a Condition×Time interaction effect in negative affect, $\eta^2_p = .230$, $p_{d\times2} < .001$, and cortisol, $\eta^2_p = .100$, $p_{d\times2} < .001$ (see Fig. 3). Decomposing these interactions, we found that the stress and control conditions did not differ in either negative affect ($\delta = -0.11$, $p_{d\times2} = .586$) or cortisol ($\delta = 0.16$, $p_{d\times2} = .433$) at baseline, whereas, relative to control participants, participants in the stress condition showed higher levels of both negative affect ($\delta = 1.65$, $p_{d\times2} < .001$) and cortisol

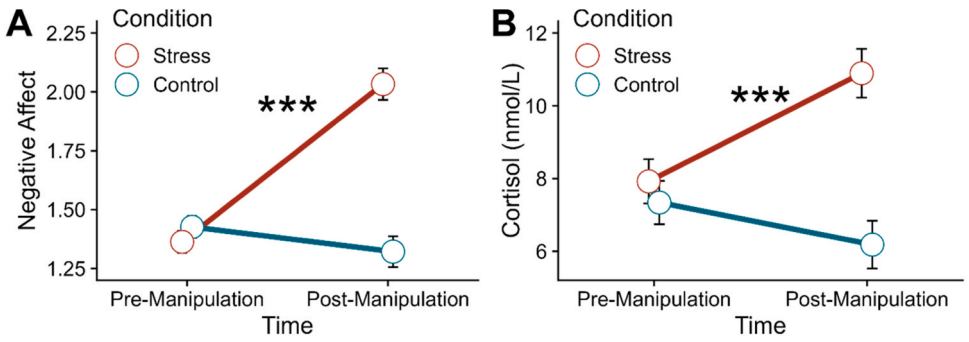

Fig. 3. Stress effects on negative affect (A) and cortisol (B). The manipulation increased negative affect and cortisol in the stress condition, not the control condition. Depicted means and error bars are marginal means and standard errors from observed data.

($\delta = 1.05$, $p_{d\times2} < .001$) post-manipulation.

To determine negative affect and cortisol responder groups, we compared the fits of models estimating one to nine latent clusters with equal and unequal variances for each outcome (18 models for negative affect responses; 18 models for cortisol responses) across conditions. We fit the model across experimental conditions for two reasons. First, coming to the lab during pandemic safety protocols may have been stressful for undergraduates, and given the ambiguity within the control condition, we wanted to allow control participants into responder groups if their data merited it and vice versa. Second, more importantly, if cortisol or negative affect exert causal effects on outcomes of interests, they should do so regardless of the experimental condition to which a participant was assigned. Therefore, we estimated response groups across conditions. In these analyses, we found that the best-fitting model for negative affect responses was a two-group, unequal variance model, BIC= −232.6, all ΔBICs> 11.3. In contrast, the best-fitting model for cortisol responses was a three-group, unequal variance model, BIC= −756.6, all ΔBICs> 3.77. Fig. 4 depicts these response groups.

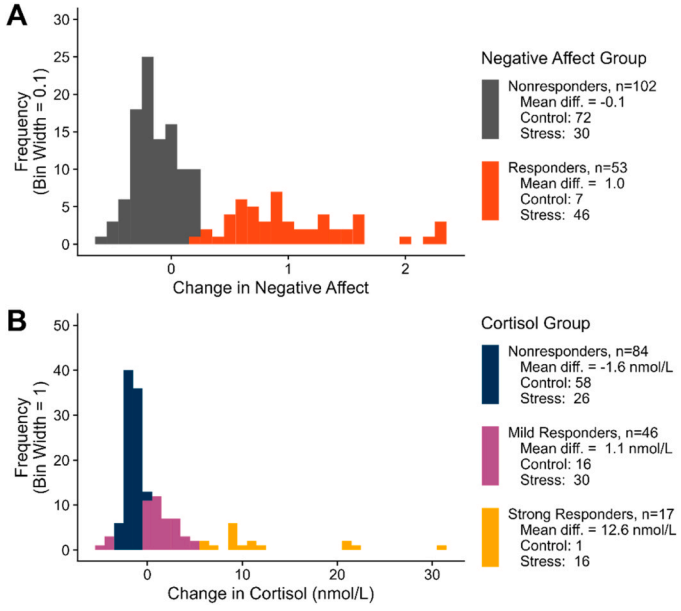

Fig. 4. Negative affect (A) and cortisol (B) responses to the manipulation by latent cluster. Reassigning the four mild cortisol responders with negative values to the nonresponder group only strengthened the cortisol responder results.

3.2. *Effects of stress on, and associations of stress responses with, the primary outcomes*

We next examined the effects of the stress manipulation on the control-related cognitive and control-related affective outcomes in the modified Stroop task.

3.2.1. *Effects of stress on primary outcomes*

Consistent with expectations, we found that, relative to the control condition, acute stress increased both post-error slowing (μ_{stress}=180.73, σ_{stress}=144.56; $\mu_{control}$=125.34, $\sigma_{control}$=109.41), $\mu_{difference}$= 55.40, $p_{d\times2}$ = .049, $\delta = 0.43$, and stimulus conflict-induced decreases in affect (μ_{stress}= −0.05, σ_{stress}=0.21; $\mu_{control}$=0.04, $\sigma_{control}$=0.13), $\mu_{difference}$= −0.09, $p_{d\times2}$ = .012, $\delta = -0.52$ (Fig. 5). Stress also either significantly increased or tended to increase heterogeneity (i.e., variability) both of these outcomes ($\sigma_{difference}$=35.15, $p_{d\times2}$ =.137, and $\sigma_{difference}$=0.08, $p_{d\times2}$ =.013, respectively), indicating that there may be subgroups in the effects of stress on these outcomes. However, contrary to expectations, stress did not influence either Stroop interference effects or error-induced Δaffect overall, $|\delta|s < 0.09$, $p_{d\times2}s > .613$, nor did it influence hetereogeneity in these outcomes, $p_{d\times2}s > .689$.

3.2.2. *Stress response associations with primary outcomes*

3.2.2.1. Negative affect. We found that pre- to post-manipulation change in negative affect predicted post-error slowing, $\beta = .188$, $p_{d\times2}$ = .039, and marginally predicted stimulus conflict-induced Δaffect, $\beta = -.146$, $p_{d\times2}$ = .068, whereas it was not a significant predictor of Stroop interference effects, $\beta = -.040$, $p_{d\times2}$ = .612, or error-induced Δaffect, $\beta = -.069$, $p_{d\times2}$ = .401. Removing five outlying negative affect values > 1.9 (Fig. 4a) reduced the association between pre- to post-manipulation change in negative affect and post-error slowing to nonsignificance, $\beta = .160$, $p_{d\times2}$ = .075 (and did not change the association with Stroop interference, $p_{d\times2}$ =.619), but numerically strengthened the association between pre- to post-manipulation change in negative affect and both stimulus conflict-induced Δaffect, $\beta = -.185$, $p_{d\times2}$ = .023, and error-induced Δaffect, $\beta = -.139$, p_d= .095.

We next tested threshold associations by examining negative affect responder group differences in our primary outcomes. However, none of the above outcomes differed by negative affect response group, $p_{d\times2}s > .272$.

3.2.2.2. Cortisol. For associations of pre- to post-manipulation changes in cortisol with control-related cognitive and affective outcomes, we found that cortisol response was associated with Stroop interference effects, $\beta = -.160$, $p_{d\times2}$ = .050, but not with post-error slowing, $\beta = .096$, $p_{d\times2}$ = .316, stimulus conflict-induced Δaffect, $\beta = -.149$, $p_{d\times2}$ = .073, or error-induced Δaffect, $\beta = -.081$, $p_{d\times2}$ = .346. However, removing the four cortisol outliers with changes above 20 nmol/L (see Fig. 4b) strengthened these associations substantially. In particular,

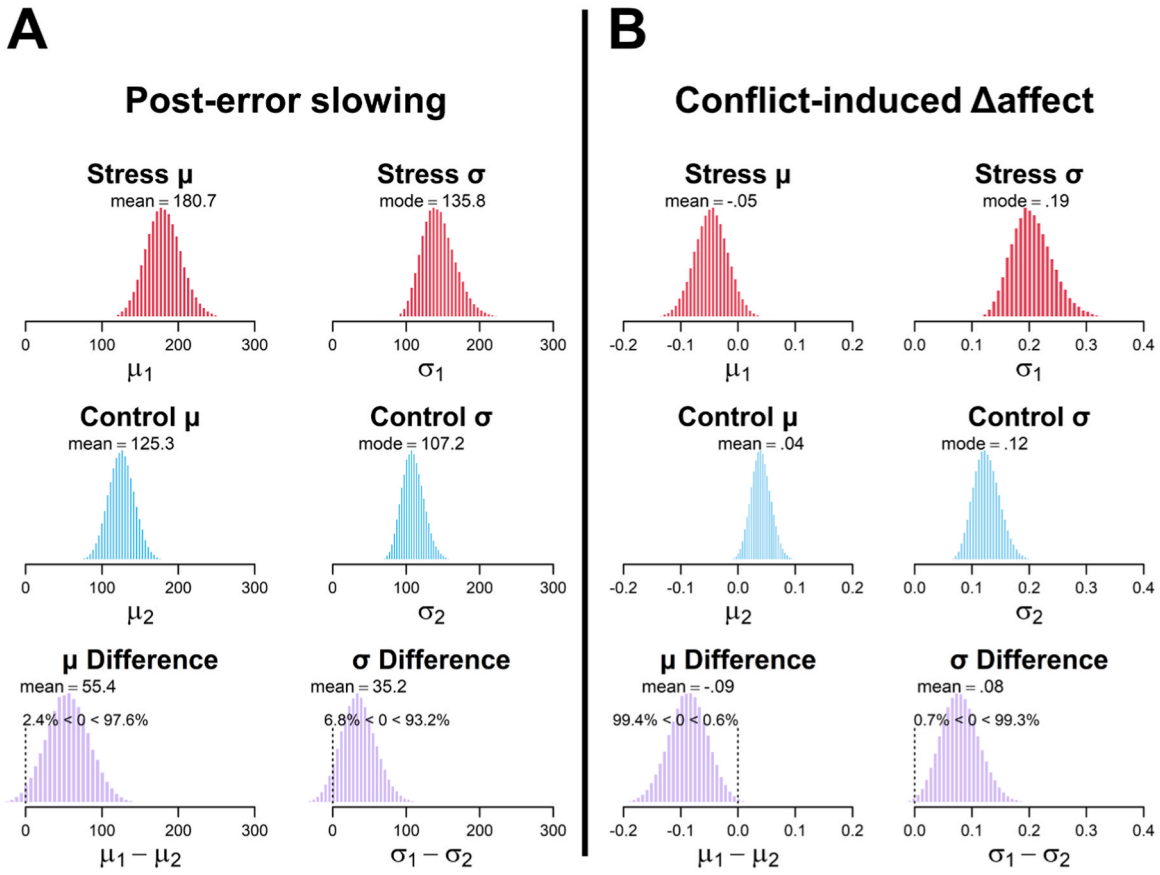

Fig. 5. Effects of stress on post-error slowing (A) and conflict-induced Δaffect (B). Participants in the stress condition showed significantly greater post-error slowing, and significantly greater decreases in affect following correct responses to incongruent (vs. congruent) trials, relative to participants in the control condition.

after removing outliers, cortisol responses were significant predictors of greater post-error slowing, $\beta = .294$, $p_{d\times2} = .002$, smaller Stroop interference effects, $\beta = -.190$, $p_{d\times2} = .022$, and stimulus conflict-induced Δaffect, $\beta = -.186$, $p_{d\times2} = .025$, and were marginally associated with error-induced Δaffect, $\beta = -.160$, $p_{d\times2} = .061$.

We next tested threshold or categorically distinct relations by examining cortisol responder group differences in our primary outcomes. In these analyses, we found that cortisol response group was a marginal predictor of post-error slowing, $R^2 = .055$, $p_{d\times2} = .050$. Probing this, we found that strong cortisol responders ($\mu = 286.0$ ms) showed both more post-error slowing than mild responders ($\mu = 143.0$ ms), $\mu_{diff} = 143.0$ ms, μ_{diff} 95% credible interval: [20.4 ms, 265.0 ms], $p_{d\times2} = .023$, and marginally more post-error slowing than nonresponders ($\mu = 172.1$ ms), $\mu_{diff} = 113.9$ ms, μ_{diff} 95% credible interval: [− 2.8 ms, 230.9 ms], $p_{d\times2} = .055$, whereas mild responders and nonresponders did not differ, $\mu_{diff} = 29.1$ ms, μ_{diff} 95% credible interval: [− 51.8 ms, 110.0 ms], $p_{d\times2} = .475$ (Fig. 6).

Similarly, cortisol response group was a marginal predictor of Stroop interference effects, $R^2 = .057$, $p_{d\times2} = .062$. Probing this, we found that strong cortisol responders ($\mu = 73.0$ ms) showed smaller Stroop interference effects than both mild responders ($\mu = 113.8$ ms), $\mu_{diff} = 40.7$ ms, μ_{diff} 95% credible interval: [7.8 ms, 73.5 ms], $p_{d\times2} = .015$, and nonresponders ($\mu = 113.0$ ms), $\mu_{diff} = 40.0$ ms, μ_{diff} 95% credible interval: [9.4 ms, 70.4 ms], $p_{d\times2} = .011$, but mild responders and nonresponders did not differ, $\mu_{diff} = -0.7$ ms, μ_{diff} 95% credible interval: [− 21.8 ms, 20.4 ms], $p_{d\times2} = .943$ (Fig. 6).

In relation to control-related affective outcomes, cortisol response group was a significant omnibus predictor of stimulus conflict-induced Δaffect, $R^2 = .058$, $p_{d\times2} = .011$. Probing this, we found that strong cortisol responders ($\mu = -0.19$) showed marginally greater decreases in affect on correct incongruent trials relative to correct congruent trials than mild responders ($\mu = -0.03$), $\mu_{diff} = -0.16$, μ_{diff} 95% credible interval: [− 0.03, 0.34], $p_{d\times2} = .096$, and significantly greater conflict-induced decreases in affect than nonresponders ($\mu = 0.04$), $\mu_{diff} = -0.23$, μ_{diff} 95% credible interval: [0.06, 0.41], $p_{d\times2} = .009$, whereas mild responders and nonresponders did not differ, $\mu_{diff} = -0.07$, μ_{diff} 95% credible interval: [− 0.19, 0.05], $p_{d\times2} = .229$ (Fig. 6). Notably, only strong cortisol responders showed the predicted significant decrease in affect (i.e., only their 95% credible interval of the mean did not include 0) following conflict trials in the absence of an error, indicating that large cortisol responses may produce an affective phenotype more sensitive to task difficulty even in the absence of an error.

Finally, cortisol response group was a significant omnibus predictor of error-induced Δ affect, $R^2 = .046$, $p_{d\times2} = .033$. Probing this, we found that strong cortisol responders ($\mu = -0.82$) showed significantly greater decreases in affect following errors relative to correct responses than nonresponders ($\mu = -0.40$), $\mu_{diff} = -0.41$, μ_{diff} 95% credible interval: [− 0.80, − 0.03], $p_{d\times2} = .035$. Mild responders ($\mu = -0.53$) did not differ from strong responders or nonresponders, $p_{d\times2}$s > .208 (Fig. 6).

3.3. Mediation analyses

Finally, we explored potential statistical mediation or indirect effects of condition, PANAS negative affect response group, or cortisol response group on Stroop effects and post-error slowing via either stimulus-conflict-induced changes in affect or task-error-induced negative affect. However, none of these analyses were significant: stress condition mediation analyses, $p_{d\times2}$s > .814; PANAS negative affect responder group mediation analyses, $p_{d\times2}$s > .498; cortisol responder group mediation analyses, $p_{d\times2}$s > .164. Therefore, the effects of stress on—or associations of either stress-manipulation-related changes in negative affect or cortisol with—control-related cognitive outcomes (i.e., post-

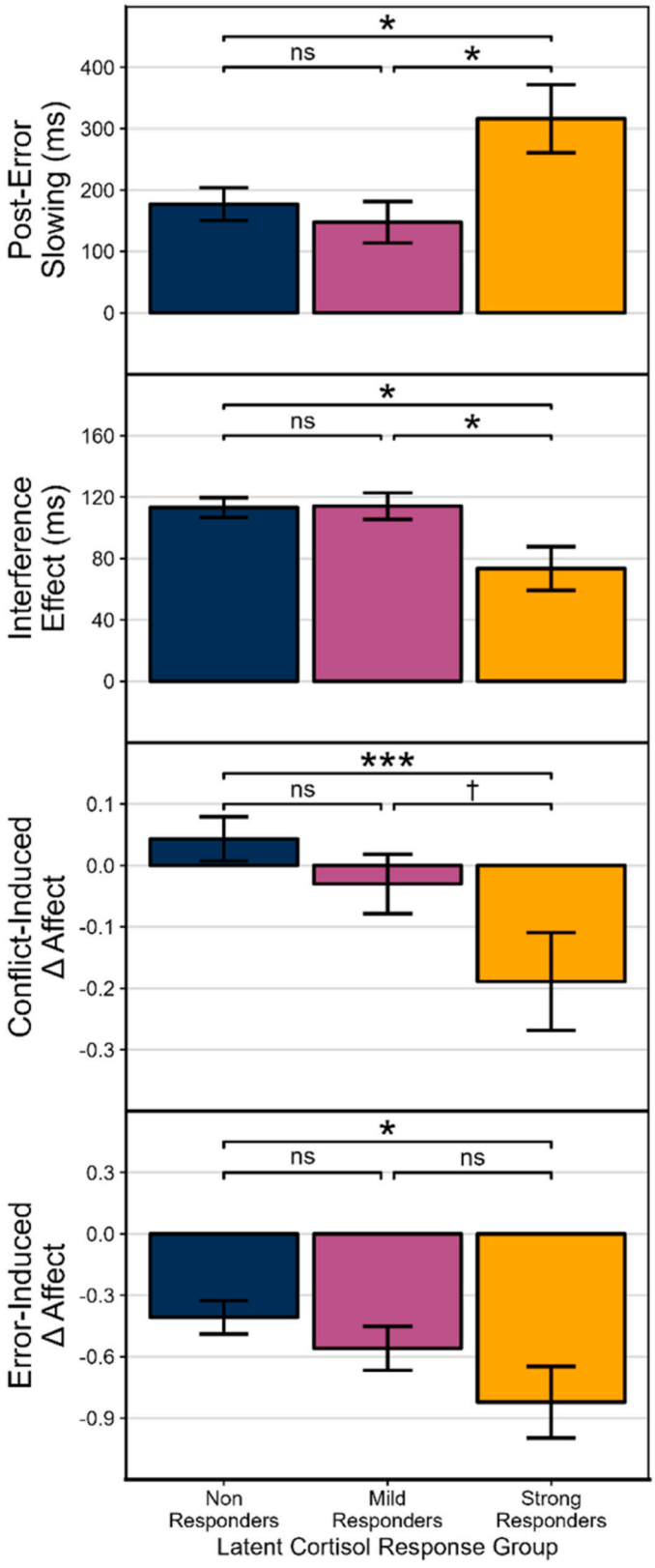

Fig. 6. Participants with strong cortisol responses to the manipulation differed in control-related cognitive and affective outcomes from mild and non-responders. Marginal means, their standard errors, and their significance tests in observed data are shown.

error slowing and Stroop interference effects) were not driven by their effects on control-related, task-specific affect dynamics (i.e., either stimulus-conflict-induced changes in affect or task-error-induced changes in affect). In other words, although stress is influencing both control-related cognitive outcomes and control-related affective outcomes, these effects may occur via at least partly distinct mechanisms.

3.4. Additional analyses

We present the results of alternative analyses (e.g., using different responder group categorizations) in Supplemental Material.

4. Discussion

Although stress is known to impair performance on many task outcomes thought to measure executive functions, no work to date had assessed whether these performance impairments primarily reflect a modification of the affective foundations of executive (or cognitive) control, rather than an impairment in control itself. We addressed that gap in the present study. We found that although stress influenced both control-related cognitive outcomes and control-related affective outcomes, stress did not influence control-related cognitive outcomes through its control-related affective effects. These results thus suggest that stress influences Stroop task performance through control per se, or at least some cognitive process itself, rather than via the affective processes involved in initiating control as assessed in this study.

Consistent with prior work on affective dynamics within executive function tasks (Inzlicht et al., 2015; Saunders et al., 2015), we found that participants reported greater negative affect following errors compared to correct responses. Moreover, greater error-related decreases in negative affect were related to greater post-error slowing (data not shown). These results suggest that error-related negative affect may be an important catalyst in the decision to exert control.

We also found that participants in the stress condition experienced significantly greater negative affect, both relative to their baseline and relative to participants in the control condition, following incongruent trials to which they responded correctly, relative to correct congruent trials. These results therefore suggest that stress may alter the affective dynamics underlying the exertion of cognitive control, contributing to a state of negative affect as a result of exerting control. This finding is consistent with other work that has found that stress increases cognitive effort avoidance (Bogdanov et al., 2021). Importantly, though, our results suggest that stress may lead to effort avoidance while also decreasing cognitive control capabilities; stressed individuals may choose to exert less control when possible because they have less control to give, rather than performing worse on cognitive tasks because they are exerting less control for no other reason than choice.

An interesting finding alluded to above is that *only* participants in the acute stress condition exhibited an increase in negative affect—or a decrease in positive affect—as a result of stimulus conflict in the absence of a task error. This change in affect is a critical prediction of the theory of the emotional foundations of cognitive control (Inzlicht et al., 2015; Saunders et al., 2015), and yet it only appears to emerge when participants are stressed. These results therefore suggest that non-stressed individuals, although they dislike committing task errors, are not inherently averse to exerting control itself—assuming that they are able to answer correctly—whereas stressed individuals do conform to the predictions of the theory of emotional foundations of cognitive control. Together, the results highlight important refinements to the theory of emotional foundations of cognitive control, suggesting complex interactions between basal stress or affect and task- and control-related affective dynamics.

An important caveat to the above, however, is that affect is more complex than a one-dimensional scale from good to bad. Indeed, dimensional approaches to affective states have shown that these states can vary along multiple dimensions (Kaczmarek et al., 2021; Warriner

et al., 2013). It is possible that the links between cortisol and Stroop interference would have been statistically mediated by affective dynamics had affect been examined in more fine-grained detail. However, we chose to assess affect in this way for two reasons. First, many people are relatively unable to differentiate dimensions of affect or emotions (Feldman Barrett, 1998). Therefore, we did not attempt to assess components of participants' affective states in part for this reason. Second, these affect prompts occurred regularly, and both the number of breaks and the length of breaks between trials influences cognitive task performance by reducing cognitive fatigue (Gilsoul et al., 2022; Lim and Kwok, 2016). As a result, we chose to keep these affect prompts as simple as possible to minimize their cognitive-fatigue-mitigating influence. Future work should examine other potentially relevant affective dynamics, such as reward, that both are altered by stress and influence executive control in order to determine whether the control-related effects of stress are mediated by other affective dynamics.

Another interesting finding that emerged was the marked difference between the strong cortisol responders and both mild and non-responders in post-error slowing. Although we can only speculate about why this finding emerged, we believe that this marked difference is suggestive of categorical or threshold-type effects by cortisol responses in relation to cognitive outcomes. More speculatively, this finding could be taken to suggest categorically different effects of mild versus moderate-to-severe stress on cognitive outcomes (e.g., Shields et al., 2019b). Future work should attempt to explore these possibilities.

The effects of stress on and associations of cortisol with control-related cognitive outcomes merit discussion. Both stress and cortisol were linked to greater post-error slowing. As described in the Method section, post-error slowing in this task does not reflect error detection processes themselves, but a resource bottleneck indicative of poorer control (e.g., Lavro et al., 2018). Intriguingly, however, although stress was unrelated to Stroop interference effects, cortisol was related to *lower* Stroop interference effects—indicating that strong cortisol responses predicted better executive control. This differential association of cortisol with distinct forms of executive functioning is similar to the effects of more severe stressors than the stressor used in this study (i.e., moderate-to-severe stressors, unlike the mild stressor in this study) on executive functions (Dierolf et al., 2018, 2017; Sänger et al., 2014; Shields et al., 2016), suggesting that cortisol may play a critical role in stress-induced differential effects on control processes (see also Schwabe et al., 2013). However, the lack of effect of stress on Stroop interference suggests that stress may exert at least partly opposite effects on processes underpinning Stroop interference effects via a cortisol-independent pathway.

The association we observed between strong cortisol responses to the manipulation and Stroop effects is consistent with prior work examining the effects of stress on Stroop task performance, which has found a reduction in Stroop interference effects (Chajut and Algom, 2003). The cognitive bases of these effects (e.g., changes in response inhibition, cognitive inhibition, etc.) are unclear, however, since Stroop interference effects index and conflate numerous cognitive processes (Botvinick et al., 2001; Cieslik et al., 2015; Jacoby et al., 2003; Ulrich et al., 2015). The association with cortisol makes it tempting to speculate that improvements in response inhibition underlie these effects, as cortisol administration improves metrics of response inhibition (Shields et al., 2015), whereas a mineralocorticoid receptor antagonist abolishes the typically observed stress-related enhancement in response inhibition (Schwabe et al., 2013). In contrast, stress often impairs performance on metrics of cognitive inhibitory control (e.g., selective attention or interference control; Sänger et al., 2014; Shields et al., 2016). Future work using computational modeling or paradigms that independently manipulate the importance of response inhibition versus cognitive inhibition to task outcomes could help to tease apart the cognitive mechanisms underlying this stress effect.

We observed a wide array of cortisol responses to the manipulation, including one control participant exhibiting a strong response, which merits speculative discussion. It is possible that we observed this wide array due to then-current events: Some of the data collection occurred in 2021, prior to widespread COVID vaccine availability, which may have influenced our results. For example, participants in the stress and control conditions who believed that they might have to interact with an experimenter in person at some point during the study might have experienced heightened distress due to health-related anxiety. Alternatively, individual differences in the perception of video interviews may have differentially influenced cortisol trajectories. For example, some stress-condition participants may have viewed video interviews as highly aversive and show strong responses, whereas others may have seen them as less aversive than in-person interviews. This idea could also explain the strong response in one control participant: Each control participant was the sole member of their video call during the control task, entailing that they observed their own speech on fullscreen. Importantly, individuals with social anxiety find this video self-observation highly aversive (Vriends et al., 2017). Indeed, eyetracking indices of self-viewing on video calls is a strong predictor of self-reported anxiety and stress even after the call has finished (Vriends et al., 2017). A third possibility relates to eating or drinking prior to arrival, but we view this as less likely: We instructed participants not to eat or drink anything besides water within the two hours before their arrival, verified compliance with these instructions upon arrival, and included a 15-minute acclimation period to provide a more similar baseline across participants regardless of behavior prior to arrival. However, as in any acute stress study, it is still possible that some participants ate shortly prior to arrival and then lied upon arrival.

A final important point for discussion is the magnitude of the manipulation-induced change in negative affect. In particular, approximately 30 participants in our stress group did not show an increase in negative affect. This lack of change in approximately half of the participants is fairly common when the PANAS is used rather than visual analogue scales for stress (e.g., Berretz et al., 2022; Simon et al., 2022; Villada et al., 2016). This may be related to fewer demand characteristics present in the PANAS than visual analogue scale items of stress within the context of stress, or it may be related to the PANAS assessing negative affect rather than stress per se; regardless, the response rate and magnitude of the PANAS change that we observed is consistent with a number of prior studies (e.g., Shields, 2020).

This study has several strengths, including a large sample size, a novel task with probabilistic affect probes, an experimental design, and appropriate cluster analyses for classifying latent stress response groups. However, it has several limitations that should be noted. First, our stressor was a Zoom adaptation of the TSST. This stressor produced a much smaller cortisol response than a typical TSST, which presumably accounts for the lack of condition effects on task outcomes. Our cortisol responder and correlational analyses, however, suggest that a typical TSST, with its stronger average stress induction, might reduce Stroop interference overall. Second, we relied on self-reported affect in the task, and it is possible that self-reported affect is not a very reliable indicator of participants' true affective state. However, this methodological issue plagues nearly all affect research and suggests that some confidence in inference could be made. Future work should follow up with physiological measures of real-time negative arousal, such as skin conductance. Third, we included a young adult sample, and college-age participants might differ in affective mechanisms underlying the utilization of control during stress. Fourth, as alluded to above, Stroop interference effects are thought to be a reflection of numerous processes, including controlled attention, automatic attentional activation, inhibitory processes, decisional processes such as response caution or threshold, and motor processes such as non-decision time. These results, therefore, cannot speak to whether the observed effects of stress were driven by or due to any one of these specific cognitive processes. Future work should attempt to address this limitation. Fifth, our strong cortisol responder group was relatively small ($n = 17$), and inferences related to strong cortisol responses in this study should thus be made cautiously. Sixth,

although our results were mostly robust to the inclusion or exclusion of outliers, we note that removal of four outlying values in pre- to post-manipulation changes in PANAS-derived negative affect reduced some associations of that negative affect change with our primary outcomes to nonsignificance. Conversely, removing outliers strengthened the cortisol results. Therefore, these correlational results should be interpreted with caution. Finally, our sample was predominantly white, and somewhat predominantly female. It is possible that these results would have differed with samples differing by culture, race, ethnicity, or sex. Future work should replicate these results in a non-WEIRD sample.

In conclusion, we examined the effects of stress on, and associations of stress responses with, both control-related cognitive outcomes and control-related affective dynamics underlying the exertion of control. We found that stress influenced, and cortisol related to, both control-related performance outcomes and control-related affective outcomes. However, the effects of stress on—or associations between cortisol and—control-related performance outcomes were not mediated by affective dynamics related to exerting control or committing errors. Stress may thus influence control-related performance outcomes via control or other cognitive processes, rather than via affective dynamics related to negative affect that occurs as a result of errors or task conflict. Future work should attempt to determine whether other affective dynamics, such as reward-related dynamics, rather than a simple reduction in affective valence, might differ under stress in such a way as to influence control-related performance. In short, the effects of stress on control-related performance outcomes do not appear to be driven by control-modulatory affective dynamics related to committing an error or dealing with stimulus conflict: Stress seems to influence control processes themselves. To illustrate these dynamics more concretely, imagine having a stressful disagreement with someone. In this, relative to when you are not stressed, you may find it more aversive than usual to try to stop yourself from being rude, and—again, relative to not being stressed—you may have a harder time than usual withholding rude behaviors or statements. Our findings support the existence of both of these effects, and our findings further suggest that, perhaps surprisingly, these effects appear to be relatively independent. Put simply, stress both decreases the effectiveness and increases the aversiveness of exerting executive control, and these effects appear to be at least somewhat independent.

Declaration of Competing Interest

None.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.psyneuen.2023.106942.

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