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Influence of acute stress on response inhibition in healthy men: An ERP study

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Abstract

The current study investigated the influence of acute stress and the resulting cortisol increase on response inhibition and its underlying cortical processes, using EEG. Before and after an acute stressor or a control condition, 39 healthy men performed a go/no-go task while ERPs (N2, P3), reaction times, errors, and salivary cortisol were measured. Acute stress impaired neither accuracy nor reaction times, but differentially affected the neural correlates of response inhibition; namely, stress led to enhanced amplitudes of the N2 difference waves (N2d, no-go minus go), indicating enhanced response inhibition and conflict monitoring. Moreover, participants responding to the stressor with an acute substantial rise in cortisol (high cortisol responders) showed reduced amplitudes of the P3 of the difference waves (P3d, no-go minus go) after the stressor, indicating an impaired evaluation and finalization of the inhibitory process. Our findings indicate that stress leads to a reallocation of cognitive resources to the neural subprocesses of inhibitory control, strengthening premotor response inhibition and the detection of response conflict, while concurrently diminishing the subsequent finalization process within the stream of processing.

Descriptors: Cognitive control, Response inhibition, Acute stress, Cortisol, N2, P3, ERP

Executive functions are an umbrella term for different effortful top-down processes that enable us to adapt and optimize our handling of new or constantly changing conditions in everyday life. Thus, executive functions make it possible to focus on a goal, switch between activities, make future plans, inhibit an impulse, or resist temptation (Diamond, 2013; Gilbert & Burgess, 2008). Working memory, cognitive flexibility, and inhibition are considered to be the three core executive functions (Diamond, 2013).

Within the last decade, executive functions have gained attention in the field of stress research (Oei, Everaerd, Elzinga, van Well, & Bermond, 2006; Plessow, Fischer, Kirschbaum, & Goschke, 2011; Shields, Bonner, & Moons, 2015), which is not least due to the fact that a stressor or a stressful situation is an example par excellence of a circumstance requiring vigilance and adaptive strategies. Furthermore, executive functions or cognitive control processes, respectively, neuroanatomically rely on prefrontal cortex (PFC) functioning (Miller & Cohen, 2001), a structure particularly sensitive to effects of stress and the regulation of the stress response (Arnsten, 2009; McEwen & Morrison, 2013; Ulrich-Lai & Herman, 2009). Animal studies have shown that negative effects of stress on the PFC and its function operate via both the rapid stress response of the sympathetic nervous system as well as via the somewhat slower pathway of the hypothalamus-pituitary-adrenal (HPA) axis (Arnsten, 2009; Cerqueira, Almeida, & Sousa, 2008; Liston, 2006; McEwen & Morrison, 2013). Receptors of cortisol/corticosterone, the end product of the HPA axis, occur in high density in the PFC (de Kloet, Holsboer, & Joëls, 2005).

So far, human research of stress and executive functions has mainly focused on working memory (WM), revealing rather consistently impaired performance under high working load after stress (e.g., Ellenbogen, Schwartzman, Stewart, & Walker, 2002; Gärtner, Rohde-Liebenau, Grimm, & Bajbouj, 2014; Oei et al., 2006; Schoofs, Preuss, & Wolf, 2008, but see Schoofs, Pabst, Brand, & Wolf, 2013). Similarly, goal-directed behavior or cognitive control in the sense of task switching has been found to be negatively influenced by stress (Plessow et al., 2011; Plessow, Kiesel, & Kirschbaum, 2012; Steinhauser, Maier, & Hübner, 2007).

In contrast, inhibitory control in the proper sense of impulse control or response inhibition, defined by Aron, Robbins, and Poldrack (2004) as "the cognitive process required to cancel an intended movement" (p. 170), has hardly been investigated in the context of stress. This core executive function is of particular

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interest since it mutually supports WM and is, together with WM, necessary for cognitive flexibility (Diamond, 2013). As well, it is considered a prerequisite of self-regulation, and deficits in inhibition have been linked to different types of addiction, antisocial behavior, and several psychological illnesses (Bari & Robbins, 2013; Barkley, 2001). To our knowledge, only a very few studies have examined the effect of stress or acute elevated cortisol levels on inhibitory control (Oei, Tollenaar, Spinhoven, & Elzinga, 2009) or on response inhibition itself (Schlosser et al., 2013; Scholz et al., 2009; Schwabe, Hoffken, Tegenthoff, & Wolf, 2013; Wolf et al., 2001), yielding inconsistent results. For example, Scholz et al. (2009) reported impaired response inhibition in a go/no-go task after stress induction via the Trier Social Stress Test (TSST), whereas Schwabe et al. (2013) found faster reactions in a stop-signal task after the socially evaluated cold pressor test (SECPT). With regard to cortisol administration, Schlosser et al. (2013) revealed enhancing effects on response inhibition after administration of hydrocortisone in healthy control participants. Similarly, Oei et al. (2009) found improved inhibition of irrelevant information after hydrocortisone treatment. In contrast, Wolf et al. (2001) reported no effects of hydrocortisone injection on response inhibition in a Stroop color and word test.

These divergent findings might be caused at least in some part by methodological differences such as the specific type of stressor, the selected paradigm, or the investigated aspect of cognitive control. Furthermore, the above-cited studies concentrated on effects on a behavioral level (i.e., reaction times, accuracy), although there is profound knowledge and extensive literature on the neurophysiological basis of inhibitory control and response inhibition (e.g., ERPs; Bokura, Yamaguchi, & Kobayashi, 2001; Falkenstein, Hoormann, & Hohnsbein, 1999, fMRI: Huster, Enriquez-Geppert, Lavallee, Falkenstein, & Herrmann, 2013). ERPs allow a more distinct and specific examination of the underlying cognitive information processing, especially concerning the chronology of processing and the cortical resources included therein (Hillyard & Kutas, 1983). ERPs measured in go/no-go tasks have consistently revealed typical differences in no-go compared to go stimuli: stimulus-locked N2 and P3 ERP components are larger and more frontally distributed in no-go trials compared to go trials. Although the discussion about the functional significance of no-go N2 is not complete yet (Falkenstein, 2006), both ERPs are considered to be related with two aspects of inhibitory control (Liu et al., 2014). The so-called no-go/N2 is discussed to reflect inhibition itself or conflict monitoring, while the no-go/P3 is commonly regarded to reflect the finalization of the inhibition process (e.g., Donkers & van Boxtel, 2004; Jodo & Kayama, 1992; Kiefer, Marzinzik, Weisbrod, Scherg, & Spitzer, 1998; Kropotov, Ponomarev, Hollup, & Mueller, 2011; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; Schapkin, Falkenstein, Marks, & Griefahn, 2007). Besides these ERPs, the N2 and P3 of the difference wave (i.e., the N2d and P3d), obtained by subtracting go from no-go ERPs, are often used to specify the go/no-go effect. These brain potentials have been shown to be sensitive to task characteristics and demands (Benikos, Johnstone, & Roodenrys, 2013; Eimer, 1993; Gajewski & Falkenstein, 2013) as well as substances (Wit, Enggasser, & Richards, 2002) and symptoms of (sub)clinical populations that are linked to reduced inhibitory control (Fallgatter et al., 2004; Oddy & Barry, 2009). Therefore, they might be particularly useful to detect stress- and cortisol-induced alterations in response inhibition.

The present ERP study aimed to further investigate the influence of acute stress and the resulting increase in cortisol levels on behavioral measures of response inhibition and its neural correlates. Stress was induced via the socially evaluated cold pressor test (Schwabe, Haddad, & Schächinger, 2008), and several salivary cortisol measurements were taken in the course of the experiment for validation purposes. Since individual cortisol responses to a stressor might show considerable variation (Kirschbaum et al., 1995; Kudielka, Hellhammer, & Wüst, 2009; Schwabe et al., 2008) and we were particularly interested in the impact of stress-induced cortisol on response inhibition, we post hoc split participants of the stress group into cortisol high and low responders according to their stress-induced cortisol increase. Response inhibition was measured with a simple nonemotional go/no-go task before and after the stressor. In addition to behavioral data, stimulus-locked ERPs (N2, P3; N2d, P3d) for go and no-go stimuli as well as the difference wave no − go/go were analyzed, in order to test whether acute stress impairs or improves response inhibition, reflected in reduced or enhanced N2 in particular, as well as P3 amplitudes, respectively.

Method

Participants

Forty-one male students recruited from the University of Trier, Germany, participated in the study. All subjects were right-handed, nonsmokers, and physically and psychologically healthy. Exclusion criteria were any acute or chronic physical disease or mental disorder, including a history of the latter, use of medication, and nonnative German speaker. Additionally, students of psychology were excluded to ensure unbiased behavior during the experiment. Due to overall extreme (over ± 3 *SD*) or missing salivary cortisol values after the stress induction, two participants were removed from the analysis, leaving 39 participants for analysis with a mean age of 23.44 years (*SD* = 2.70, range 19–30 years) and a mean body mass index of 23.01 kg/m^2 (*SD* = 2.47). The experiment was conducted in accordance with the Declaration of Helsinki, and the research ethics committee of the University of Trier approved the study. All participants gave their written informed consent and were compensated with €35 or with course credit for participation.

Procedure

Prior to the experimental session, participants were invited to an interview, during which exclusion criteria were checked and information about the aim and procedure of the study (i.e., the investigation of the relationship between stress and different cognitive functions) was given. Crucially, participants were informed at full length that they might be exposed to a stress procedure comprising cold water, videotaping, and observation. Furthermore, eligible participants were required to refrain from physical exercise on the day prior, as well as alcohol, caffeinated drinks, and meals within 1 h prior to the experimental session.

The experiment itself was conducted between 12:00 noon and approximately 7:00 p.m., starting at 12:00, 2:30, and 5:00 p.m., when endogenous cortisol levels are relatively low (Schreiber et al., 2006). All participants were randomly assigned to the stress or control procedure and examined individually. In due consideration of the classification of the stressed participants into high and low cortisol responders, twice as many participants were stressed as underwent the control condition to ensure equal cell sizes. Participants were seated in a dimly lit, sound-attenuated room, 1 m from the monitor (19" Eizo FlexScan S2031W) and EEG and electrooculogram (EOG) recording devices were prepared. The

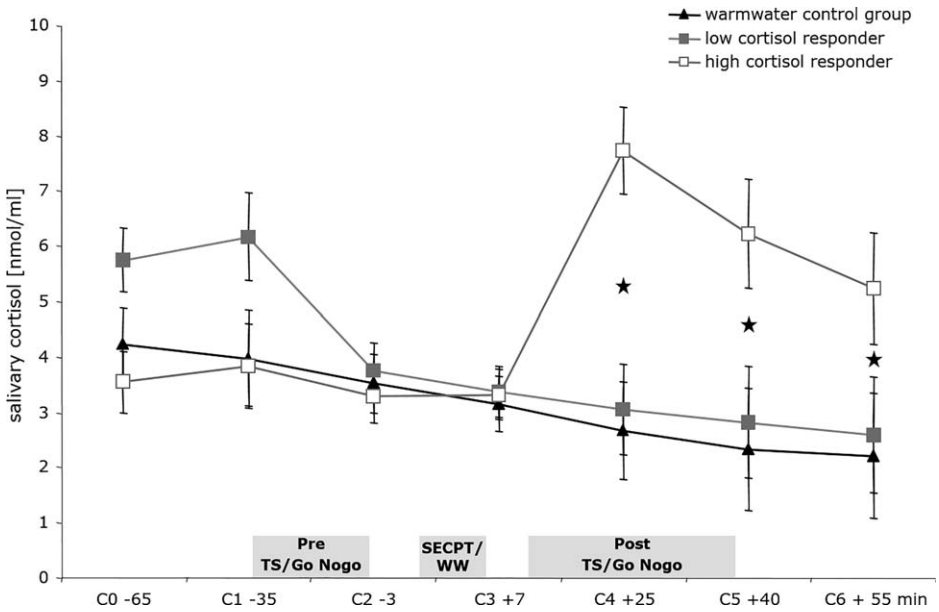

Figure 1. Mean levels of free salivary cortisol during the experimental session for high and low cortisol responders and the warm water control group. Error bars indicate standard errors of the mean. SECPT = socially evaluated cold pressor test; WW = warm water control procedure. The different orders of the task-switching (TS) and go/no-go task were balanced across participants. $*p < .05$.

participants received all instructions via the computer screen. Before and after the stress procedure (SECPT) or the warm water control procedure, participants performed a block of two cognitive tasks each, a go/no-go paradigm and a task-switching paradigm (for a description and results of the latter, see Fechtner, 2012). The order of these tasks was balanced across participants. During the course of the experiment, participants completed short state questionnaires several times and provided seven saliva samples for cortisol analysis. After removal of the physiological recording devices, participants were extensively debriefed and compensated for their participation. In total, the experiment lasted about 2 h.

Go/No-Go Task

Response inhibition was measured using a go/no-go paradigm. The letters *X* and *Y* served as go or no-go stimuli, respectively. The letters were presented in white front Courier Newsize 36 in the middle of a black screen. Each block started with a white fixation cross of 3,000 ms duration in the center of the screen. Next, the letter appeared for 400 ms, followed by a black screen until the participants responded. After the response, a white fixation cross was presented until the end of the trial. If no response followed, the black screen had a maximal duration of 1,100 ms, until the fixation cross was shown for 1,000 ms. The interstimulus interval was set to 2,500 ms. Two blocks with 180 trials with 90 trials go and 90 trials no-go each were presented in the experiment. Before the first block, a practice block with 16 trials was carried out. Stimuli were equiprobable (50% go, 50% no-go) and presented in random order with the restriction that three letters of a condition appeared in succession at most. The assignment of the letter to the go and no-go condition was counterbalanced across participants. Participants were instructed to press a button with the forefinger of their right hand as quickly as possible if the go stimulus appeared and to withhold the response to the no-go stimulus.

E-Prime presentation software (E-prime 2.0, Psychological Software Tools, Pittsburgh, PA) was used to present the stimuli and record the reaction times during the tasks.

Socially Evaluated Cold Pressor Test (SECPT)

Participants who were assigned to the stress condition were exposed to the SECPT (Schwabe et al., 2008). An unfamiliar female experimenter who acted neutrally and in a distant manner asked them to immerse their left hand up to the wrist into ice water (0–3°C) and to look at a camera throughout the whole procedure, as their facial expressions would be analyzed. Meanwhile, the experimenter watched them closely, took notes, and stopped the time. At the end of 3 min, they were asked to remove their hand. No further communication between experimenter and participants was permitted, and participants were unaware of the elapsed time. Participants in the nonstressful control condition underwent the same procedure with warm water (WW, 37–39°C) instead of ice water. No participant removed his hand from the ice water before the 3 min were up.

Salivary Cortisol Measurement

Saliva samples for cortisol analysis were obtained at seven assessment points over the course of the experiment (see Figure 1) using Salivette collection devices (Sarstedt, Nürnbrecht, Germany): before the start of the experiment (C0, about −65 min with reference to the beginning of the SECPT/WW), before the first block of both cognitive tasks (C1, about −35 min), before the SECPT/WW (C2, −3 min), after the SECPT/WW (C3, +7 min), after the second block of the first cognitive task (C4, +25 min), after the second block of the second cognitive task (C5, +40 min), and at the end of the experiment (C6, +55 min). Sampling instructions were given via computer, and Salivettes were positioned on the table in front of the participants. Immediately after the experiment, samples were frozen for biochemical analysis with a time-resolved immunoassay with fluorescence detection as described in detail

Table 1. *Characteristics and Behavioral Data in the Go/No-Go Task of Participants in the Three SECPT Groups*

	Overall ($N = 39$) *M* (*SD*)	Warm water control group ($n = 12$) *M* (*SD*)	Low cortisol responders ($n = 13$) *M* (*SD*)	High cortisol responders ($n = 14$) *M* (*SD*)
Age	23.44 (2.70)	22.75 (1.96)	22.92 (1.98)	24.50 (3.55)
BMI	23.01 (2.47)	23.12 (2.15)	22.66 (2.83)	23.24 (2.52)
Cortisol increase due to the SECPT/WW (C4 − C2)	1.14 (3.51)	-.85 (1.20)	-.71 (1.02)	4.43 (3.80)
No. of go errors pre-SECPT/WW	.59 (1.60)	.50 (1.17)	1.15 (2.48)	.14 (.36)
No. of go errors post-SECPT/WW	.46 (.85)	.25 (.45)	.38 (.65)	.71 (1.20)
No. of no-go errors pre-SECPT/WW	3.21 (3.74)	2.50 (1.88)	3.15 (3.05)	3.86 (5.32)
No. of no-go errors post-SECPT/WW	2.69 (2.77)	1.83 (1.99)	3.54 (3.31)	2.64 (2.76)
Reaction times pre-SECPT/WW [ms]	437.17 (35.57)	441.17 (31.33)	442.23 (45.87)	429.04 (29.51)
Reaction times post-SECPT/WW [ms]	432.78 (31.37)	430.79 (31.91)	437.69 (34.40)	429.86 (29.77)

Note. *M* = mean; *SD* = standard deviation; BMI = body mass index; SECPT = socially evaluated cold pressor test; WW = warm water control procedure.

elsewhere (Dressendörfer, Kirschbaum, Rohde, Stahl, & Strasburger, 1992). Intra- and interassay variability were less than 10% and 12%, respectively.

EEG Recording and Quantification

The EEG was recorded from 32 Ag/AgCl electrode sites including the mastoids according to the 10-10 electrode reference system (Chatrian, Lettich, & Nelson, 1988) with the Easy-Cap electrode system (Falk Minow Services, Munich). All sites were referenced to FCz. A bipolar horizontal EOG was recorded from the epicanthus of each eye, and a bipolar vertical EOG was recorded from supra- and infraorbital positions of the left eye. A BrainAmp amplifier (input impedance: 10 MΩ; Brain Products, GmbH) in AC mode was used to record the EEG and EOG at 1000 Hz using a pass-band set to 0.016 to 499 Hz (−12 dB/octave roll-off). All impedances of the EEG electrodes were maintained below 5 kΩ. Data were stored to hard disk for later analysis using BrainVision Analyzer 2 (Brain Products, Munich, Germany).

The EEG was rereferenced offline to linked mastoids. The data were resampled at 200 Hz and low-pass filtered using a digital filter with high cutoff of 12 Hz, 24 dB/oct. Artifacts due to eye movements were corrected semiautomatically via the algorithm developed by Gratton, Coles, and Donchin, 1983. EEG of trials with accurate responses were epoched offline into periods of 1,200 ms, starting 200 ms prior to go and no-go stimuli onset, respectively. A baseline correction was performed using the first 200-ms interval as reference. Trials with nonphysiological artifacts were excluded from analysis via semiautomatic artifact rejection. Separate averages were computed for each electrode and individual for go and no-go trials before (pre-) and after (post-) the SECPT or WW control condition, respectively. Subsequently, difference waveforms (no-go − go) were computed for each block. Using the grand average across participants and the topography to guide window selection, mean amplitudes (μV) of the stimulus-locked N2 and P3 components were selected for statistical analysis within the time intervals 200–270 ms (N2) and 300–370 ms (P3) at frontal (F3, Fz, F4) and frontocentral (FC3, FCz, FC4) electrodes, where no-go − N2 and no-go − P3 showed maximum amplitudes. Since the visual inspection of the grand average across participants and the topography of the difference wave no-go − go revealed a slightly earlier peak of N2 and P3 of the difference wave no-go − go (N2d, P3d), time windows for analyzed mean amplitudes were adjusted accordingly: 190–260 ms (N2d), 315–385 ms (P3d).

Statistical Analyses

The data were edited with Microsoft Excel 2003 and analyzed with IBM SPSS Statistics 20.

Stress manipulation. Based on their cortisol reaction in response to the SECPT, participants of the stress condition were post hoc allocated to a high or low cortisol responder group. The stress-induced cortisol response of each individual was computed by calculating the difference of the cortisol levels C4 (+25 min after SECPT when, according to Dickerson & Kemeny, 2004, the peak cortisol response is to occur) and C2 (−3 min before SECPT). A median split (1.37 nmol/l) of this cortisol change divided the participants of the stress condition (SECPT, $n = 27$) into high cortisol responders ($n = 14$) and low cortisol responders ($n = 13$). A Group (high cortisol responders, low cortisol responders, warm water control group) × Time of cortisol measurement (repeated measurement, C0–C6) analysis of variance (ANOVA) was conducted to check whether the stress induction was successful and how long the cortisol increase lasted. Additionally, the difference of cortisol at time points C4 (+25 min after SECPT/WW) and C2 (−3 min before SECPT/WW) was submitted to a group (high cortisol responders, low cortisol responders, warm water control group) ANOVA to test significance of the stress groups' categorization.[1]

Behavioral data. The numbers of errors in go and no-go trials were summed for each individual. Participants of all groups showed a very high accuracy before and after the SECPT or control procedure, with only slightly more errors in go trials for high cortisol responders after the SECPT (see Table 1). Due to this ceiling effect of task performance (Cramer & Howitt, 2004), no further analyses were conducted. With regard of the reaction times (RTs in milliseconds), only go trials with correct responses were analyzed. Outliers were removed on an individual basis by visual inspection of the frequency distribution of the RTs. Median RTs were submitted to a Group (high cortisol responders, low cortisol responders, warm water control group) × Block (repeated measurement, before SECPT/WW (pre-) versus after SECPT/WW (post-)) ANOVA.

Electrophysiological data. Mean N2 and P3 amplitudes were submitted to separate Group (low cortisol responders, high cortisol

1. These analyses were calculated on the basis of 38 participants, as one participant of the warm water control group had a missing value at C2. All other analyses were based on the whole sample of $N = 39$.

responders, warm water control group) × Go/No-Go (go vs. no-go stimuli) × Block (pre- vs. post-SECPT/WW) × Caudality (frontal vs. frontocentral) × Lateralization (left vs. midline vs. right) ANOVAs, with repeated measurement on the last four factors. Similarly, mean amplitudes of N2d and P3d were subjected to separate Group (low cortisol responders, high cortisol responders, warm water control group) × Block (pre- vs. post-SECPT/WW) × Caudality (frontal vs. frontocentral) × Lateralization (left vs. midline vs. right) ANOVAs with repeated measurement on the last three factors.

Effect sizes of significant results are reported as proportion of explained variance (η^2, partial eta squared). Where appropriate, Dunn's multiple comparison tests were used as post hoc tests (Kirk, 1995). For each Dunn's test, the critical difference ψ_{Dunn} ($\alpha = .05$) and number of comparisons C are specified. In case the assumption of sphericity was violated, the degrees of freedom for all ANOVAs were corrected by Huynh-Feldt epsilon (HF-ε, Huynh & Feldt, 1976). The statistical significance level was set to $\alpha = .05$ (two-tailed).

Power values for the relevant statistical analyses are specified according to Hager (2004). The hypothesis comprised a three-way interaction of SECPT Group × Go/No-Go × Block for the N2 and P3 amplitudes. With a sample size of 39 participants, a given significance level of .05, and an assumed population correlation, supported by our empirical data, of at least $\rho = .60$ for respective repeated measurements, this three-way interaction can detect a small to medium effect of $\Omega^2 \geq .05$ with a probability of 1 β (statistical power) > .98. For RTs and N2d as well as P3d, the hypothesis comprised a two-way interaction of SECPT Group × Block, which can detect a small-to-medium effect of $\Omega^2 \geq .05$ with a probability of 1 β (statistical power) > .80. Should these interactions be further qualified by caudality and/or lateralization in case of ERPs, the power would increase due to increasing numbers of observations by including within-subject factors.

Results

Stress Induction

High cortisol responders showed, as expected, a clear cortisol increase in response to the stressor. In contrast, low cortisol responders and participants of the warm water control group did not differ significantly from each other, and both showed even a slight decrease in cortisol levels (see Table 1; $F(2,35) = 19.54$, $p < .001$, $\eta^2 = .53$; $\psi_{Dunn} = 2.48$, C = 3). As well, as shown in Figure 1, high cortisol responders had higher cortisol levels after the SECPT from time point C4 until C6 compared to low cortisol responders and to the warm water control group, while no differences were found between time points C0 and C3, $F(12,210) = 6.55$, $p < .001$, HF-$\varepsilon = .40$, $\eta^2 = .27$; $\psi_{Dunn} = 2.61$, C = 21.

Impact of Stress on Response Inhibition: Reaction Times

The three groups did not differ in their reaction times before or after the SECPT/WW control procedure, respectively (see Table 1, all $Fs < 2.63$, all $ps > .10$).

ERP Results: Go/No-Go Manipulation Check

The N2 and P3 amplitude showed both the expected go/no-go pattern with more negative N2 amplitudes and more positive P3 amplitudes for no-go trials compared to go trials at analyzed frontal and frontocentral electrodes (see Figure 2A, go/no-go main effect N2: $F(1,36) = 36.97$, $p < .001$, $\eta^2 = .51$; P3: $F(1,36) = 18.37$, $p < .001$, $\eta^2 = .34$). This go/no-go effect was maximal at frontal and frontocentral midline to right electrode sites for the N2 and maximal at frontal and frontocentral left to midline electrodes for the P3 (Go/No-Go × Caudality × Lateralization N2: $F(2,72) = 7.60$, $p < .01$, $\eta^2 = .17$, $\psi_{Dunn} = .29$, C = 6; P3: $F(2,72) = 35.85$, $p < .001$, HF-$\varepsilon = .97$, $\eta^2 = .50$, $\psi_{Dunn} = .29$, C = 6). The analyses of the topographies of the difference wave components N2d and P3d confirmed this pattern (see Figure 2B,C; Caudality × Lateralization, N2d: $F(2,72) = 8.30$, $p < .001$, $\eta^2 = .19$, $\psi_{Dunn} = .39$, C = 9; P3d: $F(2,72) = 37.66$, $p < .001$, HF-$\varepsilon = .94$, $\eta^2 = .51$, $\psi_{Dunn} = .39$, C = 9).

ERP Results: Impact of Stress

Stress significantly influenced the magnitude of N2 and P3 amplitude (N2: Group × Go/No-Go × Block × Caudality, $F(2,36) = 3.45$, $p < .05$, $\eta^2 = .16$; P3: Group × Go/No-Go × Block × Caudality × Lateralization, $F(4,72) = 2.93$, $p < .05$, HF-$\varepsilon = .85$, $\eta^2 = .13$). Comparing no-go versus go N2 amplitudes within each SECPT group, the post hoc test showed that participants of all three groups showed the expected no-go < go N2 effect at frontal and frontocentral leads before and after the SECPT or warm water control procedure, respectively ($\psi_{Dunn} = .38$, C = 12, Figure 3A). Similarly, the no-go > go P3 effect was found at relevant electrode sites (i.e., left to midline frontal and frontocentral) in all three groups before and after the SECPT/WW procedure ($\psi_{Dunn} = .59$, C = 36, Figure 3A). However, further visual inspection of the go/no-go N2 and P3 effects, illustrated by the N2d and P3d of the difference wave no/go − go, suggested a different magnitude in amplitude and topography in the three groups comparing pre- and post-SECPT/WW procedure measurement (see Figure 3B). Thus, to further elucidate the impact of stress on the go/no-go effect, the N2d and P3d of the difference wave no-go − go were analyzed with a Group × Block × Caudality × Lateralization ANOVA.

In line with the results of the N2 and P3, stress significantly influenced the magnitude of N2d and P3d; namely, the analysis of the N2d revealed a significant Group × Block × Caudality interaction, $F(2,36) = 4.06$, $p < .05$, $\eta^2 = .18$. As depicted in Figure 3B, 4, the warm water control group showed a reduced N2d amplitude at frontocentral leads after the warm water control procedure relative to beforehand. In contrast, high and low cortisol responders showed both enlarged (i.e., more negative) N2d amplitudes after the SECPT relative to beforehand. While, according to post hoc tests ($\psi_{Dunn} = .50$, C = 12), these pre–post changes failed significance, the comparison of this pre–post difference between the three groups showed that the Nd2 amplitude significantly increased at frontocentral electrodes in high and low cortisol responders in comparison to the warm water control group (see Figure 4).

With regard to the P3d, the ANOVA revealed a significant Caudality × Lateralization × Block × Group interaction, $F(4,72) = 3.69$; $p < .01$; $\eta^2 = .17$; Figure 3. Comparing the P3d amplitudes pre- versus post-SECPT/WW procedure, the post hoc tests showed a significant reduction for the warm water group at FCz, but a significant increase at FC4. By the same manner, the stress group (i.e., low and high cortisol responders) showed a significant reduction of the P3d amplitude after the SECPT relative to beforehand. However, while this reduction was limited to FCz for low cortisol responders, it was found at all frontocentral electrodes for high cortisol responders (see Figure 3, 5). This pre–post P3d reduction was

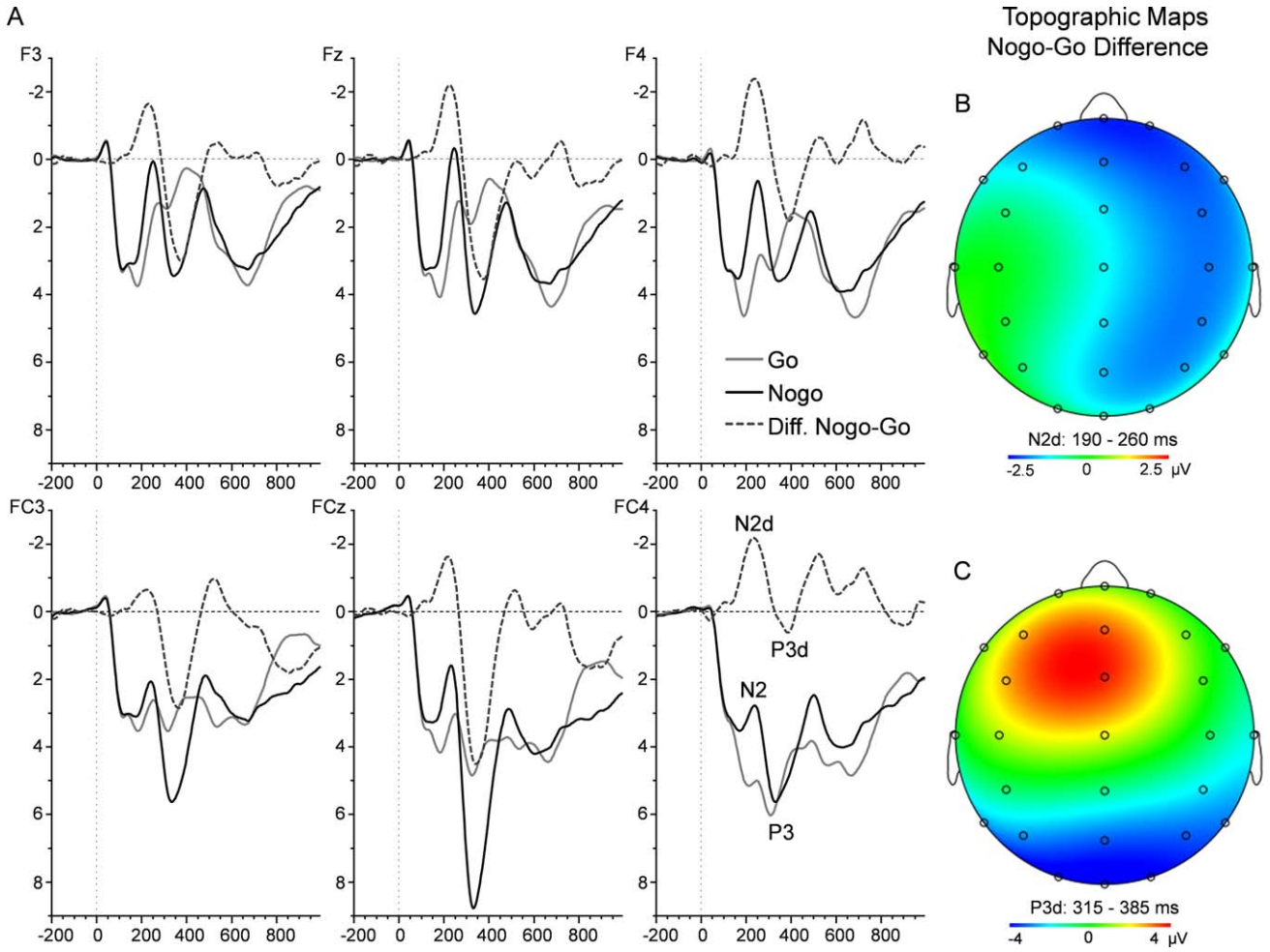

Figure 2. A: Grand-averaged ERPs at frontal (F3, Fz, F4) and frontocentral (FC3, FCz, FC4) electrodes for go trials (light lines), no-go trials (dark lines), and no-go − go difference waves (dashed lines) averaged over block (pre-, post-) and group (high, low cortisol responders, warm water control group). B, C:. Topographic maps of the no-go − go difference within the time window of the Nd2 (B) and P3d (C) amplitude. No-go relative to go stimuli elicited enhanced N2 and P3 amplitudes at frontal to frontocentral sites.

strongest in high cortisol responders compared to the two other groups, which did not differ at FCz but at FC4, due to the P3d increase in the warm water control group ($\psi_{Dunn} = .59$; $C = 36$, see Figure 5).

Discussion

The present study investigated the influence of a psychophysiological stressor, and the resulting cortisol increase, on behavioral and electrocortical measurements of cognitive control, specifically response inhibition.

The stress induction via the SECPT was successful. High cortisol responders showed a considerable increase of free cortisol in response to the stressor, similar to that found in other studies using this stressor (e.g., Lass-Hennemann et al., 2011; Schwabe & Wolf, 2009).

Impact of Stress on Response Inhibition Performance and Electrophysiological Correlates

Acute stress had no influence on performance in the go/no-go task. In general, accuracy was very high, and reaction times remained constant throughout both blocks for all participants. In contrast, stress altered electrophysiological correlates of response inhibition. While all three groups showed the expected go/no-go effects on the analyzed components N2 and P3, acute stress altered the magnitude of these effects as a function of stress-induced HPA axis activation, as shown by the N2d and P3d of the difference wave.

N2/N2d. Frequently, enlarged N2/N2d amplitudes in no-go compared to go trials are assumed to reflect a top-down response inhibition process prior to the motor response, essential for successful inhibition (Falkenstein et al., 1999; Falkenstein, Hoormann, & Hohnsbein, 2002; Fallgatter & Strik, 1999) and/or conflict monitoring (Falkenstein, 2006; Nieuwenhuis et al., 2003). Since all groups showed the no-go < go N2 effect before and after the SECPT/warm water control condition, these processes were active in the stress group (i.e., low and high cortisol responders) as well as in the control group. Moreover, stress did not impair these processes, but analyses of the N2d showed that stress led to enhanced frontocentral N2d amplitudes in high and low cortisol responders relative to the warm water control group. Prior studies found the N2d, the indicator of the no-go < go N2 effect, to be enlarged with practice and successful response inhibition (Falkenstein et al., 1999; Schapkin et al., 2007). Accordingly, the present results suggest that shortly after acute stress this early stage of the response inhibition

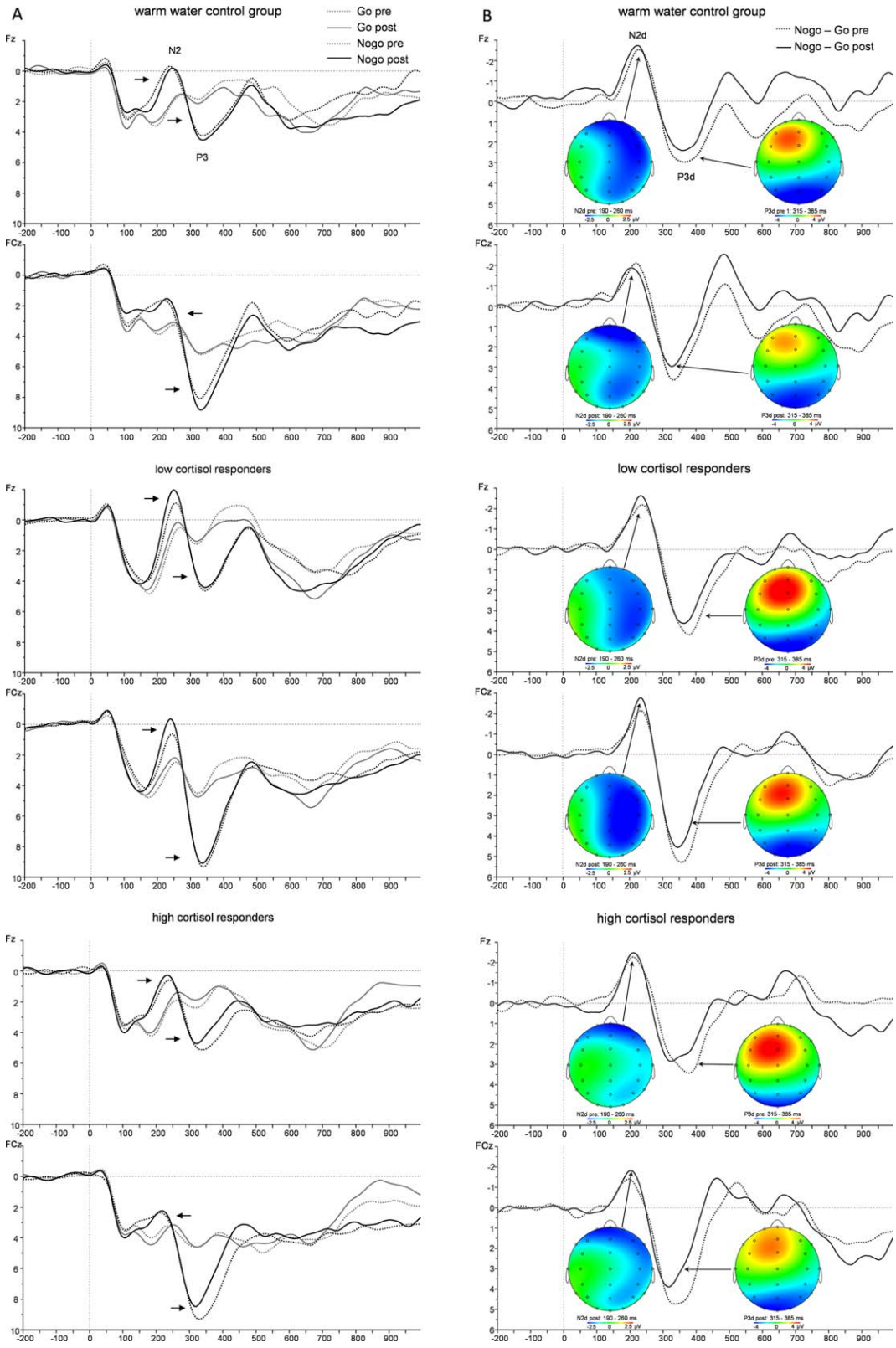

Figure 3. A: Grand-averaged ERP waveforms at Fz and FCz for go and no-go trials for the three groups (warm water control group, low cortisol responders, high cortisol responders) before and after the socially evaluated cold pressor test (SECPT)/warm water control procedure (WW). All three groups showed the expected go/no-go N2 and P3 effects (marked by arrows). B: No-go − go difference waves at Fz and FCz as well as topographical maps for time window of the N2d (190–260 ms) and the P3d amplitudes (315–385 ms) for each of the three groups (warm water control group, low cortisol responders, high cortisol responders) before the SECPT/WW (pre-) and after (post-). High and low cortisol responders showed increased N2d but reduced P3d amplitudes after the SECPT.

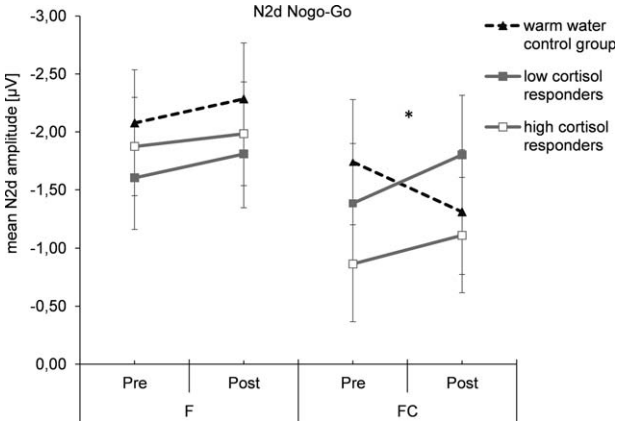

Figure 4. Mean N2d amplitudes of the difference wave no-go − go for the three groups (warm water control group, low cortisol responders, high cortisol responders) before and after the SECPT/warm water control procedure (pre-/post-) at frontal (F) and frontocentral (FC) electrodes. SECPT = socially evaluated cold pressor test. Error bars indicate standard errors of the mean. *Significant differences between the three groups for the change of the N2d amplitude from pre- to post-; $p < .05$. Warm water control group showed a reduction of the N2d at frontocentral electrodes after the warm water control procedure, while the low and high cortisol responders showed an increase from pre- to post-SECPT.

process is strengthened, and stressed participants showed increased premotor response inhibition and conflict monitoring, irrespective of the stress-induced cortisol increase.

P3/P3d. Similar to the N2, the no-go P3 is thought to reflect response inhibition, albeit functionally disconnected at later stages of this process (Falkenstein et al., 2002). Smith, Johnstone, and Barry (2008) found supporting evidence that the no-go P3 is associated with motor response inhibition, and further research suggests that this component might reflect the evaluation or finalization of the inhibitory process (Band & van Boxtel, 1999; Bruin, Wijers, & van Staveren, 2001). Again, all groups showed the expected no-go > go P3 effect before and after the stress or control procedure, indicating that this aspect of response inhibition was active after the stressor as well. However, stress altered this stage of information processing as a function of stress-induced cortisol increase, leading to reduced P3d amplitudes in high cortisol responders. Accordingly, only those participants who responded to the stressor with a significant increase in cortisol showed impaired motor inhibition and finalization of the inhibitory process, while no alteration was found in participants without an HPA activation after stress (i.e., low cortisol responders). In sum, the present electrophysiological results suggest that stress improved early stages of response inhibition processing, reflected by enhanced N2d amplitudes, but concurrently impaired later stages of the response inhibition process in participants with stress-induced rise in cortisol, indicated by reduced P3d amplitudes.

The fact that both components were differently affected by stress and cortisol may seem unexpected considering the fact that N2 and P3 were initially interpreted as a single complex N2/P3 (Simson, Vaughan, & Ritter, 1977). However, there is profound evidence that both components are modulated by different neurobiological pathways (Bokura et al., 2001; Huster, Westerhausen, Pantev, & Konrad, 2010). Findings of EEG inverse modeling and simultaneous EEG-fMRI quite consistently indicate the anterior and medial cingulate cortex as the major neural generator of the N2 and presupplementary motor areas, the insula, the posterior midcingulate, and posterior cingulate cortices, as well as the inferior frontal gyrus (IFG) as part of the broader network underlying the no-go P3 generation (Baumeister et al., 2014; Huster et al., 2013; Nieuwenhuis et al., 2003; Pires, Leitão, Guerrini, & Simões, 2014). The association of the no-go P3 amplitude with the IFG gained further support by a recent study of Cunillera and colleagues, showing a reduction of this component induced by stimulation the right IFG with tDCS (Cunillera, Brignani, Cucurell, Fuentemilla, & Miniussi, 2015). This may offer an explanation of our findings, since different cortical structures are activated or deactivated in response to stress, which depend (among other systems) on the stress-induced rise of cortisol (Dedovic, Duchesne, Andrews, Engert, & Pruessner, 2009; Kern et al., 2008; Kogler et al., 2015). Supporting our findings of altered no-go/P3 amplitudes exclusively in high cortisol responders, Taylor et al. (2008) and Wang et al. (2005) found the activity of the right IFG to be associated with cortisol reactivity to an acute stressor, while the association of the anterior and medial cingulate cortices as suggested neural generators of the N2 with stress alone and specifically stress-induced cortisol increase is rather inconclusive (Dedovic, Duchesne et al., 2009; Dedovic, Rexroth et al., 2009).

So far, ERP studies in the context of stress and executive function are rare and, to our knowledge, there is no such study investigating response inhibition as a core executive function. Studies investigating related topics support the present findings insofar as they showed acute stress to affect selectively different stages of the stream of processing with enhanced N2 amplitude in an oddball task in social drinkers, during action cascading processes, and a change detection task (Ceballos, Giuliano, Wicha, & Graham, 2012; Sänger, Bechtold, Schoofs, Blaszkewicz, & Wascher, 2014; Yildiz, Wolf, & Beste, 2014). Similarly, reduced no-go/P3 amplitudes are associated with aggression (Verona & Bresin, 2015). Stress (and, in particular, cortisol) is assumed to promote aggressive behavior (Böhnke, Bertsch, Kruk, Richter, & Naumann, 2010;

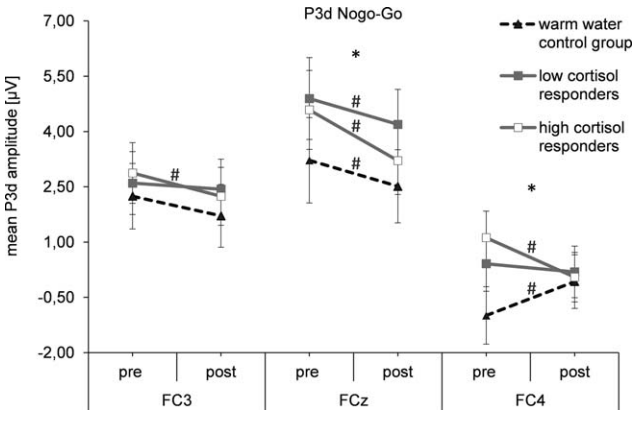

Figure 5. Mean P3d amplitudes of the difference wave no-go − go for the three groups (warm water control group, low cortisol responders, high cortisol responders) before and after the SECPT/warm water control procedure (pre-/post-) at FC3, FCz, and FC4. SECPT = socially evaluated cold pressor test. Error bars indicate standard errors of the mean. #Significant change from pre- to post-SECPT/WW procedures. *Significant differences between the three groups for the change of the P3d amplitude from pre- to post-; $p < .05$. High cortisol responders showed the strongest reduction of the P3d at FCz and FC4 relative to low cortisol responders and the warm water control group.

Geniole, Carre, & McCormick, 2011). However, stress effects (at least at early stages of information processing) might be restricted to frontal-frontocentral sites, as the study by Sänger et al. (2014) indicates, reporting enhanced frontal N2 amplitudes but reduced posterior N1pc and N2pc amplitudes after acute stress.

Behavior. While the nonsignificant stress effects on response inhibition accuracy are in line with earlier studies revealing no significant influence of stress on error rates, the absence of stress effects on reaction times contradicts previous findings, which revealed detrimental as well as beneficial effects of acute stress (Scholz et al., 2009; Schwabe et al., 2013). As outlined by Schlosser et al. (2013) and others, different methodical reasons (e.g., extent of HPA axis activation) may account for these inconsistent findings and the nonsignificant stress effect on response inhibition performance in the present study. In respect of stress effects on WM, for example, working memory load has been identified as a crucial moderator in recent meta-analyses by Shields, Sazma, and Yonelinas (2016). For instance, Oei et al. (2006) found impairing effects after the TSST only in the case of high workload in a Sternberg paradigm. Similarly, Gärtner et al. (2014) reported impaired performance after acute stress induction via negative film clips in the three-back condition, but not in the easier two-back condition of an *n*-back working memory task. In light of the relevance of high workload to reveal impairing effects of stress on WM, task characteristics in particular may have been inappropriate to reproduce stress effects on performance in the present study (i.e., ceiling effect of task performance accuracy). Accordingly, the individual accuracy rate of 50% in the stop-signal task used in the study of Schwabe et al. (2013) might have contributed to the significant stress effect on stop-signal reaction times.

In summary, the present findings suggest that stress and cortisol seem to differentially fortify or weaken specific subprocesses of inhibitory control. Thus, stress does not inevitably involve a depletion of cognitive resources, but rather leads to a reallocation of those with enhanced premotor response inhibition and conflict monitoring at the expense of motor inhibition and process finalization in the case of high cortisol levels. In light of previous studies on stress impacts on prefrontal cortex-based executive functions (e.g., Plessow et al., 2011; Schoofs et al., 2008), we would expect impaired response inhibition after acute stress. Hence, alterations observed in the N2d and P3d amplitudes might reflect compensatory mechanisms to overcome the negative impact of acute stress. Compensatory mechanisms and their neural correlates have been mainly discussed in research of aging and its concomitant cognitive deficits (Kropotov, Ponomarev, Tereshchenko, Muller, & Jancke, 2016; Riis et al., 2008). Here, authors predominately interpret reduced ERP components in cognitive tasks (go/no-go, novelty oddball tasks) as reflection of cognitive deficits in the sense of fewer available resources, while enhanced amplitudes are considered to indicate additional engagement as well as recruitment of cognitive resources (Daffner et al., 2011; Kropotov et al., 2016; Riis et al., 2008). Speaking in terms of the cognitive-energetical framework[2] by Hockey (1997), this would imply that the impact of stress is compensated with fortified early stages of response inhibition processing (i.e., enhanced N2d) but imply so-called latent performance decrements (Hockey, 1997, p. 82), which might be reflected in cortisol-dependently altered electrophysiological correlates of motor response inhibition (i.e., the reduced P3d). Consequently, the opposed impact on the different subprocesses annul each other, resulting in unimpaired behavior. However, under more demanding or unfavorable circumstances, these compensatory strategies might not be sufficient, as studies on WM or with a stronger stressor indicate (Oei et al., 2006; Scholz et al., 2009).

On the other hand, reduced frontal P3 amplitudes have been interpreted as more efficient processing, namely, spending fewer cognitive resources in a given task (Riis et al., 2008). Thus, it is possible that stress and cortisol may eventually improve response inhibition, as demonstrated by Schwabe et al. (2013). Accordingly, reduced P3d amplitudes in high cortisol responders might indicate a less resource-demanding, more efficient premotor inhibition process. From an evolutionary point of view, enhanced response inhibition and vigilance in response to stress constitute an adaptive behavior by strengthening the adaptive reaction and suppressing the inappropriate action tendencies. The beneficial effect of acute rise in cortisol on response inhibition is supported by a meta-analysis by Shields et al. (2015), reporting enhanced response inhibition due to rapid nongenomic effects of exogenous applied cortisol, highlighting the relevance of timing between the stressor and the requirement to exercise inhibitory control (Hermans, Henckens, Marloes, Joels, & Fernandez, 2014). Similarly, in a recent meta-analysis on acute stress effects on core executive functions, Shields, Sazma, & Yonelinas (2016) concluded that acute stress enhances response inhibition. Assuming that acute stress strengthens response inhibition suggests that stress does not only differentially affect subprocesses of this executive function, but that stress (i.e., cortisol or other physiological aspects of the stress response) might have a different impact on the distinct subregions of the PFC on which the different executive functions each rely (Schlosser et al., 2013, p. 444; Schwabe et al., 2013, p. 2325). This notion is endorsed by finding that different regions of the PFC show different activity patterns in the processing and regulation of a stressor (Dedovic, Duchesne et al., 2009).

The present study was the first to investigate effects of an acute stressor on response inhibition using electrophysiological measurements. Some limitations should be addressed. First, the post hoc classification of the SECPT group into low and high cortisol responders provides the opportunity to disentangle the specific associations of endogenous stress-induced cortisol increase from further impacts of the stress test. However, concomitantly, this quasiexperimental approach does not allow causal conclusions and may cause confounding, for instance, with personality or physiological/biological traits. Moreover, the study only included healthy young men; for this reason, the present results cannot be readily generalized to women. There are few studies on acute stress and response inhibition or other core executive functions comprising both male and female participants, some of which did not report sex differences (e.g., Schwabe et al., 2013; Plessow et al., 2012), while others found women and men to be differently affected (e.g., Schoofs et al., 2013; Shields, Trainor, Lam, & Yonelinas, 2016). As well, sex differences in the stress response are known (e.g., Kajantie & Phillips, 2006), and there is at least some evidence for sex differences in response inhibition and its neural correlates (e.g., Omura & Kusumoto, 2015). Thus, further research is needed to clarify possible sex effects in this context.

Conclusion

The present study revealed effects of acute stress on the neural correlates of response inhibition, while behavioral performance

2. This framework states that regulatory processes required for coping with stress allocate resources at the expense of performance, but that the cognitive system is able to adapt to these restricted resources and still maintain performance by adopting a less capacity-demanding performance-protection strategy.

remained unimpaired. Stress led to a reallocation of cognitive resources to specific subprocesses of response inhibition as a function of induced HPA axis activation. Stress itself led to augmented N2d amplitudes, indicating enhanced premotor response inhibition and conflict monitoring, irrespective of the amount of HPA axis activation. In contrast, a stress-induced rise in cortisol was associated with a reduced P3d, indicating an impaired evaluation and finalization of the inhibitory process. Whether these alterations within the stream of processing reflect compensatory mechanisms to overcome a stress-induced impairment on a behavioral level or whether stress eventually improves behavioral inhibition needs further research. Nevertheless, the present study provides insight into the effects of stress and cortisol on inhibitory control, underlining the advantage of electrocortical measurements to capture a comprehensive and elaborate picture of different aspects of the underlying cognitive processes.

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