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The impact of stress on feedback and error processing during behavioral adaptation

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ABSTRACT

Stress is known to influence learning in a complex fashion. The present study aimed to examine, in how far feedback-based behavioral adaptation and electrophysiological correlates of error and feedback processing during this process are altered after acute stress. To this end, a learning task involving conditions with contingent and non-contingent monetary feedback was applied to 40 healthy young men (two groups of 20 each). The participants of one group were stressed using the socially evaluated cold pressor test. A second group of participants underwent a control procedure before the task was administered and brain activity was assessed by means of electroencephalography. The analysis focused on the feedback-related negativity (FRN) and the error-related negativity (ERN). Stressed participants did not differ from controls in learning performance. They showed, however, an elevated FRN amplitude difference between punishment and reward compared to controls. Moreover, stressed but not control participants' FRN amplitudes reflected feedback contingency after learning and thus an outcome prediction error. For response-locked potentials, no significant group differences were found. These results indicate that stress leads to a stronger recruitment of the so-called reward system in the processing of performance feedback during feedback-based behavioral adaptation.

1. Introduction

The ability to monitor and adjust one's own behavior is critical in adapting to the environment. An important process contributing to behavioral adaptation is feedback-based learning. Here, animals as well as humans gradually learn to prefer responses which are followed by positive consequences over responses that are accompanied by negative consequences. In humans, positive consequences can be primary or secondary rewards, but also cognitive feedback in the sense of “correct” or “wrong”.

In recent years it was found that stressful events in humans alter a variety of cognitive functions including learning and memory via effects of the glucocorticoid hormone cortisol (for e.g. Cahill et al., 2003; Elzinga et al., 2005; Schwabe and Wolf, 2010). Most studies to date have focused on the effect on declarative memory (for e.g. Cahill et al., 2003; Elzinga et al., 2005; Schwabe et al., 2008; Schwabe and Wolf, 2010). In contrast, non-declarative learning processes such as those involved in feedback-based behavioral adaption have only rarely been studied under stress. For instance, a study by Petzold et al (2010) reported a selective decrease in learning from negative feedback for stressed subjects compared to controls, whereas learning from positive feedback as well as general acquisition performance were not affected. The latter finding was corroborated in studies by Schwabe et al. (2010), (2012), who reported no differences for the learning curves between stress and control groups in different instrumental learning paradigms (Schwabe et al., 2010, 2012; Schwabe and Wolf, 2012).

The evaluation of reward feedback is closely linked to the neurotransmitter dopamine (DA) (Arias-Carrión et al., 2010). As a part of the so called reward system, DA neurons in the midbrain code a reward prediction error (Schultz and Dickinson, 2000) and project these error signals to the basal ganglia and to the medial prefrontal cortex including the anterior cingulate cortex (ACC). As an electrophysiological correlate of feedback monitoring in humans, prior research has identified the feedback-related negativity (FRN), a negative event-related potential (ERP) that peaks between 200 ms and 300 ms after a feedback stimulus. The FRN has its neural source in the ACC and is most pronounced after unexpected negative feedback (Bellebaum and Daum, 2008; Gehring and Willoughby, 2002; Hajcak et al., 2007; Holroyd and Coles, 2002; Miltner et al., 1997). It is closely related to the response-locked error negativity (Ne; Falkenstein, 1991) or error-related negativity

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(ERN; Gehring et al., 1993), which codes errors in performance within a time window up to 100 ms after the response and has been reported to be also generated in the ACC (Dehaene et al., 1994). The functional coupling between FRN and ERN is also evident in the process of feedback-based behavioral adaptation. After subjects have learned about action-outcome contingencies, but not before, erroneous responses elicit a significant ERN. The FRN, on the other hand, is reduced after learning, because subjects can judge their behavior already based on their response and don't have to wait for the feedback. When action – outcome associations cannot be learned, the FRN is more pronounced and the ERN absent (Bellebaum and Colosio, 2014; Eppinger et al., 2008; Holroyd and Coles, 2002). It has to be noted, however, that there are also important differences between the ERN and the FRN, for example in the underlying topography. Applying a principal component analysis, one recent study found a single central component for the ERN and two components, one central and one prefrontal one, for the FRN (Potts et al., 2010).

The influence of stress on these neural correlates of feedback and error processing has only rarely been addressed. Two studies showed decreased feedback-related theta-band activity and diminished FRN amplitudes for stressed participants, respectively (Banis and Lorist, 2012; Cavanagh et al., 2011). Concerning the influence of stress on error processing, an inconsistent pattern has emerged so far. In situations of socially evaluated stress some authors reported larger ERN amplitudes (Hajcak et al., 2005; Hajcak et al., 2004), some revealed no effects of stress on ERN amplitudes (Hsu et al., 2003) and still others found reduced ERN amplitudes in response to stress (Cavanagh and Allen, 2008). According to the reinforcement-learning (RL) theory, both FRN and ERN indirectly reflect dopaminergic activity in response to unpredicted negative events (Holroyd and Coles, 2002). Indeed, pharmacological investigations reported increased ERN amplitudes in response to an increased DA efflux, induced by administration of the DA agonist amphetamine, and attenuated ERN amplitudes for DA level decreases after the administration of a DA antagonist (De Bruijn et al., 2004; De Bruijn et al., 2006; Zirnheld et al., 2004). Studies including patients with disorders affecting the DA system also showed the importance of DA for performance monitoring. For example, Kim et al. (2006) reported diminished ERN amplitudes for schizophrenic patients. Findings of reduced FRN (Martínez-Horta et al., 2014) and ERN amplitudes were also obtained in studies with Parkinson patients (Stemmer et al., 2007) The latter result, however, emerged for both, Parkinson patients on and off medication, raising questions about the exact role of DA (Willemssen et al., 2008). Further insight into the relationship between ERN/FRN amplitudes on the one hand and the DA system on the other hand was gained in genetic studies (Ullsperger, 2010). While Frank et al. (2007) in an early study on error processing did not find a significant relationship between the COMT genotype and the ERN, two more recent studies suggested that ERN and FRN are similarly modulated by an interaction between dopaminergic gene variants and dopaminergic substances (Mueller et al., 2014; Mueller et al., 2011). High amplitudes for both components were found for subjects with either overall low or high prefrontal DA levels. Those subjects in whom the combination of genotype and pharmacological treatment resulted in medium DA levels showed reduced amplitudes.

Effects of stress on neural correlates of behavioral adaptation thus require stress-related mechanisms acting on DA and the reward system. Histological investigations in humans and animals indeed revealed the existence of cortisol binding glucocorticoid-receptors in structures which are mediating behavioral adaptation, such as ventral tegmental area (VTA) nucleus accumbens (NAcc), substantia nigra (SN) and the medial prefrontal cortex (Butts et al., 2011; Czyrak and Chocyk, 2001; Oswald et al., 2005). It was also found that stress leads to an enhanced efflux of DA, for instance in the ventral striatum and in the frontal and medial PFC (Arnsten and Goldman-Rakic, 1998; Barrot et al., 2000; Butts et al., 2011; Cho and Little, 1999; Lataster et al., 2011; Pruessner et al., 2004; Saal et al., 2003). Neural structures belonging to the reward system, and the dopaminergic system in particular, are thus affected by the stress response, and it therefore appears likely that stress affects processes related to feedback learning and behavioral adaptation (Barik et al., 2010; Czyrak and Chocyk, 2001; Diorio et al., 1993; Oswald et al., 2005).

Taken together, the few studies that have addressed effects of stress on processes related to behavioral adaptation have focused on different aspects such as feedback learning, feedback or error processing and have yielded mixed results. The present study aimed to examine the effect of a socially evaluated stressor, which is proven to activate the HPA-axis, on feedback-based behavioral adaptation as well as feedback and error processing in the same paradigm. We applied a task in which action-outcome associations could be learned in a contingent feedback condition and in which, after learning, response- and feedback-locked ERPs could be compared to those in a non-contingent feedback control condition. The task thus enabled us to examine the influence of stress on the relationship between learning, the FRN and the ERN. Due to its effects on the DA system we hypothesized that stress would enhance the post-learning amplitudes of the ERN for errors in the contingent feedback condition and of the FRN for negative feedback in the non-contingent feedback condition. In accordance with previous studies, no effect on learning performance was expected.

2. Materials and methods

2.1. Participants

40 healthy male subjects participated in this EEG-experiment. Twenty were assigned to a stress group (mean age=23.1 years, standard deviation (SD)=2.7; 18 were right-handed, 2 left-handed) and twenty to a control group (mean age=25.8 years, SD=3.4; 17 right-handed, 3 left-handed). The participants were students of the Ruhr University Bochum and had a normal or corrected-to-normal vision. Upon completion of the experiment participants received a financial reimbursement or course credit. All participants had no acute or chronic psychiatric, physiological or neurological disorder and were free from medication. Only non-smokers with a body mass index (BMI) between 19 and 28 were included in this experiment. Due to the diurnal rhythm of the stress hormone cortisol, testing always took place between 9.00 a.m. and 2.00 p.m. Participants who previously experienced the socially evaluated cold pressor-test (SECPT, see below) or who were familiar with the Asian symbols used in the learning task (see below) were excluded. All participants gave written informed consent and the study was approved by the Ethics Committee of the Faculty of Psychology at Ruhr University Bochum.

2.2. Stress induction

For the induction of acute stress in an experimental setting we used the socially evaluated cold-pressor test (SECPT) by Schwabe et al. (2008). During the SECPT, participants have to put their right hand up to their wrist into ice cold water (0–2 °C) for three minutes. At the same time, participants are recorded by a videocamera and monitored by a neutral to cold behaving experimenter. Participants are told, that the video recordings will be used for the analysis of their facial expression. During the cold water condition, participants are also instructed to watch their facial expression and behavior in a mirror positioned in front of them. The control

condition for the SECPT is a warm water condition. In this condition subjects have to place their right hand up to their wrist into warm water for three minutes (35–37 °C). In the control condition participants are neither videotaped, nor monitored by an experimenter, and they are not asked to watch themselvse in a mirror. Furthermore, the experimenter consistently acts emotionally appropriate to the participant. In the present study, blood pressure and heart rate were measured with a Dinamap vital signs monitor (Critikon, Tampa, FL) at the beginning, in the middle and at the end of the stress induction or control condition. After the treatment, participants were asked to rate how difficult, unpleasant, stressful and painful the stress or control condition was for them. In order to determine cortisol concentrations three saliva samples were collected. The first sample was taken right before the SECPT/control condition (baseline), the second 15 minutes after completion of the stress induction or control condition (+15 min post treatment) and the third directly after the learning task (see below, +45 min post treatment). The saliva was gathered by means of salivettes (Sarstedt, Nuembrecht, Germany) which were stored at −20 °C until the analysis was conducted. Free cortisol concentrations were analyzed with a commercial chemo luminescence immunoassay (CLIA; IBL International, Hamburg, Germany). Inter- and intra-assay variations were below 10%.

2.3. The learning task

The learning task was a modified version of a paradigm previously used by Holroyd and Coles (2002), Eppinger et al. (2008) and Bellebaum and Colosio (2014). During the task, participants were asked to learn stimulus-response-outcome associations and adapt their behavior in consequence. A trial of the learning task always started with the presentation of a fixation cross (variable duration between 500 and 1000 ms), followed by an Asian symbol (the imperative stimulus) with two response options, indicated by two red rectangles presented to the right and left of the symbol. This screen was presented for 500 ms. Subjects now had to choose one of the response options by pressing the left or right "strg-buttons" of a computer keyboard. After the imperative stimulus had disappeared, only the rectangles indicating the response buttons were shown for 500 ms. Maximum response time was thus 1000 ms. As soon as the subjects made their choice, the corresponding rectangle turned green and stayed on the screen for 200 ms, followed by 500 ms of a black screen. Subsequently, participants received monetary feedback, which was presented for 500 ms. For "correct" responses participants were rewarded with 20 cents, "incorrect" answers led to a punishment of 10 cents (see Fig. 1 for the sequence of events in one trial and for trial timing). Based on the feedback, the participants' task was to learn which symbol-response associations predicted reward. However, feedback validity varied between different symbols. Alltogether, six stimuli were used (referred to as A–F in the following). The feedback validity for stimuli A and B was 100%, that is, for these stimuli, a certain response (left for stimulus A and right for B) always led to reward, whereas the alternative response always led to punishment (100%-validity condition, Fig. 1). For stimuli C and D (80%-validity condition), there was a probabilistic stimulus-action-outcome association. Participants received contingent feedback in 80% of the trials and non-contingent feedback in 20% of the trials (i.e. punishment for correct responses and reward for error responses, see Fig. 1). Finally in the non-contingent feedback condition, reward or punishment was given randomly, regardless of the participants' responses (50%-validity condition, see Fig. 1). During the task, stimuli from different conditions were presented in a randomized order.

All subjects completed two versions of the learning task ("a" and "b"). The two versions only differed with respect to the stimuli presented (Fig. 1 shows the stimuli of both task versions). The structure and reward probabilities for stimuli A–F were identical. Two versions of the task were used to yield enough post-learning trials for ERP analysis. With two task versions, subjects had two chances to learn and reach the post-learning phase. At the same time, changing the stimuli made sure that subjects kept being engaged in the task. Previous studies applying similar paradigms also used more then one task version (e.g. Eppinger et al., 2008). Each version of the task was subdivided into five blocks with 60 trials each (20 per validity condition, 10 per stimulus), yielding 300 trials per version and 600 trials in total. The order of task versions was counterbalanced between subjects. Before participants started the first of the two task versions, they performed eight practice trials which did not enter the analysis.

2.4. Electroencephalographic recording

During the learning task, an Electroencephalogramm (EEG) was recorded from 30 scalp sites with silver/silver-chloride electrodes according to the international 10–20 system (F7, F3, Fz, F4, F8, FT7, FC3, FCz, FC4, FT8, T7, C3, Cz, C4, T8, TP7, CP3, CPz, CP4, TP8, P7, P3, Pz, P4, P8, PO7, PO3, POz, PO4, PO8). The amplifier used for recordings was a "Brainamp standard" amplifier (Brain Products, Gilching, Germany) with 32 channels. For recording of the data,

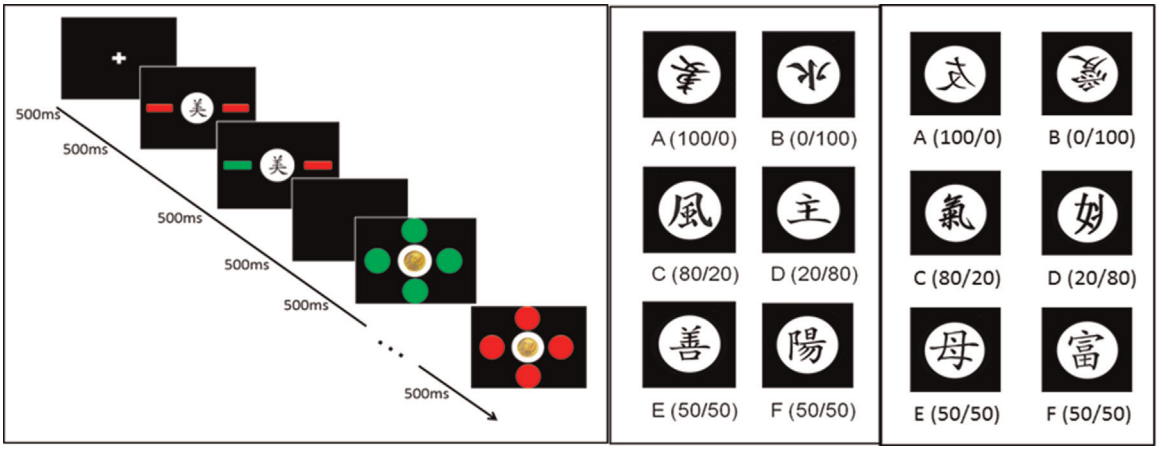

Fig. 1. The "learning" task. Left: Based on monetary feedback participants had to learn which Asian symbol was associated with the right or left response button to be either rewarded by 20 cents (surrounded by 4 green circles) or punished with 10 cents (surrounded by 4 red circles). Right: Both sets of stimuli with the reward probabilities when pressing the left or right button, respectively. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

the "Vision Recorder" software (Brain Products) was used. Data were recorded with a sample rate of 500 Hz. Two electrodes placed on the left and right mastoids served as references. The impedances were kept below 10 kΩ.

2.5. Procedure

When participants came to the lab, they were first asked to fill out an informed consent. Then subjects were prepared for the EEG recording. When impedances reached the desired values, participants gave their first saliva sample. Subsequently, subjects participated in the SECPT or control condition. The stress or control treatment was followed by an intermission of 15 min, in which the stress hormone cortisol was expected to rise. During the intermission the questionnaire concerning the subjective ratings of the treatment was administered and impedances were checked again. After exactly 15 min, the second saliva sample was collected and participants were instructed for the learning task, followed by the short training phase, before EEG recording and the learning task started. The two versions of the learning task took 13 min each. Upon completion of the task, participants gave their last saliva sample, approximately 45 min after the completion of the SECPT or control-condition. Before leaving, participants were debriefed and received either financial reimbursement or course credit. The whole procedure including EEG preparation and debriedfing took about two hours.

2.6. Analysis of behavioral and EEG data

To analyze if stress had an effect on learning, performance accuracy was compared between the stress and control groups in the different conditions of the learning task. While correct responses were always rewarded for the 100% validity condition, response accuracy was determined irrespective of the feedback in the particular trial in the 80%-validity condition. We considered responses to be correct, when subjects chose the response option that led to reward most of the time, as was done on the vast majority of previous studies on probabilistic feedback learning, because this strategy maximized reward. For the 50%-validity condition, the left and right response buttons were arbitrarily considered as "correct" for stimuli E and F, respectively, even though learning was not possible in this condition.

As outlined in the introduction, successful learning in the task differentially affects the ERN and FRN. Therefore, a learning criterion was applied for the ERP analysis, to determine, a pre- and a post-learning phase for each individual subject, and ERP analyzes were then conducted for the post-learning phase only. The learning criterion was reached for the 100% or 80%-validity conditions, when a response accuracy level of 65% was achieved and held until the end of the experiment. In order to make sure that ERP effects in the contingent feedback condition of the post-learning phase were not caused by unspecific fatigue, practice or attention effects, the experiment was also divided into two phases for the 50%-validity control condition, based on the mean learning pace in the other conditions (see Bellebaum and Colosio, 2014 and Eppinger et al., 2008 for similar procedures). Post-learning phases were determined separately for the two versions of the task (a and b) and the data were then pooled for the analysis.

Moreover, ERPs were pooled for the 100%- and 80% -validity conditions, yielding a "contingent feedback condition", to have enough error trials in the post-learning phase. For the 80%-validity condition only the contingent trials were considered, that is, those trials in which responses were followed by contingent feedback (reward for correct responses and punishment for errors), because only in these trials, outcomes were in accordance with expectations after learning. The trials of the 50%-validity condition entered the "non-contingent feedback" condition, for which also only those trials were considered for analysis, in which positive feedback followed "correct" responses and negative feedback was a consequence of "incorrect" choices. This was necessary in order to make sure that in all conditions, rewards and punishments considered for analysis were always following a particular button press for a particular stimulus.

EEG data were analyzed off-line using the Brain Vision Analyzer software (Brain Products, Gilching, Germany) and Matlab (Mathworks, Natick, MA, USA). At first, vertical eye movement and blink artifacts were corrected by conducting an independent component analysis (ICA) on the raw data on single subject level. Within this procedure, the data of the 30 scalp electrodes were split into temporally independent and spatially fixed components (Lee et al., 1999). All 30 components of every single subject were scanned to find one component with a symmetric, frontally positive topography, which could represent eye movements. This component was then removed from the raw data by means of the ICA back transform procedure. The resulting signal was checked for remaining blink artifacts, and only if artifacts were still seen, a second component was removed. Then a high-pass filter of 0.5 Hz and a low-pass filter of 40 Hz were applied to the back-transformed data. Afterwards, segmentation was accomplished for each subject.

For the analysis of the FRN, segments were built for punishment and reward in the contingent and non-contingent feedback conditions in the post-learning phase. The segments started 200 ms and ended 800 ms after the presentation of monetary feedback. During artifact rejection segments with a voltage range higher than 150 μV were again excluded. The baseline correction was performed relative to the average amplitude in the 200 ms before feedback presentation. Afterwards, the average signal was calculated for each subject in each condition. The FRN was scored by means of a peak-to-peak analysis at electrode position Fz. First, the maximum negative peak between 180 ms and 350 ms after monetary feedback was determined. Then the maximum positive peak amplitude in the time window before the negative peak with a minimal latency of 150 ms was subtracted from the negative peak amplitude.

For the ERN, the segments ranged from 200 ms before to 400 ms after the response. Segments were formed separately for false and correct responses in the contingent and non-contingent feedback conditions in the post-learning phase. Following the segmentation, data were baseline corrected relative to the period from −200 ms to −100 ms before the response. During the subsequent artifact rejection, segments with a voltage range higher than 150 μV were excluded. Then the data were averaged for each subject and each condition. The ERN was defined as the maximum negative peak amplitude in the time window from −50 to 100 ms relative to response onset at electrode site Fz for error trials. For comparison, a "correct related negativity" (CRN) was scored in the same way for correct trials.

2.7. Statistical analysis

The effect of stress induction on the cortisol level, as well as systolic and diastolic blood pressure was analyzed by means of a repeated-measures analysis of variance (ANOVA) with the three level factor Time (first to third assessment) and the between-subjects factor Treatment (stress vs. control). Between-group comparisons of the ratings were conducted by means of two-sample *t*-tests.

To compare performance accuracy between the stress and control groups, a repeated measures ANOVA was conducted. The within-subjects factors were Learning condition and Block. To match the analysis of the ERP data, trials of the 100%- and the 80%-validity conditions were pooled also for behavioral data analysis, yielding the conditions contingent vs. non-contingent. The factor Block comprised

5 steps (blocks 1–5). The between-subjects factor was Treatment (stress vs. control). Greenhouse-Geisser corrections were performed where appropriate. Finally, to examine stress effects on the ERN or FRN, repeated-measures ANOVAs comprising the factors Contingency (contingent vs. non-contingent), Accuracy (error vs. correct) for the ERN or Valence (reward vs. punishment) for the FRN and the between-subjects factor Treatment (stress vs. control) were conducted. All significant interactions were further analyzed by post hoc *t*-tests. For all statistical tests the threshold for significance was set to $p < .05$. For post-hoc *t*-tests, a Bonferroni correction was applied dividing this value by the number of tests calculated.

3. Results

3.1. Stress data

In response to the stress induction, significant changes in the subjective stress ratings, in blood pressure and in the salivary cortisol concentration were detected for stressed participants compared to controls. However, one participant failed to show a stress-induced cortisol increase (cortisol level increase of less than 1.5 mnol/l above baseline; (Miller et al., 2013) and therefore had to be excluded from all data analyzes. In one control subject, the baseline saliva sample could not be analyzed. As data from the other two samples were available, this subject was not excluded from further data analysis (see below for details). The stress condition was rated as significantly more difficult, more unpleasant, more stressful and more painful compared to the control condition (all $ts(37) > 10.21$; all $p < .01$; see Table 1 for the mean ratings of the two groups). Furthermore, for diastolic as well as systolic blood pressure significant Time by Treatment interactions were found: systole $F(2,36) = 7.70$ $p < .01$; diastole $F(2,36) = 7.11$, $p < .01$; see Table 1. In the resolution of these interactions, a significantly higher systolic blood pressure was found in the stress group compared to the control group for all three assessments (pre-treatment: $t(37) = 3.71$; $p < .01$; 15 min. post-treatment: $t(37) = 5.62$; $p < .01$; 45 min post-treatment: $t(37) = 5.95$; $p < .01$), with the difference being strongest for the 15 and 45 min post-treatment assessments. A similar pattern was found for diastolic blood pressure. Again, the values were higher for stressed participants over all three assessments (pre-treatment: $t(37) = 3.77$; $p < .01$; 15 min post-treatment: $t(37) = 6.08$; $p < .01$; 45 min post-treatment: $t(37) = 4.15$; $p < .01$). Here, the largest difference between groups emerged for the 15 min post-treatment measurement.

Table 1
Means and standard errors (SEM) of subjective ratings and blood pressure for the stress and control group.

	Control	Stress
Subjective assessments		
Difficulty	1.5 ± 1.9	51.6 ± 8.0***
Unpleasantness	8.0 ± 4.2	54.7 ± 6.7***
Stressfulness	4.0 ± 1.8	46.3 ± 6.9***
Painfulness	2.0 ± 1.6	63.7 ± 6.0***
Blood pressure in (mm Hg)		
Systole		
Start of treatment	129.8 ± 3.3	145.2 ± 2.5***
During treatment	123.3 ± 3.7	151.2 ± 3.4***
End of treatment	122.8 ± 3.0	151.2 ± 3.8***
Diastole		
Start of treatment	73.4 ± 1.9	82.2 ± 1.5***
During treatment	71.9 ± 1.9	88.2 ± 2.0***
End of treatment	70.6 ± 2.0	84.0 ± 2.6***

Data represents means for stress vs. control group with ± SEM,
*** $p < .001$, Bonferroni corrected.

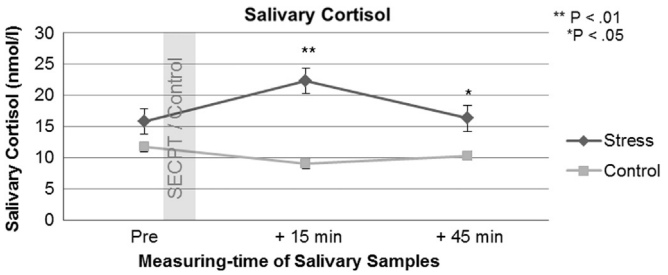

Fig. 2. Average levels of salivary cortisol concentration for stressed and control-participants (± standard error). The "pre-measurement" represents the baseline sample collected directly before the SECPT or control condition was implemented, whereas the other two samples were collected 15 min and 45 min after exposure. A significant increase of cortisol in response to the SECPT occurred. *P*-values refer to between-group differences.

The group analysis of cortisol was based on 19 participants of the stress group and 19 controls (one non-responder and missing data for T1, respectively, see above). The ANOVA revealed a significant main effect of Treatment ($F(1,35) = 15.01$, $p < .01$) and a trend towards a main effect of Time ($F(2,35) = 2.82$, $p = .07$) which was qualified by a significant interaction of both factors ($F(2,35) = 10.58$, $p < .01$). Post-hoc *t*-tests revealed that cortisol levels were descriptively, but not significantly enhanced in the stress relative to the control group for baseline samples (first assessment; ($t(36) = 2.02$; $p = .05$). Fifteen minutes after stress induction, however, the concentration of salivary cortisol was significantly different between the stress-compared to the control-group ($t(36) = 5.11$; $p < .01$; see Fig. 2). Forty-five minutes after treatment, and thus after the completion of the learning task, the difference between groups was less pronounced, but still significant ($t(36) = 2.53$; $p < .02$; see Fig. 2). The cortisol level of the control participant for whom data of the baseline assessment was missing was well within the control participants' range. His cortisol levels were 0.02 and 0.01 standard deviations below the mean of the other controls for time points 2 and 3, respectively. Thus, he was not excluded from further data analyzes.

3.2. Behavioral data

As the subjects only had 1000 ms to respond we analyzed the number of misses first. On average, the number of misses across both task versions was 4.9 and thus very low. Only five subjects had more than 10 misses, with the largest number being 17. Thus, none of the participants had to be excluded due to the number of misses.

Five subjects (three control subjects and two stressed participants) did not reach the learning criterion in any of the contingent feedback conditions in both task versions. As we were interested in the general effect of stress on feedback learning, these subjects were still included in the analysis of the behavioral learning data and only the stress non-responder was excluded. Fig. 3 shows the learning curves for the contingent and non-contingent feedback conditions in stressed and control subjects. A repeated-measures ANOVA revealed no significant effects of Treatment or interactions with treatment (all $ps > .05$). Main effects of Contingency ($F(1,37) = 83.65$, $p < .01$) and Block were found (linear trend: $F(1,37) = 36.38$, $p < .01$), which were qualified by a significant two-way interaction (linear trend: $F(1,37) = 22.38$ $p < .01$), indicating that performance accuracy increased across blocks for contingent (linear trend: $F(1,38) = 61.98$, $p < .01$), but not for non-contingent feedback (linear trend: $F(1,38) = 0.25$, $p = .88$).

3.3. EEG data

As we expected to see specific effects of treatment (stress vs. control) in the contingent and non-contingent feedback conditions

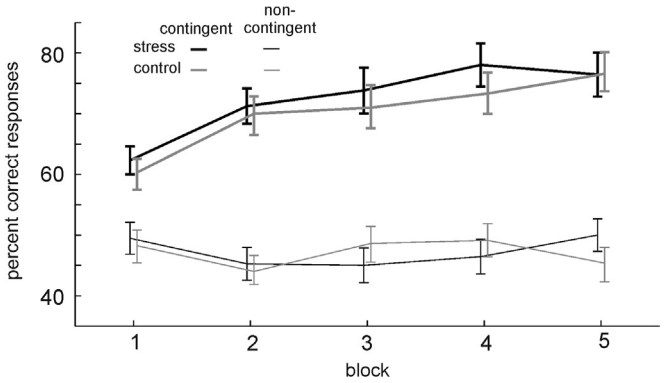

Fig. 3. Behavioral data. Average learning accuracy in the contigent and non-contingent task condition across blocks (means and SEM) over both task versions, separated into stress and control condition. No difference in learning performance accuracy was found between stress and control group. Each block comprised 60 trials.

Table 2
Means and SEM of the number of trials entering analysis for the FRN and the ERN/CRN (range in brackets).

	Stress	Control
FRN		
Non-contingent punishment	37.6 ± 2.9 (23–64)	38.1 ± 2.7 (21–64)
Non-contingent reward	30.1 ± 2.5 (16–50)	31.1 ± 2.7 (11–57)
Contingent punishment	25.6 ± 2.6 (11–43)	28.7 ± 2.7 (10–47)
Contingent reward	182.9 ± 15.3 (83–269)	180.5 ± 15.1 (68–278)
ERN/CRN		
Non-contingent error	75.8 ± 5.2 (40–126)	73.7 ± 5.3 (43–124)
Non-contingent correct	64.5 ± 4.5 (35–99)	65.6 ± 5.2 (29–112)
Contingent error	29.3 ± 2.6 (13–47)	31.6 ± 2.8 (11–48)
Contingent correct	199.9 ± 18.0 (68–296)	198.2 ± 17.9 (68–308)

in the post-learning phase, the five non-learners were excluded from all ERP analyzes in addition to the one stress non-responder, yielding 17 participants in each group. For the remaining subjects, the minimum number of trials entering FRN or ERN analysis was 11. Table 2 lists the average number of trials included in the different groups and conditions for the FRN and ERN. To further validate the main effects, analyzes were repeated in a reduced sample of 13 stressed and 14 control participants, who all had at least 16 trials in every experimental condition (see footnotes below).

3.4. FRN

In Fig. 4, the mean ERP amplitudes for monetary punishment and reward in the post-learning phase are displayed for the stress and control groups, separately for the contingent and non-contingent feedback conditions. Table 3 lists the average FRN peak amplitudes in all conditions for the stress and control groups. Apart from the frequently reported main effect of Valence (larger FRNs after punishment than reward: $F(1,32)=32.02$; $p<.01$) the repeated-measures ANOVA[1] on FRN amplitudes resulted in a significant main effect of Treatment ($F(1,32)=9.39$; $p<.01$), indicating higher FRN amplitudes in stressed participants than in controls, and in a significant two-way (Treatment by feedback Valence; $F(1,32)=10.17$; $p<.01$) and, most importantly, three-way interaction (Treatment, Valence and Contingency; ($F(1,32)=4.43$; $p=.04$). The only other effect reaching significance was the interaction between Valence and Contingency ($F(1,32)=4.73$; $p=.04$), indicating that the FRN amplitude difference between punishment and reward was generally larger for the non-contingent feedback condition.

To resolve the three-way interaction, separate ANOVAs were conducted for stressed and control subjects involving the factors Valence and Contingency. Only for the stressed ($F(1,16)=6.73$; $p=.02$), but not for the control participants ($F(1,16)<0.01$; $p=.95$) a significant interaction emerged. The post-hoc t-tests yielded significantly larger FRNs in response to punishment than reward for the non-contingent feedback condition ($t(16)=-5.50$; $p<.01$) and a trend towards a significant difference in the same direction for the contingent feedback condition ($t(16)=-2.39$; $p=.03$) in stressed participants.

3.5. ERN/CRN

Fig. 5 shows the grand-average response-locked ERPs for correct and incorrect responses during the post-learning phases after stress or control treatment, separately for the contingent and the non-contingent feedback conditions. Peak amplitudes for the ERN/CRN are listed in Table 4. The ANOVA[2] did not yield a significant main effect of Treatment ($F(1,32)=0.52$; $p=.48$). The interaction between treatment and accuracy approached significance ($F(1,32)=3.62$; $p=.07$) – the amplitude difference between ERN and CRN was descriptively larger in stressed than in control participants due to higher positive amplitudes for the CRN. This difference was, however, mainly driven by one participant, who scored very high on the CRN amplitude. Removing this participant from the analyzes reduced the mean amplitude value for the stress group to 2.16 μV and the SEM to 1.03 μV (see Table 4 for comparison). The main effect of Accuracy indicates that the ERN amplitude following errors is generally larger than the CRN amplitude following correct responses ($F(1,32)=49.71$; $p<.01$). The analysis also revealed a significant interaction between Accuracy and Contingency ($F(1,32)=51.31$; $p<.01$). When the contingent and non-contingent feedback conditions were considered separately, significant differences between ERN and CRN were only seen for contingent feedback ($t(33)=-7.32$; $p<.01$; $t(33)=1.56$; $p=.13$ for the non-contingent feedback condition). The three-way interaction involving all factors did not approach significance ($F(1,32)=1.76$; $p=.20$).

4. Discussion

The aim of this work was to investigate the impact of socially evaluated stress on feedback-based behavioral adaptation and on electrophysiological correlates of feedback and error processing. Two groups of participants accomplished a feedback-based learning task during which performance accuracy could be monitored based on feedback and, after subjects had learned, based on behavioral responses. Before performing the learning task, participants of one group were exposed to a stress condition, whereas

[1] The FRN analysis for only those subjects with more than 16 trials in every condition yielded similar effects of Treatment. Again, a significant main effect of Treatment ($F(1,25)=7.72$; $p=.01$) and a Treatment by Valence interaction ($F(1,25)=5.04$; $p=.03$) emerged. For the three-way interaction a trend was found ($F(1,25)=3.35$; $p=.08$).

[2] Also for the ERN, the analysis with the reduced sample revealed a comparable result pattern. No effects of Treatment were found (for the main effect of and all interactions with Treatment: all $F(1,25)<3.43$; all $p>.05$).

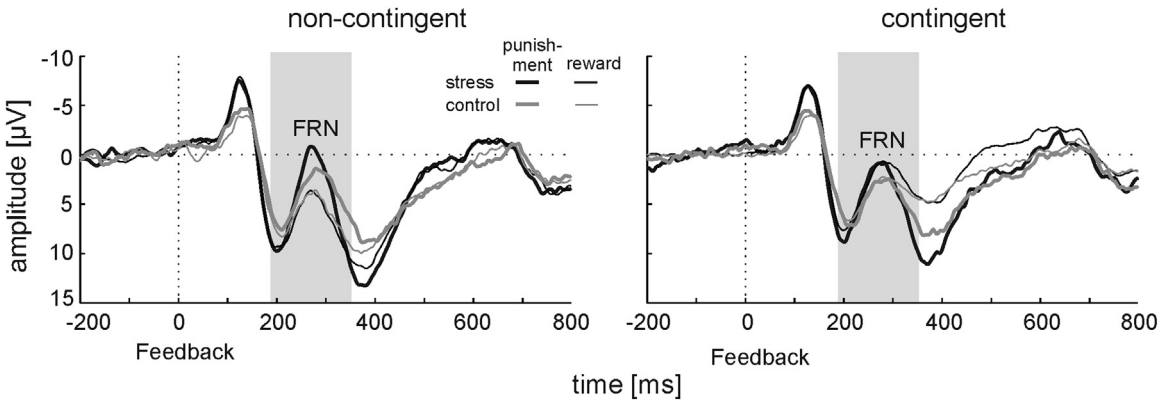

Fig. 4. Grand-average ERPs from electrode position Fz for contingent and non-contingent reward and punishment. The amplitudes represent data of the post-learning phase. The black lines illustrate the electrophysiological reaction of the stress group and the grey lines the ERPs of the control group. The thick lines indicate punishment and the thin lines reward.The bars shaded in grey indicate the time interval in which the FRN was analyzed. The analysis revealed significantly larger FRN differences between punishment and reward in the stress than in the control group, particularly in the non-contingent feedback condition (see Results section for details).

Table 3
Means and SEM for FRN amplitudes in the stress and control group in the different conditions (in μV).

	Stress	Control
Non-contingent punishment	−13.06 ± 1.33	−7.65 ± 1.01
Non-contingent reward	−8.41 ± 1.08	−6.73 ± 0.85
Contingent punishment	−11.09 ± 1.24	−6.45 ± 0.90
Contingent reward	−9.28 ± 0.95	−5.57 ± 0.57

participants of the other group underwent a stress free control condition. Cortisol concentration and blood pressure measurements demonstrated that stress induction was successful (Kirschbaum and Hellhammer, 1994). A numerical difference of cortisol concentrations between groups already at the baseline assessment may be due to the reserved behavior of the experimenter up to the stress condition. Importantly, a significant increase in cortisol concentration from baseline was then achieved only in the stress condition. Concerning processes related to behavioral adaptation, the learning paradigm did not reveal an effect of stress on feedback-based learning performance. There were, however, differences in feedback processing between groups in the post-learning phase: The FRN amplitude difference between negative and positive feedback was generally larger in the stressed than in the control subjects. Moreover, only in the stressed participants, post-learning FRN amplitudes reflected feedback contingency, with larger differences between punishment and reward for non-contingent feedback. Although for response-locked potentials the amplitude difference between ERN (following errors) and CRN (following correct responses) was also numerically larger in stressed than in control subjects, this effect was not significant.

The processing of feedback in general and the occurrence of the FRN in particular have been linked to the dopaminergic system (Frank et al., 2004; Holroyd and Coles, 2002; Schultz and Dickinson, 2000). Concerning the exact role of DA in performance monitoring, recent studies suggest that the relationship between ERN/FRN on the one hand and prefrontal DA level on the other hand follows a U-shaped function (Mueller et al. 2011; Mueller et al., 2014), based on the dual state theory of prefrontal DA effects (Durstewitz and Seamans, 2008). Although we did not control for the dopamine level in the participants of our study, it seems likely that the stress effects on feedback processing observed in the present study were mediated by stress influences on dopamine, as in humans as well as in animals, stress supposedly leads to an increased release of DA (Cho and Little, 1999; Oswald et al., 2005; Saal et al., 2003). Prior research reported decreased feedback-related theta-band activity and diminished FRN amplitudes for stressed participants, respectively (Banis and Lorist, 2012; Cavanagh et al., 2011). These studies are, however, not directly comparable to the present study, as the stressors were administered while subjects engaged in a task and ERPs were recorded. The stressors might therefore have directly interfered with performance and/or processing. With respect to the postulated relationship between prefrontal DA level and ERN/FRN (Mueller

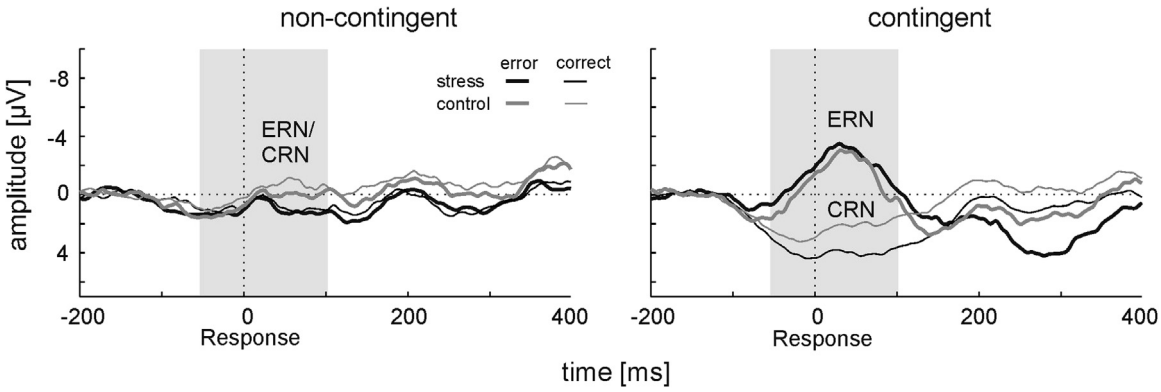

Fig. 5. Grand-average ERPs from electrode position Fz for correct and erroneous responses during contingent and non-contingent feedback conditions. The amplitudes represent data of the post-learning phase. The black lines illustrate the electrophysiological reaction of the stress group and the grey lines the ERPs of the control group. The thick lines represent the reaction to errors (ERN) and the thin lines indicate reactions to correct responses (CRN).The bars shaded in grey indicate the time interval in which the ERN/CRN was analyzed.

Table 4
Means and SEM for ERN (errors) and CRN (correct responses) amplitudes in the stress and control group in the contingent and non-contingent feedback conditions (in μV).

	Stress	Control
Non-contingent error	−1.21 ± 0.91	−1.52 ± 0.51
Non-contingent correct	−1.54 ± 0.78	−2.22 ± 0.49
Contingent error	−5.24 ± 0.98	−4.60 ± 0.70
Contingent correct	3.44 ± 1.60	0.89 ± 0.74

et al. 2011; Mueller et al., 2014) the enhanced FRN amplitudes of the present study might be a result of overall high prefrontal DA levels induced by the stress-induced high cortisol levels compared to medium DA levels in non-stressed participants. This interpretation is also in line with the behavioral finding of reduced negative learning induced by stress (Petzold et al., 2010), as DA was shown to foster positive learning (Frank et al., 2004).

The work by Mueller et al. (2014) on the role of DA for the FRN focused on the "FRN effect", that is, the amplitude difference between negative and positive feedback, which was also modulated in the present study. It is thus not clear, if DA affects the processing of negative or positive feedback or both. The pattern of FRN amplitudes observed in the present study suggests that the FRN following negative feedback was specifically enhanced after stress compared to controls, which, at first sight, doesn't seem to fit to the reduced negative learning after stress described by Petzold et al. (2010). Importantly, FRN amplitude differences for punishment and reward were not only generally enhanced for stressed participants but also reflected feedback contingency, in contrast to those in control subjects. Elevated FRNs for punishment were only found in the non-contingent feedback condition, during which participants could not learn to predict the outcomes of their choices and therefore had to continue to rely on feedback to monitor their performance. In contrast, subjects could predict the outcomes of their choices in the contingent feedback condition in the post-learning phase. The non-contingent and contingent feedback conditions were thus associated with high and low prediction errors, respectively. It has to be noted, however, that this specific effect in stressed participants was somewhat reduced in a reduced sample of participants with 16 or more trials in every condition.

The FRN has been postulated (Holroyd and Coles, 2002) and shown (Bellebaum and Daum, 2008; Hajcak et al., 2007; Holroyd et al., 2009) to reflect a negative outcome prediction error, underlining its functional meaning as a teaching signal in reinforcement learning. The finding of the present study that the FRN reflects feedback contingency particularly in stressed participants thus appears to suggest that reinforcement learning mechanisms were strongly engaged in the stressed subjects. As outlined above, the processing of and learning from performance feedback has been linked to the DA system and to regions receiving DA projections like the striatum and the ACC. Recent evidence suggests, however, that structures related to declarative memory formation such as the hippocampus and medial temporal lobe can also be involved in feedback processing (Foerde et al., 2013; Foerde and Shohamy, 2012). The hippocampus has been shown to code reward prediction errors in parallel to the basal ganglia, probably propagated via DA projections from the midbrain (Dickerson et al., 2011). Both structures can work together in feedback- and non-feedback-based probabilistic classification learning (Foerde and Shohamy, 2011). At the same time, the relative contribution of the BG and the hippocampus/medial temporal lobe in this type of learning can vary during task performance and based on the applied learning strategy (Poldrack et al., 2001; Shohamy et al., 2004). In the context of the present study it is of particular relevance that stress leads to a shift towards more procedural, striatum-based learning strategies during classification learning, while hippocampus-dependent learning is impaired (Schwabe and Wolf, 2012). Also in the present study, declarative and non-declarative mechanisms may have contributed to learning, especially given the deterministic nature of parts of the to-be-learned associations. It is conceivable that stress led to an enhanced involvement of the striatum and thus of non-declarative learning compared to controls, which might be reflected in the enhanced contingency effect of the FRN. In fact, recent source analysis and combined ERP-imaging studies revealed that, in addition to the source in the ACC, the striatum contributes to FRN generation (Becker et al., 2014; Carlson et al., 2011; Foti et al., 2011). A stronger role of hippocampus-dependent learning may, on the other hand, account for the absence of a feedback contingency effect in the control subjects and for the comparable learning curves in stressed and non-stressed participants. It has to be noted, however, that previous studies with similar paradigms found a contingency effect in young healthy subjects without a stress influence (e.g. Eppinger et al., 2008; Bellebaum and Colosio, 2014). The negative finding on learning performance is in line with previous investigations, which also reported comparable feedback-based acquisition of stimulus–response associations in stressed and control participants (Petzold et al., 2010; Schwabe et al., 2010, 2012; Schwabe and Wolf, 2012).

A current debate in the FRN literature relates to the question whether the "FRN effect" is caused by processes specific for negative or positive feedback processing or both. Although the present study was not designed to address this question, it is interesting to note that the contingency effect in stressed participants appears to be caused by both an enhanced (i.e. more negative) FRN for punishment and a decreased (i.e. less negative) FRN for reward. Stress thus appears to affect the processing of feedback in general, which would be in line with the interpretation of a general shift towards more striatum-based learning under stress, as the striatum is involved in learning from both positive and negative prediction errors (Seymour et al., 2007).

Further stress effects may be related to processing in the prefrontal cortex. In a study focusing on the topography of ERP components, Potts et al. (2010) found a central and a prefrontal component underlying the FRN. They assumed that the ACC, reflected in the central component, is responsible for the evaluation of an event with respect to the failure of reaching motivational goals and that the prefrontal component is relevant for the adjustment of reward expectations based on prediction errors. It is known that acute stress influences processing in the prefrontal cortex (Arnsten and Goldman-Rakic, 1998; McEwen, 2007). During the non-contingent learning condition reward expectations are continuously updated and stress might thus specifically influence this updating process.

Interestingly, in the same study (Potts et al., 2010) differences between ERN and FRN were described concerning the underlying sources. For the ERN only one central component was found. This could help to explain the missing stress effect on the ERN in the present study, as it hints at partially separate processes underlying ERN and FRN. Prior research on the effect of stress on ERN amplitudes revealed a heterogenic picture (Cavanagh and Allen, 2008; Hajcak et al., 2004; Hajcak et al., 2006; Hsu et al., 2003; Tops et al., 2006). In the present study, the difference between ERP amplitudes for errors and correct responses was numerically larger in stressed than in non-stressed subjects. This difference was, however, far from reaching significance, so that it can be concluded that feedback processing was more strongly affected by stress in the present study. Future studies will have to further elucidate similarities and differences between stress effects on neural mechanisms of response- and feedback-monitoring.

Finally, the finding of enhanced FRN amplitudes might be related to compensatory neuronal networks. For instance, it was shown that the amygdala is especially relevant for the processing of negative feedback and has reciprocal connections with the ACC (Kita and Kitai, 1990; Yacubian et al., 2006). Moreover, it was revealed that DA storage capacity in the amygdala correlates positively with the blood-oxygen level dependent signal observed in the amygdala and the ACC during the processing of negative feedback (Kienast et al., 2008). It thus seems that there is a positive correlation between DA release in the amygdala and increased processing of negative feedback.

As a limitation it has to be mentioned that the present study does not address the mechanisms of stress effects on the processes of behavioral adaptation. Although it appears likely that the increased FRN after stress is caused by DA, further research is clearly needed to clarify how socially evaluated stress affects the processing of prediction errors in interaction with DA.

A further shortcoming is that only men were examined. Given the known influence of gender, menstrual cycle and oral contraceptives on the hypothalamus-pituitary-adrenal axis (Kirschbaum et al., 1999), the conclusions we can draw from the present study on possible stress effects on behavioral adaption or on electrophysiological correlates of adaptation in women are limited (Merz et al., 2012, 2013).

In conclusion, socially evaluated stress seems to foster a generally stronger processing of negative feedback, reflected in significantly larger FRN amplitudes. A stronger representation of feedback contingency, and thus reward prediction errors, in the FRN of stressed participants is an indicator of a general shift towards more striatum-based non-declarative learning induced by stress.

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