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Good to be stressed? Improved response inhibition and error processing after acute stress in young and older men

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ABSTRACT

While aging and stress are both known to affect cognitive functions, little is known on whether and how age modulates stress effects on executive functions and their neural correlates. The current study investigated the effect of acute stress on response inhibition and error processing and their underlying cortical processes in younger and older healthy men, using EEG. Forty-nine participants (30 young) were stressed with the Trier Social Stress Test (16 young, 9 older) or underwent a friendly control procedure (14 young, 10 older) and subsequently performed a Go/No-Go task with two levels of task difficulty while performance (reaction time, error rate), stimulus-locked (N2, P3) and response-locked (Ne, Pe) ERPs were measured. Previous results on age-related cognitive deficits were replicated, with slower responses and reduced and delayed N2 and P3 components, as well as reduced Ne and Pe components in older participants. Independent of age, acute stress improved response inhibition, reflected in higher accuracy for compatible trials and enhanced inhibition-related components (N2, P3 and N2d, P3d of the difference waves No-Go minus Go), and improved error processing, reflected in enhanced error-related components (Ne, Pe and Ne_d, Pe_d of the difference waves error minus correct trial). Our findings indicate that acute stress leads to a reallocation of cognitive resources, strengthening inhibition and error processing in young and older healthy men to a similar degree. Neural generators of the analyzed ERPs are mainly part of the salience network, which is upregulated immediately after stress. This offers an explanation as to why response inhibition, in contrast to other executive functions, improves after acute stress.

1. Introduction

Research conducted in laboratory animals has repeatedly demonstrated that acute stress impairs PFC functioning via rapid effects of catecholamines and/or glucocorticoids (Arnsten, 2009). Human functional neuroimaging work has revealed a boosted vigilance network in the immediate aftermath of acute stress that went along with reduced executive (PFC-mediated) control (Hermans et al., 2014), suggesting that during stress, top-down control is reduced and stimulus-driven behavior takes over (Hermans et al., 2014; Arnsten, 2009; Schwabe and Wolf, 2013).

With respect to its impact on core executive functions linked to the PFC, stress typically impairs performance in tests for task shifting, goal-directed behavior or working memory (Shields et al., 2016a; Oei et al., 2006; Plessow et al., 2012; Schoofs et al., 2009). However, there seems to be one exception to the rule. A recent meta-analysis by Shields et al. (2016a) detected an overall beneficial effect of stress on response inhibition as assessed by stop signal or Go/No-Go tasks. This conclusion, however, was based on only six studies showing a substantial between-study variance. In part, the somewhat unclear empirical picture can be explained by the different behavioral tasks employed, some of which seem to be too easy and as such not sensitive enough to detect beneficial effects of stress on response inhibition. Enhanced workload and task difficulty have been shown to be relevant moderating factors in stress effects on executive functions, with larger stress effects under high cognitive load (Shields et al., 2016a; Gärtner et al., 2014). Therefore, the use of tasks with a sufficient level of difficulty in combination with sensitive electrophysiological (EEG/ERP) measurements with a high temporal resolution could be a promising approach in future research in this area. ERPs measured in Go/No-Go tasks have

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consistently revealed stimulus-locked N2 and P3 ERP components to be larger and more frontally distributed in No-Go compared to Go trials. In addition, the N2 and P3 of the difference wave (i.e., No-Go minus Go, N2d and P3d), are often used to specify the Go/No-Go effect. These ERPs relate to two different aspects of inhibitory control, namely pre-motor response inhibition or conflict monitoring (N2) and finalization of the inhibition process (P3) (Kropotov et al., 2011; Nieuwenhuis et al., 2003; Jodo and Kayama, 1992; Gajewski and Falkenstein, 2013). The neural circuitry of response inhibition comprises, amongst others, the dorsomedial and ventrolateral PFC as well as the insula, pre-supplementary motor areas and the anterior cingulate cortex (Ridderinkhof et al., 2004; Huster et al., 2010; Baumeister et al., 2014), the latter of which is also majorly involved in error processing (Ridderinkhof et al., 2004; Taylor et al., 2007).

In a recent study, acute stress was shown to alter N2d and P3d in a Go/No-Go task in young participants (Dierolf et al., 2017). Healthy young men underwent either the socially-evaluated cold pressor test or a control condition with warm water, before and after which they performed an equiprobable Go/No-Go task. While acute stress did not alter accuracy and reaction times, stimulus-locked N2d amplitudes were enhanced after the stressor. In contrast, the P3d amplitudes were reduced after the stress procedure, albeit only in participants with a considerable stress-induced increase in cortisol.

A consistent finding in developmental neuroscience is the decreased efficacy of response inhibition with aging (Lucci et al., 2013; Andres et al., 2008). This is apparent at the behavioral level (slower reaction times and/or increased number of errors), particularly in more demanding and difficult tasks, but also in ERPs (e.g., Falkenstein et al., 2002; Kropotov et al., 2016; Hämmerer et al., 2010; Vallesi, 2011; Verhaeghen and Cerella, 2002). This age-associated deficit has been linked to structural and functional changes within the PFC (e.g., West, 1996). An issue not yet investigated experimentally is the impact of acute stress on response inhibition in older compared to younger participants. Will older participants be more or less influenced by stress? Previous studies on the endocrine and cardiovascular stress response frequently reported an altered physiological stress response and an enhanced sensitivity to glucocorticoids and catecholamines with age, suggesting a different impact of stress on cognitive performance and respective cortical areas in older participants (Kudielka et al., 2004; Otte et al., 2005; Strahler et al., 2010; Arnsten et al., 1994; Arnsten and Goldman-Rakic, 1985). Previous work examining different cognitive domains has provided evidence for effects in both directions (for review see Wolf, 2015). For example, older participants were more influenced by stress with respect to its impact on interference in long-term memory recall (Hidalgo et al., 2014). In contrast, working memory was impaired after administration of the stress hormone cortisol in young but not in older participants (Wolf et al., 2001). Several additional studies investigated the impact of stress on cognition in older participants only, thus not allowing direct comparisons between the age groups (Wolf, 2015).

The goal of the current experiment was therefore to investigate how acute stress affects inhibitory control and its neural correlates in older compared to younger healthy men, under consideration of different levels of task difficulty. Stress was induced with the Trier Social Stress Test (Kirschbaum et al., 1993) and inhibitory control was measured with a Go/No-Go task with a reaction time limit and two compatibility conditions to enhance and vary task difficulty. In addition to behavioral data, stimulus-locked ERPs (N2, P3; N2d, P3d) for Go and No-Go stimuli and the difference wave No-Go minus Go were analyzed. Moreover, response-locked ERPs to false alarms (error-related negativity, Ne or ERN, and error positivity, Pe) were included in the analyses to explore stress effects on neural correlates of errors within the context of age and inhibition. Based on previous findings of a positive impact of acute stress on response inhibition, we expected enhanced accuracy and/or faster reaction times, particularly in the more complex incompatible task condition. With respect to the stimulus-locked ERPs, we expected enhanced N2d/N2 amplitudes and reduced P3d/P3 amplitudes after the TSST, with more pronounced alterations in the incompatible condition. In line with improved response inhibition behavior, we expected enhanced error processing, reflected by enhanced response-locked amplitudes. These beneficial effects may be altered by age.

2. Materials and methods

2.1. Participants

Sixty-one (40 young) healthy male participants were recruited from the Ruhr University Bochum (young), via local newspaper advertisements and by contacting associations catering to senior citizens. Exclusion criteria were smoking, any acute or chronic physical disease or mental disorder, use of medication or drugs, and being a non-native German speaker. Twelve participants had to be excluded due to technical problems during recording (5), insufficient artifact-free trials for the EEG analyses (5), or left-handedness, leaving 49 right-handed participants for analysis (age range: 19 – 75 yrs, see Table 1). Eligible participants were required to refrain from physical exercise and alcohol on the day prior, as well as beverages (except water) and meals within 1 h prior to the experimental session. The experiment was conducted in accordance with the Declaration of Helsinki and the research ethics

Table 1
Number of participants, mean age and BMI kg/m^2 (*SD*) of each group.

	young		older	
	stress group (n = 16)	control group (n = 14)	stress group (n = 9)	control group (n = 10)
age	24.87 (4.09)	23.64 (2.90)	67.22 (5.36)	67.30 (3.86)
	24.28 (3.56)		67.26 (4.50)	
BMI	23.16 (1.89)	24.10 (2.12)	26.76 (3.23)	25.90 (1.65)
	23.62 (2.02)		26.31 (2.49)	
years of educational and academic training	16.90 (2.94)	16.39 (3.11)	16.67 (3.28)	13.38 (3.28)
	16.66 (2.98)		16.00 (3.30)	

Note: Since the demographical data of one young participant in the stress group was missing, data of 48 participants are reported here. Age and BMI differed significantly between young and older participants ($F_{(1, 44)} = 1319.23$; $p = .000$; $\eta^2 = .97$; $F_{(1, 44)} = 16.96$; $p = .000$; $\eta^2 = .28$), while years of educational and academic training did not (age: $F_{(1, 44)} = 2.91$; $p = .095$, age x treatment $F_{(1, 44)} = 2.11$; $p = .153$).

committee of the Faculty of Psychology at the Ruhr University Bochum approved the study. All participants gave their written informed consent and were compensated with €25.

2.2. Procedure

All participants were randomly assigned to the stress or control procedure and examined individually, while age (young vs. older) was balanced across conditions. Older participants first performed the Mini Mental Status Test (MMST, Kessler et al., 2000) to check for cognitive impairment, revealing no evidence for early dementia (all MMST scores ≥ 27). Next, participants performed two training blocks of the Go/No-Go task followed by preparation for the electroencephalogram (EEG), after which participants underwent a stress procedure (Trierer Sozial Stress Test, TSST, Kirschbaum et al., 1993) or a control procedure (friendly TSST, Wiemers et al., 2013). Ten minutes after the end of the respective procedure, participants performed the Go/No-Go task. Before and after the TSST/friendly TSST, participants filled out the German version of PANAS (Positive and Negative Affect Schedule, Krohne et al., 1996; Watson et al., 1988), measuring their current positive and negative affect with ten items, each on a 5-point Likert Scale ranging from “very slightly” to “very much”. Over the course of the experiment participants provided four saliva samples for cortisol analysis using Salivette® collection devices (Sarstedt, Nürnbrecht, Germany): before the start of the TSST, +1 min, +10 min and + 35 min after the end of the TSST. Samples were frozen before they were sent to Prof. Kirschbaum's biochemical laboratory in Dresden, Germany. Free salivary cortisol concentrations were then determined by commercial chemiluminescence immunoassays (CALIA; IBL International, Hamburg, Germany). Inter- and intra-assay variations were below 10%.

After removal of the EEG devices, participants were debriefed and compensated for their participation. In total, the experiment lasted approximately 150 min.

2.3. Go/No-Go task

Response inhibition was measured with a Go/No-Go paradigm. The German words “DRÜCK” (press) and “STOPP” (stop) (Geneva, 26 pt, white font) served as Go and No-Go stimuli, with counterbalanced assignment. Participants were instructed to respond as fast as possible to a Go stimulus by pressing a pressure-sensitive button (see Willemssen et al., 2009) with their right thumb and to withhold a response to a No-Go stimulus. Stimuli were presented for 200 ms in random order with the restriction that a condition appeared four times in succession at most, followed by a black screen for 1000 ms (ISI). Two blocks of 240 trials each were carried out. A ratio of 70% Go to 30% No-Go was chosen, as this should result in a stronger response preparation with less accuracy and enhanced stimulus-locked amplitudes compared to an equiprobable Go/No-Go task (Bruin, 2002, and cited therein). In one block “DRÜCK” served as a Go stimulus and “STOPP” as a No-Go stimulus (compatible (c) trials), while in the remaining block the assignment was vice versa (incompatible (i) trials). The training consisted of 40 compatible and 40 incompatible trials with equiprobable presentation of Go and No-Go stimuli and accuracy feedback at the end of each block. The reaction time was limited to 550 ms and an acoustic warning signal (1000 Hz, 60db (SPL)) was presented via loudspeaker if the response did not occur in time. If the accuracy was too low, the training was repeated. The reaction time limit for the actual Go/No-Go task was set individually to the mean reaction time in the training trials with an additional 50 ms.

Our Go/ No-Go task originally comprised an additional part with the ratio 10% Go: 90% No-Go in order to extend our investigation to sustained attention besides response inhibition (Carter et al., 2013). This traditionally formatted task sustained attention to response task (TFT-SART) shows decreases in sustained attention in older participants (Staub et al., 2014). Compatibility was balanced across both ratio conditions, forming four different blocks. Four sequences with alternating compatible and incompatible blocks were randomly assigned to participants: (1) c 30% Go: 70% No-Go, i 30% Go: 70% No-Go, c 10% Go: 90% No-Go, i 10% Go: 90% No-Go, (2) i 30% Go: 70% No-Go, c 30% Go: 70% No-Go, i 10% Go: 90% No-Go, c 10% Go: 90% No-Go, (3) c 10% Go: 90% No-Go, i 10% Go: 90% No-Go, c 30% Go: 70% No-Go, i 30% Go: 70% No-Go, (4) i 10% Go: 90% No-Go, c 10% Go: 90% No-Go, i 30% Go: 70% No-Go, c 30% Go: 70% No-Go. Thus, blocks with 10% Go: 90% No-Go were separated from 30%Go: 70% No-Go blocks. The same reaction time limit was applied in both ratio conditions. Before each block, the participant was informed about the compatibility of the subsequent block.

Unfortunately, for 20 participants the number of artifact-free segments in the Go condition was too low for ERP analyses and the numbers of errors too low (< 1%) for accuracy or response-locked ERP analyses. Thus, we omit the 10%Go: 90% No-Go condition and focus our analyses merely on the two 70% Go: 30% No-Go blocks. However, for the sake of completeness, we report the behavioral results of the 10%Go: 90% No-Go blocks briefly in the following. The same analyses for accuracy and reaction times were performed as for the 30% Go: 70% No-Go blocks (see Statistical Analyses section below). Accuracy was lower in No-Go trials (error rate: *M (SEM)* = 0.10 (0.014)) relative to Go trials (0.04 (0.008); $F_{(1,45)} = 11.59$, $p = .001$, $\eta^2 = .21$), and older participants made more errors in general compared to young participants (older: 0.09 (0.011), young: 0.05 (0.009); $F_{(1,45)} = 4.83$, $p = .033$, $\eta^2 = .10$). Besides, the significant interaction between Go/No-Go and age showed that the age-related impairment was limited to the No-Go condition (older: Go 0.04 (0.012), No-Go 0.14 (0.021) young: Go 0.05 (0.010), No-Go 0.06 (0.017); $F_{(1,45)} = 6.04$, $p = .018$, $\eta^2 = .12$; $\psi_{Dunn} = 0.09$, C = 4). No further effect reach significance and stress had no impact (all $F < 3.47$, all $p > .05$). The analysis of the reaction time, which was based on only 47 participants as two older participants had no correct Go trial in the incompatible 10% Go: 90% No-Go block, revealed no significant effects (all $F < 3.03$, all $p > .05$).

2.4. TSST

The Trier Social Stress Test (TSST) was used as a stressor (Kirschbaum et al., 1993). After a five-minute preparation period, participants perform an oral presentation for a staged job interview as well as a challenging arithmetic task (quickly counting backwards in steps of 17) for a total of ten minutes while being videotaped. Additionally, they are evaluated by a panel (a woman and a man dressed in white lab coats) that deliberately refrains from any sort of feedback, thus creating a cold and reserved atmosphere. The TSST is known to reliably activate the SNS and the HPA axis (Dickerson and Kemeny, 2004). The non-stressful control condition friendly-TSST (Wiemers et al., 2013) also consists of an oral presentation and an easy arithmetic task, albeit with a supportive, friendly panel and without video recording. It thus lacks the stressful components of the TSST (social evaluative threat and uncontrollability) and does not elicit an HPA response. During both procedures, participants were sitting down to avoid shifting of the prepared EEG cap.

2.5. EEG recording and quantification

The EEG was recorded with a 64 Ag/AgCl electrode system (actiCAP, Brain Products GmbH, Gilching, Germany) according to the 10–10 electrode reference system (Chatrian et al., 1988) with the actiCAP ControlBox and QuickAmp 72 (Brain Products GmbH, Gilching, Germany). Four channels (former positions: PO8, Oz, PO10, Fp1) were used to recorded a bipolar horizontal EOG from the epicanthus of each eye and a bipolar vertical EOG from supra- and infra-orbital positions of the left eye. All EEG electrode impedances were kept below 20 kΩ. EEG and EOG were recorded in DC mode at 1000 Hz with a 200 Hz high cutoff with a grand average reference, while FCz was used as an online

reference electrode for impedance measurement.

Data was analyzed with BrainVision Analyzer 2 (Brain Products, Munich, Germany). FCz was interpolated at its original position offline (interpolation type spline, order 4, degree 10, lambda default). The data was resampled at 200 Hz and low pass filtered using a digital filter with half-power high cutoff of 12 Hz, 48 dB/oct and a Notch filter (50 Hz). The EEG was re-referenced offline to linked mastoids. Artifacts due to eye movements were corrected via the algorithm developed by Gratton et al. (1983). Trials with non-physiological artifacts were excluded from analysis via semiautomatic artifact rejection. Out of the original 72 No-Go trials and 168 Go trials in each compatibility condition, the mean number of accurate trials per participant in the *No-Go compatible* condition were (*M (SD)*) 49.63 (13.92), 47.16 (13.03) in the *No-Go incompatible* condition, 147.69 (17.44) in the *Go compatible* condition and 147.00 (25.31) in the *Go incompatible* condition. Between 80.51% and 100.00% of the segments of each participant were retained after artifact rejection. The mean number of incorrect No-Go trials per participant were 21.53 (12.80) in the *compatible* condition and 24.10 (12.62) in the *incompatible* condition. On average, 97.72% of these segments were retained after artifact rejection and used for the response-locked ERPs.

For the stimulus-locked ERPs, the EEG of trials with accurate responses were epoched offline into periods of 1000 ms, starting 200 ms prior to Go and No-Go stimuli onset. A baseline correction was performed using the first 200 ms interval as reference. Separate averages were computed for each electrode and individual for Go and No-Go trials for the two blocks (ratio 70:30 Go: No-Go compatible vs. incompatible). Subsequently, difference wave shapes (No-Go minus Go) were computed for each block. Using the grand average across participants to guide window selection, ERP maximum peak amplitude (μV) for the stimulus-locked N2 and P3 components of the original waves were detected semiautomatically for F3, Fz, F4, FC3, FCz, FC4, C3, Cz and C4 within windows of 200–300 ms post stimulus for the N2, and 325–475 ms for the P3. Similarly, to grasp the Go/ No-Go effect, ERP maximum peak amplitudes and latencies for the N2 and P3 of the difference wave, No-Go minus Go ('N2d', 'P3d') were detected for the same electrodes within the same windows. For statistical analyses, peak amplitudes were averaged over an interval of ± 3 (N2, N2d) and ± 4 (P3, P3d) data points (i.e., 35 ms and 45 ms, respectively).

For the response-locked ERPs, the EEG of correct Go trials and incorrect No-Go trials (false alarms) were epoched offline into periods of 700 ms, starting 50 ms prior to the response. A baseline correction was performed using the first 50 ms interval as reference and separate averages were computed for each electrode and individual for compatible and incompatible correct Go and incorrect No-Go trials. Next, the difference wave incorrect No-Go minus correct Go trials was built. Using the grand average across participants to guide window selection, ERP maximum peak amplitude (μV) and latency (ms) for the response-locked Ne_d and Pe_d components were detected semiautomatically as the negative amplitude peak within windows of 65–150 ms post response for the Ne_d and as the positive amplitude peak within 180–300 ms for the Pe_d. For statistical analyses, peak amplitudes were averaged over an interval of ± 2 (Ne_d) and ± 4 (Pe_d) data points (i.e., 25 ms and 45 ms, respectively) and analyzed at Fz, FCz and Cz for the Ne_d and at Fz, FCz, Cz, CPz, and Pz for the Pe_d. Besides this, within in the original waves the Ne and Pe were determined at the same electrode positions to further elucidate the error-related processes, selecting the mean amplitudes (μV) of the Ne and Pe components within the time interval 60–90 ms (Ne) and 180–300 ms (Pe).

2.5.1. Statistical Analyses

The data was edited with MATLAB and Excel 2010 and analyzed with IBM SPSS Statistics 20.

2.5.1.1. Stress manipulation and subjective measurements. To analyze the cortisol response to the stress and control procedures, an *age* (young vs. older) by *stress* (stress vs. control group) by *time of measurement* (repeated measure) mixed ANOVA was conducted. Cortisol data of one older participant in the TSST treatment group was missing; therefore, this analysis was based on 48 participants. The same analysis was conducted for the PANAS scores including the additional factor *affect* (positive vs. negative, repeated measure).

2.5.1.2. Behavioral measurements. Numbers of errors in Go and No-Go trials (i.e., missed responses and false alarms, respectively) with the 70%:30% ratio were summed up for each individual for compatible and incompatible trials separately and submitted to an *age* by *stress* by *Go No-Go error* (repeated measure) by *compatibility* (repeated measure) mixed ANOVA. The median RT [ms] of correct Go trials of each participant was submitted to the same ANOVA without the factor *Go No-Go error*.

2.5.1.3. Electrophysiological data. Our primary focus was on the mutual effect of stress and age on the Go/No-Go effect and error-related effects. Stimulus-locked N2d and P3d as well as response-locked Ne_d and Pe_d of the difference waves were therefore first submitted to separate mixed-design ANOVAs with the factors *age, stress* and the factor *compatibility* (repeated measures). For the stimulus-locked N2d and P3d, these ANOVAs included the additional within-subjects factors *caudality* (F, FC, C) and *lateralization* (left, midline, right), while they included the within-subjects factor *electrode position* (Fz, FCz, Cz for Ne_d and Fz, FCz, Cz, CPz, Pz for Pe_d) for the response-locked Ne_d and Pe_d.

To resolve the question whether possible stress effects or stress-age interactions were evoked by group differences in the crucial No-Go condition or by incorrect No-Go responses, respectively, the peak amplitudes of the N2 and P3 as well as Ne and Pe time interval of the original waves were submitted to the same corresponding analyses, with the additional factor *Go No-Go* (Go vs. No-Go, repeated measure).

For the latencies of N2d and P3d, as well as of Ne_d and Pe_d, separate mixed-design ANOVAs with the factors *age, stress* and *compatibility* (repeated measure) were calculated at the respective maximum amplitudes for No-Go minus Go (stimulus-locked ERPs; correct trials for the N2d, P3d) or No-Go minus Go errors difference waves (response-locked ERPs; incorrect No-Go trials vs. correct Go trials for the Ne_d, Pe_d), i.e., N2d at Cz, P3d at FC3, Ne_d at Cz and Pe_d at Pz.

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) and the critical difference ψ_{Dunn} ($\alpha = 0.05$) and number of comparisons C are specified. In case the assumption of sphericity was violated, the degrees of freedom for all ANOVAs were Greenhouse-Geisser corrected and corrected p-values, uncorrected degrees of freedom and Greenhouse-Geisser estimate (GG-ε) are given. The statistical significance level was set to $\alpha = 0.05$ (two-tailed).

Power values for the relevant statistical analyses are specified according to Hager (2004). Our basic hypotheses comprise an interaction between *stress* and *age*, which should be further qualified by the within-subjects factor *compatibility* for RTs and ERPs of the difference waves, and/or the within-subjects factor *Go No-Go* for accuracy and the original ERPs waves. Given our sample size of 49 participants and a significance level of 0.05, the three-way interactions *age x stress x compatibility* or *age x stress x Go No-Go* can detect a relatively small effect of $\Omega^2 \geq 0.05$ with a probability of at least 1-β (statistical power) > 0.80. This calculation assumes a plausible population correlation for reaction time measures of $\rho = 0.80$, $\rho = 0.35$ for accuracy, $\rho = 0.80$ for cortisol, $\rho = 0.40$ for ERP latencies and $\rho = 0.50$ for the ERP amplitudes. Should these interactions be further qualified by *caudality* and/or *lateralization* (N2/N2d, P3/P3d) or *electrode position* (Ne/Ne_d,Pe/Pe_d), the power even increases as the number of observations increases by including these within-subjects factors.

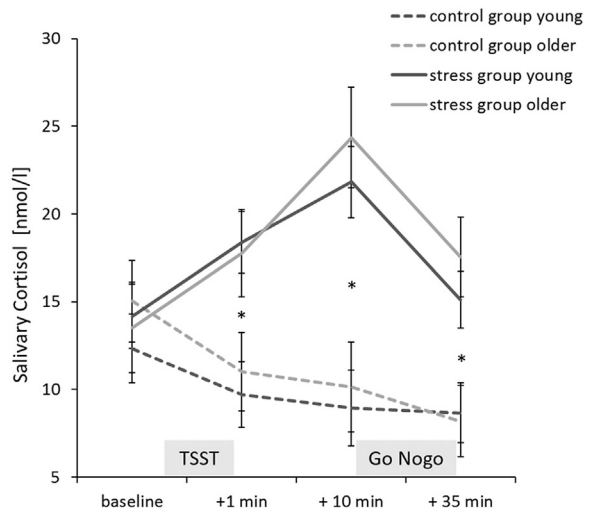

Fig. 1. Mean concentrations of salivary cortisol during the experimental session for young and older participants in the stress and the control group. The stress group showed a significant increase in cortisol after the TSST (Trier Social Stress TSST) compared to the control group after the friendly TSST. Age had no impact on cortisol concentrations. Error bars indicate standard errors of the mean (SEM). * = $p < .05$.

3. Results

3.1. Stress manipulation

The stress group showed a distinct increase in cortisol after the TSST relative to the control group (Fig. 1, *stress* x *time* $F_{(1,44)} = 24.23$, $p = .000$, GG-ε = 0.58, $\eta^2 = .36$; $\psi_{Dunn} = 2.72$, C = 4). Age had no impact (all $F < 1.67$, all $p > .10$).

Stressed participants reported enhanced negative affect as measured with the PANAS after the TSST (M (*SEM*) 1.61 (0.08)) compared to the control group (1.15 (0.08)) (Table 2, *stress* x *time x affect* $F_{(1,45)} = 4.99$, $p = .031$, $\eta^2 = .10$; $\psi_{Dunn} = 0.29$, C = 4). Age had no impact (all $F_{(1,45)} = 2.45$, $p < .05$).

3.2. Response inhibition behavior

As to be expected, more errors were made in No-Go trials (false alarms, *M (SEM)* = 0.32 (0.025)) relative to Go trials (0.01 (0.003); $F_{(1,45)} = 146.85$, $p = .000$, $\eta^2 = .77$). Besides this, the analysis revealed an effect of stress depending on compatibility (*Go No-Go* x *compatibility* x *stress*: $F_{(1,45)} = 5.67$, $p = .022$, $\eta^2 = .11$, for a summary of the behavioral and ERP results please see Supplement, Table A1). Stressed participants made fewer errors in compatible No-Go trials compared to incompatible No-Go trials and compared to the control group. Still, both groups made more errors in No-Go trials relative to Go trials ($\psi_{Dunn} = 0.06$, C = 8; Fig. 2, Table 2). Age had no impact (all $F < 1.55$, all $p > .10$).

Regarding RTs, the analysis revealed a main effect of age, with slower reactions in the older (*M (SEM)* = 261.62 (7.72)) compared to the young participants (*M (SEM)* = 238.54 (6.15) (Table 2, $F_{(1,45)} = 5.47$, $p = .024$, $\eta^2 = .11$)). Stress had no impact (all $F < 2.26$, all $p > .10$).

3.3. Stimulus-locked ERPs

3.3.1. Go No-Go effect

The N2 and P3 amplitudes of the original waves showed the expected Go/No-Go pattern, with more negative N2 amplitudes followed by more positive P3 amplitudes for No-Go trials compared to Go trials at analyzed frontal and frontocentral electrodes (*Go No-Go*: N2 $F_{(1,45)} = 6.46$, $p = .015$, $\eta^2 = .13$; P3 $F_{(1,45)} = 98.19$, $p = .001$, $\eta^2 = .69$). Analyses of the difference waves No-Go minus Go confirmed this pattern, revealing a pronounced frontocentral to central negative peak at 276.24 ms (N2d −2.11 μV (0.36)), followed by a frontocentral positivity at 384.67 ms on average (P3d 4.42 μV (0.39)).

3.3.2. Age effects

Analyses of the N2d and P3d amplitudes of the difference wave showed reduced N2d (especially at left and central electrode sites) and reduced P3d amplitudes (F, FC) in older relative to young participants (N2d *age*: $F_{(1,45)} = 4.18$, $p = .047$, $\eta^2 = .09$; *age x caudality x lateralization:* $F_{(4,180)} = 3.74$, $p = .017$, GG-ε = 0.66, $\eta^2 = .08$, $\psi_{Dunn} = 0.60$, C = 9; P3d *age x caudality*: $F_{(4,180)} = 4.74$, $p = .023$, GG-ε = 0.67, $\eta^2 = .10$, $\psi_{Dunn} = 0.81$, C = 3; see Fig. 3B, Table 3). The analysis of the N2 and P3 of the original waves showed reduced Go and No-Go amplitudes in older relative to young participants, while this reduction was stronger for No-Go N2 than Go N2, leading to a weakened No-Go > Go pattern merely present at the right electrode site (N2: *age x Go No-Go*: $F_{(1,45)} = 4.57$, $p = .038$, $\eta^2 = .09$, *age x Go No-Go x caudality x lateralization*: $F_{(4,180)} = 4.55$, $p = .007$, GG-ε = 0.65, $\eta^2 = .09$; $\psi_{Dunn} = 0.47$, C = 36, see Fig. 3A, Table 3). For the P3, the age effect was independent of Go/No-Go condition (P3 age x *caudality* $F_{(2,90)} = 9.06$, $p = .003$, GG-ε = 0.55, $\eta^2 = .17$; age x *caudality x lateralization* $F_{(4,180)} = 5.25$, $p = .002$, GG-ε = 0.75, $\eta^2 = .11$; $\psi_{Dunn} = 0.62$, C = 9 (FC, C), see Table 3). Regarding the latency of N2d and P3d, older participants exhibited delayed N2d and P3d amplitudes (N2d *M (SEM)* = 287.99 ms (4.94), P3d 407.56 ms (4.95)) relative to young participants (N2d 265.49 ms (3.93), P3d 367.75 ms (3.94)) (Table 2, N2d *age*: $F_{(1,45)} = 13.87$, $p = .001$, $\eta^2 = .24$; P3d *age*: $F_{(1,45)} = 39.61$, $p = .000$, $\eta^2 = .47$; Fig. 3B).

3.3.3. Stress effects

Stress altered N2d and P3d amplitudes (*stress x caudality x lateralization* N2d $F_{(4,180)} = 3.14$, $p = .034$, GG-ε = 0.66, $\eta^2 = .07$; P3d $F_{(4,180)} = 3.26$, $p = .024$, GG-ε = 0.74, $\eta^2 = .07$; *stress x lateralization* P3d $F_{(2,90)} = 4.04$, $p = .040$, GG-ε = 0.63, $\eta^2 = .08$). Stressed participants had a more pronounced and well-defined N2d, showing more negative N2d amplitudes on the right hemisphere (F4, FC4) and at Cz relative to the control group, who in turn showed a larger N2d on the left hemisphere, reaching significance at FC3 ($\psi_{Dunn} = 0.60$, C = 9; Fig. 4A, Fig. 4C, Table 3). The analysis of the original waves showed that this was due to larger No-Go N2 amplitudes at crucial F4 and FC4 for the stress group compared to the control group. Besides this, post-hoc tests showed slightly larger Go N2 amplitudes at Fz, FC3 and FC4, and more positive Go N2 amplitudes at Cz in the stress relative to the control group (*stress x Go No-Go x caudality x lateralization* $F_{(4,180)} = 4.14$, $p = .011$, GG-ε = 0.65, $\eta^2 = .08$; $\psi_{Dunn} = 0.47$, C = 36; Fig. 4B, Table 3). Similar to the N2d, stressed participants showed a more pronounced and definite P3d, with more positive P3d amplitudes on the left hemisphere as well as at FCz and Cz relative to the control group, but smaller P3d amplitudes at F4 ($\psi_{Dunn} = 0.73$, C = 9; Fig. 4A, Fig. 4C, Table 3). Regarding the P3 of the original Go No-Go waves, the stress group showed more positive P3 No-Go amplitudes at all electrodes, reaching significance at all electrodes but F4, where the Go P3 was significantly more positive (*stress x Go No-Go x caudality x lateralization* $F_{(4,180)} = 4.57$, $p = .004$, GG-ε = 0.80, $\eta^2 = .09$; $\psi_{Dunn} = 0.60$, C = 36; Fig. 4B). Stress did not alter N2d and P3d latencies (Table 2, all $F < 3.51$, all $p > .05$). In sum, acute stress led to enhanced N2d and P3d amplitudes due to enhanced No-Go – N2 and P3 amplitudes.

Besides this, the P3d was mutually altered by age, stress and compatibility depending on the lateralization (*age x stress x lateralization x compatibility* $F_{(2,90)} = 3.32$, $p = .041$, GG-ε = 0.99, $\eta^2 = .07$). Comparing the control group and the stress group within each age group, the post-hoc tests showed that within younger participants, stress led to enhanced P3d amplitudes at left electrode positions in

Table 2
Descriptive statistics (mean (standard error of the mean)) for RTs, accuracy, self-reports, cortisol and ERP latencies for the factors stress, age and stress x age, respectively.

	stress		*age*		*stress x age*			
	stress group	control group	young participants	older participants	stress group		control group	
					young	older	young	older
	M (SEM)	*M (SEM)*	*M (SEM)*	*M (SEM)*	*M (SEM)*	*M (SEM)*	*M (SEM)*	*M (SEM)*
RT [ms]								
compatible	257.82	248.13	241.59	264.35	243.54	272.10	239.64	256.61
	(7.69)	(7.64)	(6.75)	(8.48)	(9.22)	(12.30)	(9.86)	(11.67)
incompatible	247.81	246.58	235.50	258.89	238.23	257.38	232.76	260.40
	(7.08)	(7.03)	(6.22)	(7.81)	(8.50)	(11.33)	(9.08)	(10.75)
overall	252.81	247.35	238.54	261.62	240.89	264.74	236.20	258.51
	(7.00)	(6.96)	(6.15)	(7.72)	(8.40)	(11.20)	(8.98)	(10.62)
error rates								
Go compatible	0.012	0.012	0.013	0.010	0.017	0.007	0.009	0.014
	(0.004)	(0.004)	(0.004)	(0.005)	(0.005)	(0.007)	(0.005)	(0.006)
Go incompatible	0.012	0.018	0.015	0.015	0.013	0.011	0.017	0.020
	(0.004)	(0.004)	(0.004)	(0.005)	(0.005)	(0.007)	(0.005)	(0.006)
No-Go compatible	0.239	0.366	0.337	0.268	0.266	0.211	0.408	0.324
	(0.039)	(0.039)	(0.034)	(0.043)	(0.047)	(0.063)	(0.050)	(0.060)
No-Go incompatible	0.324	0.358	0.350	0.333	0.303	0.346	0.397	0.319
	(0.039)	(0.038)	(0.034)	(0.043)	(0.046)	(0.062)	(0.050)	(0.059)
Go overall	0.0129	0.015	0.014	0.013	0.015	0.009	0.013	0.017
	(0.004)	(0.004)	(0.004)	(0.004)	(0.005)	(0.006)	(0.005)	(0.006)
No-Go overall	0.282	0.362	0.344	0.300	0.285	0.279	0.402	0.322
	(0.036)	(0.036)	(0.031)	(0.039)	(0.043)	(0.060)	(0.046)	(0.054)
subj. measurement								
PANAS positive before TSST/friendly TSST	3.095	3.144	2.959	3.281	2.969	3.222	2.948	3.340
	(0.125)	(0.124)	(0.110)	(0.138)	(0.150)	(0.200)	(0.160)	(0.189)
PANAS positive after TSST/friendly TSST	3.030	3.122	2.958	3.194	2.881	3.178	3.035	3.210
	(0.136)	(0.135)	(0.119)	(0.150)	(0.163)	(0.217	(0.174)	(0.206)
PANAS negative before TSST/friendly TSST	1.228	1.233	1.246	1.215	1.256	1.200	1.236	1.230
	(0.061)	(0.060)	(0.053)	(0.067)	(0.073)	(0.097)	(0.078)	(0.092)
PANAS negative after TSST/friendly TSST	1.614	1.148	1.393	1.369	1.650	1.578	1.137	1.160
	(0.080)	(0.079)	(0.070)	(0.088)	(0.096)	(0.127)	(0.102)	(0.121)
Cortisol in nmol/l								
Cortisl baseline	13.853	13.681	13.254	14.280	14.174	12.334	13.532	15.028
	(1.586)	(1.517)	(1.341)	(1.738)	(1.832)	(1.958)	(2.590)	(2.317)
Cortisol + 1 min	18.086	10.361	14.053	14.394	18.398	9.708	17.774	11.015
	(1.525)	(1.458)	(1.289)	(1.671)	(1.761)	(1.883)	(2.490)	(2.227)
Cortisol + 10 min	23.089	9.546	15.385	17.251	21.823	8.948	24.356	10.145
	(1.761)	(1.684)	(1.488)	(1.929)	(2.033)	(2.173)	(2.875)	(2.572)
Cortisol + 35 min	16.329	8.429	11.890	12.868	15.110	8.669	17.548	8.188
	(1.395)	(1.334)	(1.179)	(1.528)	(1.611)	(1.722)	(2.278)	(2.038)
ERP Latencies in ms								
N2d at Cz	270.330	282.143	264.487	287.986	260.938	268.036	279.722	296.250
	(4.476)	(4.448)	(3.931)	(4.936)	(5.371)	(5.742)	(7.162)	(6.794)
P3d at FC3	388.837	386.464	367.746	407.556	366.562	368.929	411.111	404.000
	(4.487)	(4.459)	(3.941)	(4.948)	(5.384)	(5.756)	(7.179)	(6.811)
Ne_d at Cz	84.314	103.232	91.060	96.486	83.906	98.214	84.722	108.250
	(5.183)	(5.150)	(4.552)	(5715)	(6.219)	(6.649)	(8.292)	(7.867)
Pe_d at Pz	274.063	281.679	266.116	2.89.625	260.25	271.607	287.500	291.750
	(8.438)	(8.385)	(7.412)	(9.305)	(10.126)	(10.825)	(13.501)	(12.809)

particular, as well as, though less pronounced, at central and right electrode positions only for compatible trials, not for incompatible trials.

In older participants, stress was associated with a more pronounced and definite P3 for both compatible and incompatible trials, with enhanced P3d amplitudes on the left hemisphere, but reduced P3d amplitudes on the right hemisphere ($\psi_{Dunn} = 1.16$, C = 12). The analysis of the original waves showed that these effects were caused by more positive No-Go P3 amplitudes in compatible trials in young stressed participants relative to the control group. In stressed older participants, both Go and No-Go amplitudes were enhanced in compatible and incompatible trials, this effect being more pronounced for No-Go trials on left but not right electrodes sites (*age x stress x Go No-Go x lateralization x compatibility* $F_{(2,90)} = 3.25$, $p = .047$, GG-ε = 0.93, $\eta^2 = .07$; $\psi_{Dunn} = 0.91$, C = 36).

3.4. Response-locked ERPs

3.4.1. False alarm effect

The analyses of the original response-locked waves of correct Go and incorrect No-Go trials revealed the expected pattern, with an enhanced Ne, particularly pronounced at Cz, and enhanced Pe amplitudes of false alarm No-Go responses compared to correct Go responses (*Go No-Go-error* Ne $F_{(1,45)} = 60.60$, $p = .000$, $\eta^2 = .57$; Pe $F_{(1,45)} = 30.26$, $p = .000$, $\eta^2 = .40$; *electrode position x Go No-Go-error* Ne $F_{(2,90)} = 87.00$, $p = .000$, GG-ε = 0.53, $\eta^2 = .66$; Pe $F_{(4,180)} = 4.48$, $p = .017$, GG-ε = 0.45, $\eta^2 = .09$). Confirming this, the response-locked Ne_d of the difference wave No-Go-error – Go correct peaked around 94 ms and was maximal at Cz (*electrode position*: Ne_d $F_{(2,90)} = 113.04$, $p = .000$, GG-ε = 0.55, $\eta^2 = .72$; $\psi_{Dunn} = 1.36$, C = 3; Fz: − 5.95 (0.88), FCz: − 10.28 (1.11), Cz: − 14.53 (1.40)). The Pe_d peaked around 277.87 ms and was maximal at CPz and Pz (*electrode position*: Pe_d

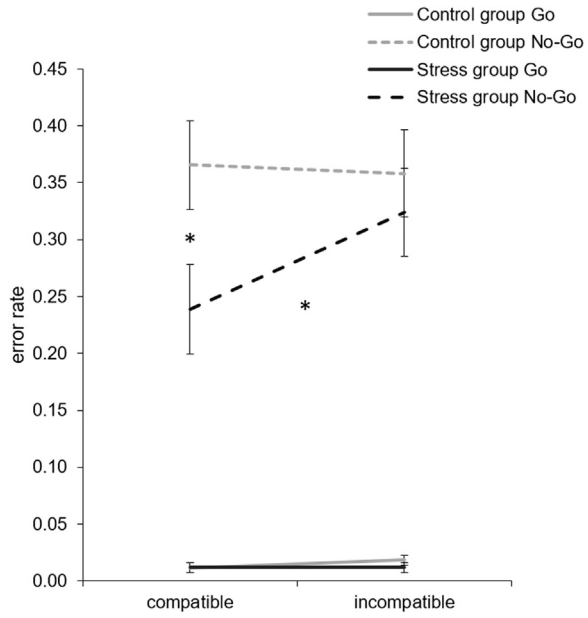

Fig. 2. Mean error rates of Go and No-Go trials for the stress and the control group, separate for compatible and incompatible blocks. Participants in the stress group made fewer errors in compatible No-Go trials relative to the control group and relative to incompatible No-Go trials. Error bars indicate *SEM*. * = $p < .05$.

$F_{(4,180)} = 10.97$, $p = .000$, GG-ε = 0.39, $\eta^2 = .20$; $\psi_{Dunn} = 2.06$, C = 10; CPz: 6.53 (0.71), Pz: 6.81 (0.71)).

3.4.2. Age effects

Older participants showed both a reduced Ne_d (FCz, Cz) and Pe_d peak (Cz to Pz) compared to young participants (*age x electrode position*: Ne_d $F_{(2,90)} = 12.08$, $p = .001$, GG-ε = 0.55, $\eta^2 = .21$; $\psi_{Dunn} = 1.92$, C = 3; Pe_d $F_{(4,180)} = 8.38$, $p = .002$, GG-ε = 0.38, $\eta^2 = .16$; $\psi_{Dunn} = 1.89$, C = 5; Fig. 5, Table 4). Analysis of the original response-locked Ne and Pe showed that the mean amplitudes for both incorrect No-Go and correct Go trials were reduced in the Ne (FCz, Cz), while only incorrect No-Go amplitudes were reduced in the Pe (Cz to Pz; Fig. 6A, Table 4). Regarding the Ne, both young and older participants showed the expected incorrect No-Go > correct Go effect at all analyzed electrodes, while older participants showed this effect for the Pe only at Fz, FCz and Pz (*age x Go No-Go error x electrode position* Ne: $F_{(2,90)} = 16.53$, $p = .000$, GG-ε = 0.53, $\eta^2 = .27$, $\psi_{Dunn} = 1.59$, C = 12, Pe $F_{(4,180)} = 11.35$, $p = .00\,0$, GG-ε = 0.46, $\eta^2 = .20$, $\psi_{Dunn} = 1.46$, C = 20; Fig. 6A, Table 4). Age did not influence the latencies of Ne_d and Pe_d (Table 2, all $F < 3.91$, all $p > .05$).

3.4.3. Stress effects

Stressed participants exhibited an enhanced Ne_d amplitude (−12.61 μV (1.56)) relative to the control group (−7.90 μV (1.55)), irrespective of age, compatibility and electrode position (*Stress* $F_{(1,45)} = 4.591$, $p = .038$, $\eta^2 = .09$; Fig. 5). At the descriptive level, the Pe_d amplitude showed the same pattern, with enhanced Pe_d amplitudes in the stress group not reaching significance (*Stress* $F_{(1,45)} = 2.83$, $p = .099$, $\eta^2 = .06$). Analyses of the Ne and Pe of the original waves showed that the stress group exhibited enhanced Ne and Pe amplitudes for incorrect No-Go trials compared to the control group (Ne: *Stress x Go No-Go error* $F_{(1,45)} = 6.57$, $p = .014$, $\eta^2 = .13$, $\psi_{Dunn} = 3.63$, C = 4; Pe *Stress x Go No-Go error* $F_{(1,45)} = 6.12$, $p = .017$, $\eta^2 = .12$, $\psi_{Dunn} = 1.83$, C = 4; Fig. 6B, Table 5). The stress group showed the expected pattern, with significantly enhanced Ne and Pe amplitudes for incorrect No-Go relative to correct Go trials, while the control group showed such a difference only for the Ne amplitude. Regarding the latencies of the Ne_d and Pe_d, the analyses revealed that stressed participants showed shorter latencies for the Ne_d compared to the control group (Table 2, stress group: 84.31 ms (5.18), control group: 103.23 ms (5.15); *stress* $F_{(1,45)} = 6.70$, $p = .013$, $\eta^2 = .13$). Stress did not alter the latency of the Pe_d (all $F < 1.81$, all $p > .05$).

4. Discussion

The present study investigated the influence of acute stress on response inhibition, error-processing and their neural correlates in healthy young and older men.

Irrespectively of age, stressed participants reported enhanced negative affect and showed substantially increased cortisol levels, similar to what was found in other studies (Goodman et al., 2017; Lai, 2014).

4.1. Impact of acute stress on response inhibition performance

After acute stress, participants made fewer errors in No-Go trials relative to the control group in the compatible condition, i.e., when the semantic meaning of the Go and No-Go stimuli corresponded to the instruction. This improvement supports the idea that stress selectively enhances response inhibition, contrasting its detrimental effect on other core executive functions (Shields et al., 2016a; Schoofs et al., 2008; Plessow et al., 2012). However, unlike previous findings (Qi et al., 2017; Dierolf et al., 2017; Shields et al., 2016a), this is the first time acute stress could be shown to alter the actual outcome of response inhibition, i.e., the accuracy in inhibiting responses to the inhibition stimuli, while reaction times in correct response trials remained constant. Due to an adaptive but strict reaction time limit, our Go/No-Go task produced relatively high No-Go error rates (i.e., > 30%). In contrast, the task used by Qi et al. (2017) might not have been sensitive enough to detect stress effects due to ceiling effects for accuracy (cf. Dierolf et al., 2017). On the other hand, acute stress did not improve accuracy in the more difficult incompatible condition, suggesting there might be a threshold in task difficulty up to which acute stress-induced improvement of accuracy emerges. This is in accordance with the Yerkes-Dodson Law, proposing a positive linear relationship between performance and arousal for simple tasks, while this relationship follows an inverted-U shaped curve for difficult tasks (Yerkes and Dodson, 1908). Besides enhanced difficulty, further characteristics of the incompatible task condition might account for the lack of stress impacts on accuracy in this condition. Beyond a mere response inhibition, incompatible trials require response selection while concurrently overcoming the automatic response activation by the semantic meaning of the stimulus, which involves shifting and reversal learning. While shifting is impaired by acute stress (Plessow et al., 2012; Shields et al., 2016a, 2016b), reversal learning might be less prone to stress effects (Butts et al., 2013; Shields et al., 2016a; Thai et al., 2013). Thus, these opposed effects of stress might cancel each other out, resulting in unaltered accuracy in incompatible trials. Further research on the impact of stress on different types of task difficulty in response inhibition is needed.

4.1.1. Impact of acute stress on Go No-Go stimulus-locked and response-locked ERPs

4.1.1.1. N2/N2d and P3/P3d. While both groups showed the expected No-Go > Go effect, stress amplified N2/N2d and P3/P3d amplitudes in both young and older participants, accompanied by more well-defined topographies, since the stress effect was most pronounced at electrode positions at which these components have their maximum amplitude. Enlarged No-Go-N2 amplitudes are interpreted as top-down response inhibition processes prior to the motor response, essential for successful inhibition (Fallgatter and Strik, 1999; Falkenstein et al., 1999) and /or conflict monitoring (Nieuwenhuis et al., 2003; Falkenstein, 2006). The P3/P3d reflects later aspects of inhibition (Falkenstein et al., 2002), associated with motor response inhibition and evaluation or finalization of the inhibitory process (Band and van Boxtel, 1999; Smith et al., 2008). Hence, the present results suggest that shortly after

Table 3
Descriptive statistics (mean (standard deviation)) for the stress and the control group as well as young and older participants for the N2d, P3d, N2 and P3 amplitudes (μV) at analyzed electrode positions. For the N2 and P3, the factor Go No-Go is considered.

			stress		*age*	
			stress group *M (SEM)*	control group *M (SEM)*	young participants *M (SEM)*	older participants *M (SEM)*
			stress x caudality x lateralization		*age x caudality x lateralization*	
N2d						
F	left		−.848 (.457)	−.986 (.454)	−1.437 (.401)	−.397 (.504)
	central		−1.833 (.491)	−1.816 (.488)	−2.607 (.431)	−1.042 (.541)
	right		−2.239 (.534)	−1.405 (.530)	−1.841 (.469)	−1.803 (.589)
FC	left		−.833 (.539)	−1.558 (.536)	−1.927 (.474)	−.464 (.595)
	central		−2.817 (.622)	−2.595 (.618)	−3.997 (.547)	−1.414 (.686)
	right		−3.249 (.689)	−2.561 (.684)	−2.913 (.605)	−2.897 (.759)
C	left		−.840 (.479)	−1.351 (.476)	−2.063 (.421)	−.128 (.528)
	central		−3.875 (.777)	−2.929 (.772)	−5.296 (.683)	−1.507 (.857)
	right		−3.339 (.609)	−2.823 (.605)	−3.471 (.535)	−2.691 (.672)
			stress x caudality x lateralization		*age x caudality*	
P3d						
F	left		5.133 (.474)	3.490 (.471)	3.853 (.453)	3.815 (.568)
	central		5.204 (.507)	3.927 (.503)		
	right		2.221 (.791)	3.029 (.768)		
FC	left		6.796 (.597)	4.355 (.593)	5.123 (.533)	4.334 (.669)
	central		6.242 (.589)	5.050 (.585)		
	right		2.918 (9.53)	3.280 (.947)		
C	left		5.979 (.602)	4.399 (.598)	5.393 (.558)	3.883 (.701)
	central		5.903 (.737)	5.455 (.732)		
	right		3.063 (.775)	3.028 (.771)		
			stress x caudality x lateralization x Go No-Go		*age x caudality x lateralization x Go No-Go*	
N2						
F	left	No - Go	.624 (.571)	.696 (.567)	−.693 (.501)	2.013 (.629)
		Go	.430 (.528)	.812 (.524)	−.132 (.463)	1.374 (582)
	central	No - Go	−.547 (.637)	−.240 (.633)	−2.082 (.560)	1.295 (.703)
		Go	.089 (.588)	.655 (.584)	−.486 (.516)	1.230 (.648)
	right	No - Go	.087 (.650)	.790 (.646)	−.559 (.571)	1.436 (.717)
		Go	1.182 (.566)	1.398 (.562)	.274 (.497)	2.306 (.624)
FC	left	No - Go	.494 (.613)	.384 (.609)	−.786 (.538)	1.664 (.676)
		Go	.123 (.657)	.934 (.653)	.221 (.577)	.836 (.725)
	central	No - Go	−1.466 (.798)	−1.016 (.793)	−3.192 (.701)	.710 (.880)
		Go	.198 (.689)	.404 (.684)	−.110 (.605)	.712 (.759)
	right	No - Go	−.706 (.701)	.385 (.696)	−1.061 (.615)	.740 (.773)
		Go	1.201 (.644)	1.855 (.639)	.849 (.565)	2.207 (.710)
C	left	No - Go	1.120 (.624)	.720 (.620)	.222 (.548)	1.617 (.688)
		Go	.934 (.647)	1.063 (.642)	1.323 (.568)	.673 (.713)
	central	No - Go	−1.440 (.958)	−1.258 (.952)	−3.189 (.841)	.491 (1.056)
		Go	1.426 (.827)	.586 (.882)	1.254 (.726)	.758 (.912)
	right	No - Go	−.001 (.704)	.385 (.700)	−.445 (.619)	.829 (.777)
		Go	2.211 (.680)	2.106 (.675)	2.142 (.597)	2.175 (.749)
			stress x caudality x lateralization x Go No-Go		*age x caudality x lateralization*	
P3						
F	left	No - Go	7.736 (.864)	6.076 (.858)	4.803 (.624)	5.232 (.784)
		Go	3.166 (.641)	3.091 (.637)		
	central	No - Go	8.747 (.789)	7.269 (.784)	5.686 (.585)	6.246 (.735)
		Go	4.078 (.628)	3.771 (.624)		
	right	No - Go	6.024 (.892)	5.867 (.886)	4.904 (.640)	4.959 (.804)
		Go	4.429 (.785)	3.405 (.780)		
FC	left	No - Go	10.729 (.863)	8.309 (.858)	7.394 (.605)	6.613 (.760)
		Go	4.486 (.642)	4.491 (.638)		
	central	No - Go	11.657 (.906)	10.104 (.900)	8.557 (.655)	7.927 (.823)
		Go	5.854 (.672)	5.352 (.668)		
	right	No - Go	8.122 (.833)	7.401 (.827)	6.893 (.568)	6.155 (.713)
		Go	5.841 (.780)	4.731 (.775)		
C	left	No - Go	10.702 (.843)	8.877 (.837)	8.521 (.604)	6.511 (.759)
		Go	5.385 (.641)	5.101 (.637)		
	central	No - Go	13.338 (1.148)	12.103 (1.141)	11.482 (.817)	8.660 (1.026)
		Go	7.969 (.820)	6.873 (.815)		
	right	No - Go	8.706 (.816)	7.326 (.811)	7.508 (.590)	6.145 (.740)

acute stress, premotor response inhibition and conflict monitoring are fortified irrespective of age, replicating previous results (Dierolf et al., 2017; Qi et al., 2017). Moreover, our present results showing amplified P3/P3d amplitudes in the stress group indicate that acute stress supports motor response inhibition and finalization of the inhibitory process.

4.1.1.2. Ne/Ne_d and Pe/Pe_d. Similar to the results from the stimulus-locked ERPs, acute stress amplified the response-locked ERP Ne/Ne_d and Pe/Pe_d amplitudes and reduced Ne_d latencies, independent of age. Previous work on the Ne/Ne_d (Falkenstein et al., 1991) or ERN (Gehring et al., 1993) suggests that this negative ERP reflects error detection. The Pe/Pe_d, i.e. error-positivity, has been linked to error

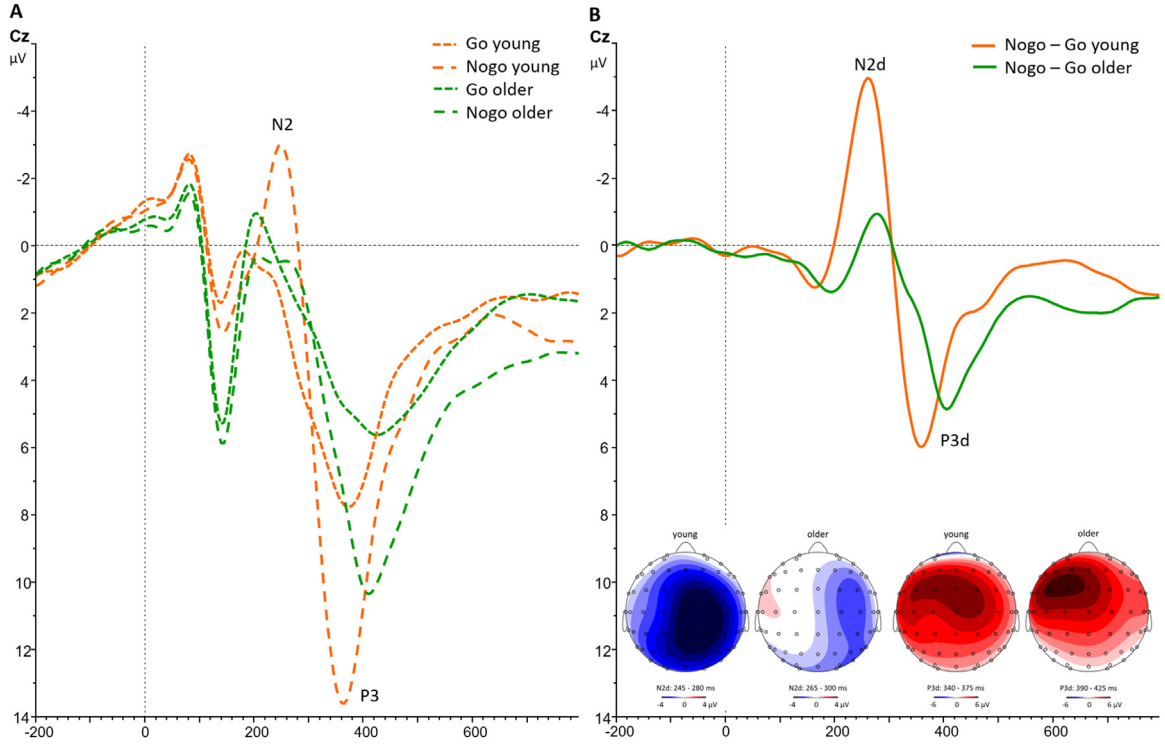

Fig. 3. Grand average ERPs at Cz of the original Go No-Go waves (left panel) and the difference wave No-Go–Go (right panel) for young (orange lines) and older participants (green lines), averaged over compatibility and stress treatment. Older participants show reduced and delayed N2d, N2, P3d and P3 amplitudes. For older participants, the No-Go > Go N2 effect was abolished at left and midline electrodes and strongly reduced at right electrode sites (see topographical maps of the No-Go – Go difference on the right).

awareness (Niessen et al., 2017; Nieuwenhuis et al., 2001) or, alternatively, might reflect the emotional response to an error (Falkenstein et al., 2000). Our present results indicate that error detection as well as error awareness were strengthened, and error detection speeded up after acute stress.

In sum, both early stages of the inhibitory processes and error processing were reinforced by stress. The ERP results are in line with increased performance accuracy in stressed participants. The No-Go-N2/N2d has been shown to be amplified with practice and effective response inhibition (Falkenstein et al., 1999; Schapkin et al., 2007). Similarly, the Ne/Ne_d is augmented in participants with high accuracy or under the emphasis of accuracy (Falkenstein et al., 2000; Gehring et al., 1993; Hajcak et al., 2005). Since the improvement in accuracy was limited to the compatible No-Go condition, a generally increased emphasis on accuracy by acute stress seems unlikely. Overall, our behavioral and ERP results are in line with the notion that inhibitory control is improved after acute stress (Shields et al., 2016a). However, since our ERP results show no impact of compatibility in contrast to the behavioral results, our findings indicate that additional later processes might be differentially affected by stress, consolidating our broader ERP effects with the more specific behavioral improvement. Taken together, our present findings replicate the results of enhanced N2d/No-Go N2 amplitudes shown in the independently designed and conducted study by Dierolf et al. (2017) and extend these findings to different levels of task complexity, an older age and error processing.

Our ERP results cast light on the reasons for this improvement in the face of the otherwise detrimental impact of stress on other executive functions. The model of the reallocation of neural resources after acute stress by Hermans et al. (2014) states that the salience network is enhanced directly after acute stress at the cost of the executive control network, highlighting the crucial factor of time lag between stress induction and cognitive testing. This reciprocal pattern lasts for about one hour, after which it is reversed. The initial downregulation of the executive control network after acute stress might explain the impairment of other executive functions by acute stress (Shields et al., 2016a). However, the main neural generator of the N2 and ERN is the dorsal anterior cingulate cortex (dACC) (Taylor et al., 2007; Pandey et al., 2012), which is part of the salience network (Hermans et al., 2014). Since our study measured response inhibition within one hour after acute stress, the improvement is in the line with the prediction made by the model. The broader network generating the No-Go P3 (Nieuwenhuis et al., 2003; Baumeister et al., 2014; Huster et al., 2010; Stock et al., 2016) cannot be assigned exclusively to the salience network or the executive control network. Activation of the inferior frontal gyrus / ventrolateral PFC during psychological stress, modulated by the increase in cortisol, has been reported by Dedovic et al. (2009). This activation is associated with a reduced No-Go P3 (Jamadar et al., 2010), which could lead to diminished P3 immediately after the stressor, explaining our previous results (Dierolf et al., 2017). At the same time, the activation of the salience network comprising the insula and parts of the ACC, both involved in the generation of the P3 (Baumeister et al., 2014; Zhang et al., 2012), could lead to an eventual reversal of the Nogo-P3 reduction, resulting in enhanced amplitudes in our stress group 20 min after the stressor. Regarding the Pe, evidence of its neural generator(s) is less conclusive. It indicates partially overlapping neural sources (ACC) for the Pe and ERN (Taylor et al., 2007; Luck and Kappenman, 2013), which might account for the similar stress-induced enhancement of ERN and Pe.

Besides the timing between stressor and response inhibition performance, different types of stressors might account for the divergent findings on stress modulation of the No-Go P3 and response inhibition behavior (Dierolf et al., 2017; Qi et al., 2017), since brain response patterns specific to individual stress induction paradigms have been reported by van Oort et al. (2017). Moreover, a recent study by Jiang and Rau (2017) indicates that the stimulus type and its valence might be a determining factor as well.

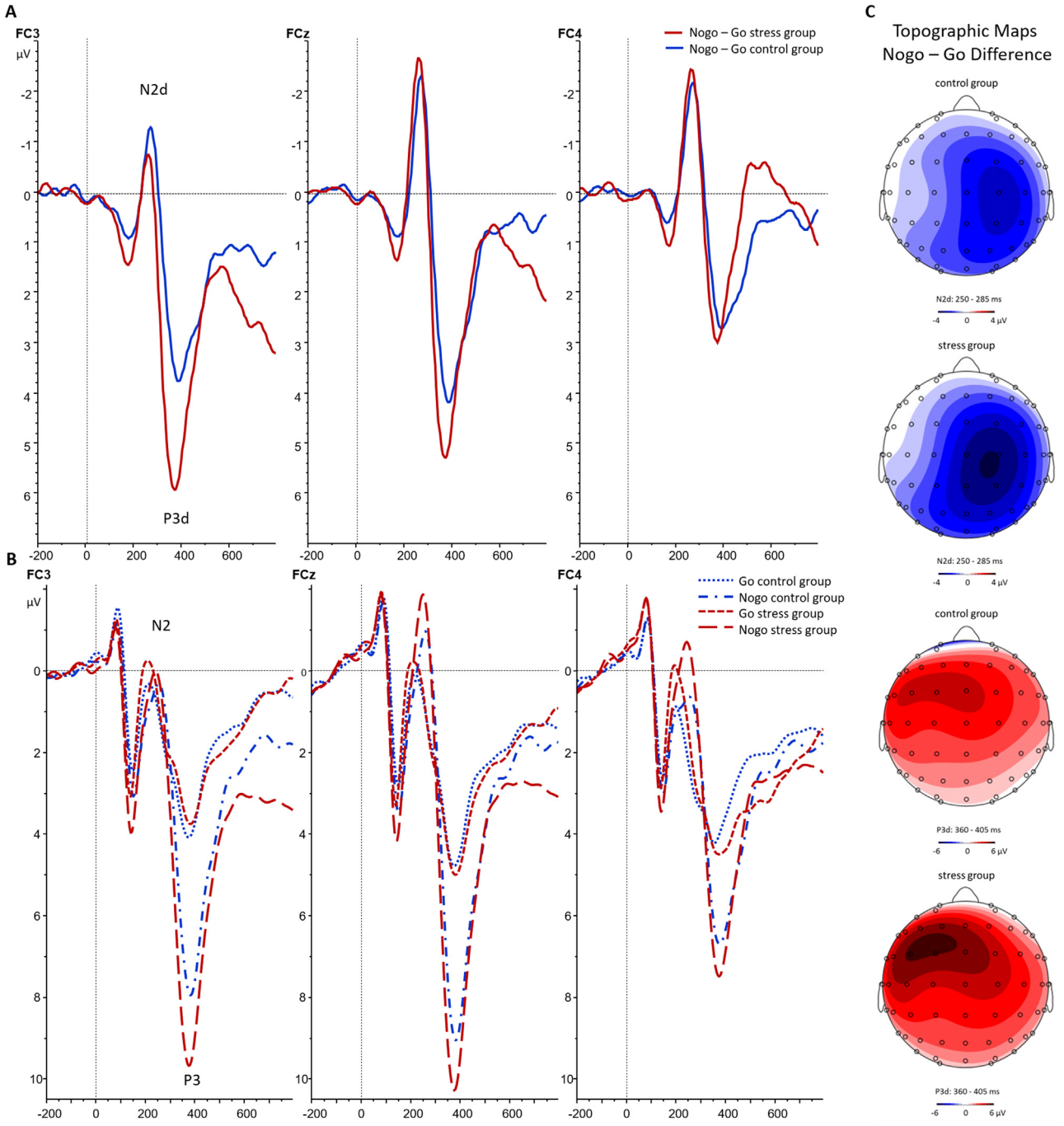

Fig. 4. A. Grand average ERPs at FC3, FCz and FC4 for the No-Go minus Go difference waves. B. Go trials (pointed line) and No-Go trials (dashed line). C. Topographic maps of the No-Go minus Go difference wave for the time window of the N2d and P3d for participants in the stress group and the control group, averaged over compatibility and age. Stressed participants showed enhanced N2d and P3d amplitudes.

4.1.1.3. Impact of age on response inhibition performance and ERPs. Independent of stress induction, older participants showed a speed-accuracy trade-off compared to young participants, replicating previous results (Falkenstein et al., 2002; Vallesi, 2011). This trade-off was accompanied by alterations in ERPs, in that older participants exhibited reduced and delayed stimulus-locked (N2/d, P3/d) and reduced response-locked ERPs (Ne/_d, Pe/_d). This is in line with previous studies (Falkenstein et al., 2002; Mudar et al., 2015; Hoffmann and Falkenstein, 2011; Niessen et al., 2017). Accordingly, our results confirm the age-related deficits in neural processing related to cognitive control and error monitoring.

4.1.1.4. Independency of stress and age effects. With one exception, our results show independent impacts of age and stress on response inhibition. The interaction between age, stress, complexity and lateralization for the P3/P3d suggests age-related differences in latter stages of response inhibition processing depending on task difficulty. While acute stress enhanced P3d/P3 amplitudes at the topographical maximum, i.e. the left frontal to central region, of the component for both age groups in the easier compatible condition, the effect extended to the more complex incompatible condition in the older participants. This indicates age-dependent differences in the processing of more difficult task conditions (Vallesi, 2011), which leaves older people more prone to stress effects, as these extended to conditions or tasks in which

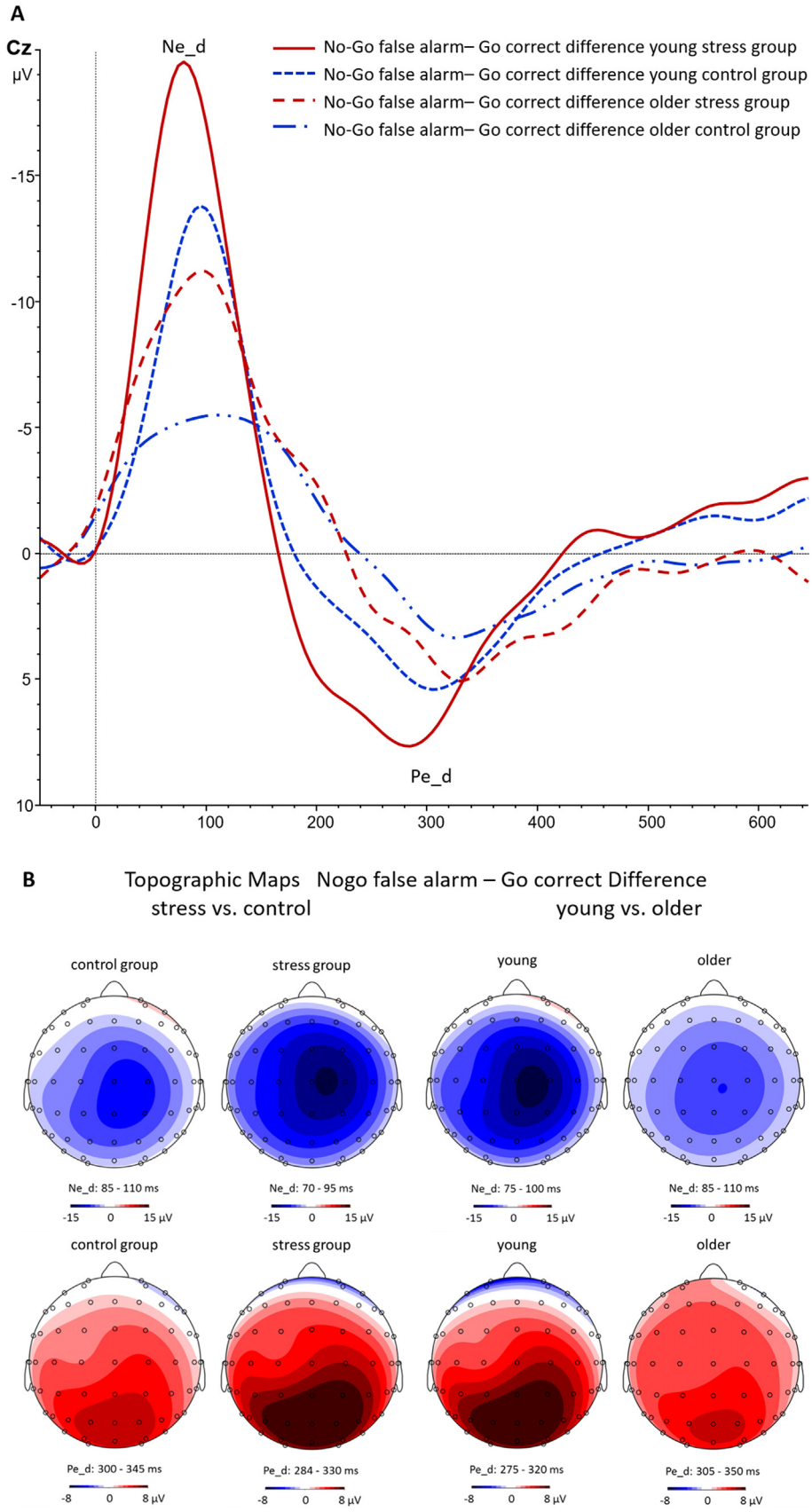

Fig. 5. A. Grand average response-locked No-Go false alarms – Go correct difference waves at Cz, for young and older participants in the stress and in the control group. B. Topographical Maps of the Ne_d and Pe_d incorrect No-Go – correct Go difference wave for the stress group and control group (left) as well as young and older participants (right). Older participants showed reduced Ne_d and Pe_d difference wave amplitudes compared to younger participants. The stress group showed enhanced Ne_d amplitudes compared to the control group.

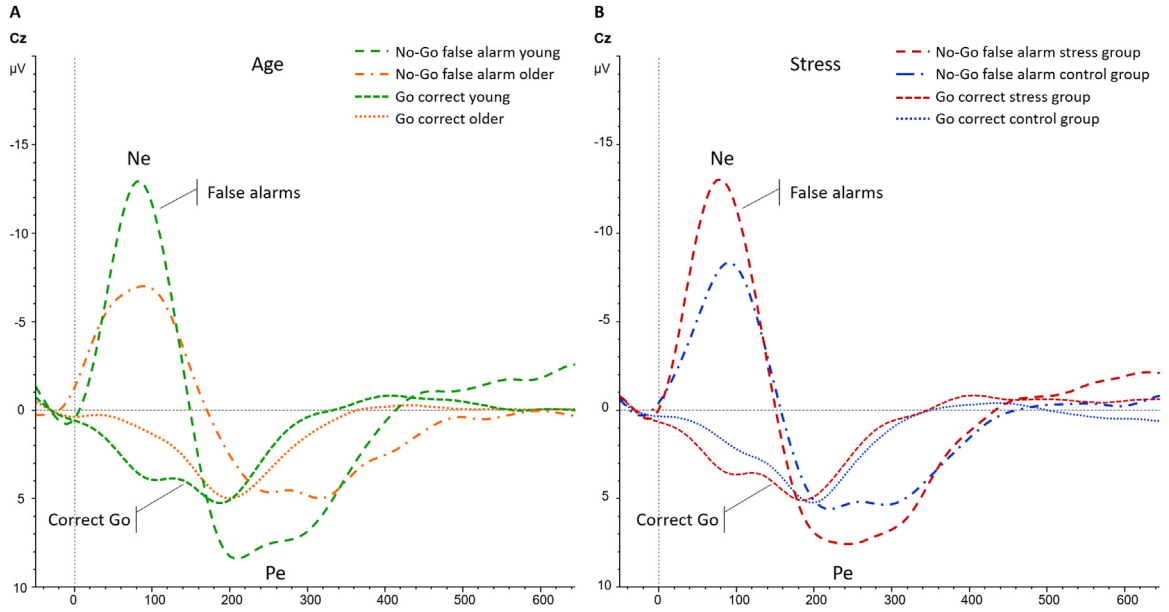

Fig. 6. Grand average response-locked ERPs for false alarm No-Go trials and correct Go trials at Cz for A. young and older participants and B. the control group and the stress group. Older participants showed a reduced Ne in incorrect No-Go and correct Go trials and a reduced Pe in incorrect No-Go trials compared to younger participants. The stress group showed enhanced Ne and Pe amplitudes for No-Go false alarms compared to the control group.

Table 4
Descriptive statistics (mean (standard deviation)) for young and older participants for the Ne_d, Ne, Pe_d and Pe amplitudes (μV) at analyzed electrode positions. For Ne and Pe, the factor Go No-Go is considered.

		age	
		young participants *M (SEM)*	older participants *M (SEM)*
Ne_d		*age x electrode position*	
Fz		− 6.718 (1.102)	− 5.187 (1.383)
FCz		− 12.281 (1.377)	− 8.271 (1.728)
Cz		− 18.098 (1.740)	− 10.970 (2.185)
Ne		*age x Go/ No-Go x electrode position*	
Fz	No-Go	− 5.403 (0.859)	− 5.056 (1.079)
	Go	− 0.434 (0.388)	1.373 (0.488)
FCz	No-Go	− 9.078 (1.053)	− 6.567 (1.322)
	Go	0.959 (0.528)	− 0.658 (0.663)
Cz	No-Go	− 11.960 (1.312)	− 6.837 (1.648)
	Go	3.111 (0.660)	0.818 (829)
Pe_d		*age x electrode position*	
Fz		3.253 (0.591)	4.275 (0.742)
FCz		5.374 (0.739)	4.549 (0.928)
Cz		7.281 (0.869)	4.165 (1.091)
CPz		8.668 (0.881)	4.385 (1.107)
Pz		8.476 (0.883)	5.146 (1.108)
Pe		*age x Go/ No-Go x electrode position*	
Fz	No-Go	1.586 (0.705)	2.836 (0.885)
	Go	− 0.115 (0.600)	0.395 (0.754)
FCz	No-Go	4.762 (0.795)	4.161 (0.999)
	Go	1.236 (0.642)	2.460 (0.806)
Cz	No-Go	7.539 (0.949)	4.255 (1.191)
	Go	2.952 (0.621)	4.047 (0.780)
CPz	No-Go	7.596 (0.919)	3.702 (1.153)
	Go	2.090 (0.551)	3.304 (0.692)
Pz	No-Go	5.884 (0.749)	3.114 (0.940)
	Go	− 0.023 (0.521)	1.016 (0.654)

Table 5
Descriptive statistics (mean (standard deviation)) for the stress group and the control group Ne and Pe amplitudes (μV) at analyzed electrode positions in the No-Go and Go condition.

	stress	
	stress group *M (SEM)*	control group *M (SEM)*
Ne		
No-Go	−9.321 (1.188)	−5.646 (1.180)
Go	1.164 (0.576)	−0.356 (0.572)
Pe		
No-Go	5.751 (0.793)	3.336 (0.841)
Go	1.682 (0.582)	1.791 (0.616)

age-specific processing or compensatory mechanisms come into play. However, this finding needs further support from future studies. Overall, stress does not seem to interfere with response inhibition processing, including possible compensatory mechanisms, in older people. Younger and older men seem to benefit equally from acute stress with regard to this executive function. However, the present findings allow no generalization to women. The knowledge on sex differences in effects of stress on executive function is still sparse. While some studies report no sex-specific findings (e.g., Plessow et al., 2012; Schwabe et al., 2013; Qi et al., 2017), other found men and women to be differently affected (e.g., Shields et al., 2016b; Merz and Wolf, 2017). Since there is some evidence for sex differences in electrophysiological correlates of response inhibition and error processing (Fischer et al., 2016; Omura and Kusumoto, 2015), and sex differences in the stress response are known (Kudielka et al., 2004), further research is needed to clarify the generalizability of our findings to women. Similarly, as our sample only included older men up to the age of 75 years, conclusions about stress effects on response inhibition and error-processing in the old-old age, and the oldest-old age are not possible.

Besides these limitations, our study and sample size were designed to discover small-to-medium effects ($\Omega^2 \geq .05$) and allow no conclusion about possible mutual effects of stress, age and compatibility with less than 5% of variance explained. Future studies might discuss and consider the possible relevance of small effects in this research context and choose their sample size accordingly. Overall, a replication of the study with a bigger sample would be desirable.

5. Conclusion

Acute stress positively influenced response inhibition accuracy and neural correlates of inhibitory control and error processing in young and older men. Neural generators reported for the event- and response-related ERPs of response inhibition mainly rely on structures that are part of the salience network. Since this network is fortified immediately after acute stress, response inhibition is enhanced in the immediate aftermath of stress, in contrast to other PFC-based executive functions.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.neuropsychologia.2018.08.020.

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