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# Punishment has a persistent effect on error-related brain activity in highly anxious individuals twenty-four hours after conditioning

Anja Riesel[a,b,*], Norbert Kathmann[b], Verena Wüllhorst[c], Iulia Banica[d], Anna Weinberg[d]

[a] Universität Hamburg, Germany
[b] Humboldt-Universität zu Berlin, Germany
[c] Technische Universität Dresden, Germany
[d] McGill University, Canada

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ABSTRACT

The ability to detect and respond to errors, and to subsequently recruit cognitive control to remediate those errors, is critical to successful adaptation in a changing environment. However, there is also evidence that, for anxious individuals, this error signal is enhanced, highlighting affective and motivational influences on error monitoring. These individual differences arise as a function of both genetic influences and learning experiences. In this study, we examined punishment-based modulation of the error-related negativity (ERN) in high and low anxious individuals across two days. Twenty-two low- and 25 high-anxious participants performed a Flanker task in a standard and punishment condition in three phases (Day one: acquisition and extinction 1, Day two: extinction 2). During the acquisition phase, errors in one condition were punished by a loud noise. This was followed by an immediate extinction phase (extinction 1), during which errors were no longer punished, and an identical extinction phase 24 h later (extinction 2). Only high anxious individuals showed increased ERN amplitudes in the punishment compared to the standard condition. This effect was not modulated by phase and was observed across acquisition and both extinction phases, such that anxious individuals appeared not to learn that the threat value of formerly-punished errors had changed in the course of the experiment. These data suggest that environmental factors (i.e., punishment) can have a persistent effect on the magnitude of the ERN, particularly for anxious individuals. This may point to a pathogenic mechanism linking learning experiences with the development of overactive error-monitoring in anxiety.

## 1. Introduction

Anxiety is an adaptive multilevel response to threat or potential threat. Anxiety functions to sharpen the senses, to provide an alarm signal, trigger adaptive behavior, and ultimately to promote survival. In threatening situations, errors can have extreme consequences; consistent with this, error commission leads to physiological changes in skin conductance, heart rate (Hajcak et al., 2003a), startle reflex (Hajcak and Foti, 2008; Riesel et al., 2013) and pupil dilatation (Critchley et al., 2005). These changes suggest defensive preparation and are frequently seen to be exaggerated in anxious individuals. Thus, it is not surprising that states and traits associated with anxiety are characterized by increased error-related brain activity (Riesel, 2019; Weinberg et al., 2015a; Weinberg et al., 2012).

Error monitoring is frequently studied by examining the error-related negativity (ERN, Falkenstein et al., 1991; Gehring et al., 1993), a negative-going ERP component that peaks over frontocentral electrodes shortly after an erroneous response. Studies using fMRI (Ridderinkhof et al., 2004), EEG (Dehaene et al., 1994; Hoffmann and Falkenstein, 2010) or a combination of both methods (Debener et al., 2005; Grutzmann et al., 2014) point to the anterior cingulate cortex (ACC) as a neuronal generator of the ERN.

The ERN is sensitive to emotional and motivational states, and in particular appears amplified in conditions of heightened error significance. Accordingly, experimental modulations that emphasize accuracy over speed (Falkenstein et al., 2000; Gehring et al., 1993), provide monetary incentives (Hajcak et al., 2005) or external performance evaluation (Hajcak et al., 2005), and manipulations that include punishment (Riesel et al., 2012) lead to enhanced ERN amplitudes. In addition, experimentally-induced emotional states that are associated with defensive motivation can lead to increased error monitoring, such as induction of worry (Moran et al., 2015), helplessness (Pfabigan et al.,

* Corresponding author at: Universität Hamburg, Institute of Psychology, Department of Clinical Psychology, Von-Melle-Park 11, 20146 Hamburg, Germany.
*E-mail address:* anja.riesel@uni-hamburg.de (A. Riesel).

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2013) and negative affect (Olvet and Hajcak, 2011; Wiswede et al., 2009a; Wiswede et al., 2009b).

In addition to these situational and motivational influences, stable individual differences in personality and psychopathology associated with affective distress and anxiety have also been linked to alterations in error-related brain activity (Weinberg et al., 2012). More specifically, increased ERN amplitudes have been reported in OCD (e.g., Endrass and Ullsperger, 2014; Riesel, 2019) and anxiety disorders (e.g., Gillan et al., 2017; Weinberg et al., 2015a; Weinberg et al., 2012) as well as in nonclinical individuals showing high OCD traits (e.g., Gründler et al., 2009; Hajcak and Simons, 2002) or trait anxiety (Aarts and Pourtois, 2010; Meyer et al., 2015; for reviews, see Weinberg et al., 2012). Further, alterations in error monitoring in OCD and anxiety seem to be independent of symptom state, and persist after symptom reduction and remission (Hajcak et al., 2008; Kujawa et al., 2016; Riesel et al., 2015). Moreover, increased ERN amplitudes can also be observed in individuals at increased risk to develop OCD or anxiety (Carrasco et al., 2013; Riesel et al., 2011; Riesel et al., 2019b) and have been shown to predict the onset of anxiety symptoms (Meyer et al., 2017; Meyer et al., 2015). These data, combined with evidence that the ERN is relatively stable and trait-like (Olvet and Hajcak, 2009a; Weinberg and Hajcak, 2011) and subject to substantial genetic influence (i.e., approximately 50%; Anokhin et al., 2008; Burwell et al., 2016), suggest that increased ERN amplitudes represent a genetically-mediated risk marker or endophenotype that indicates *vulnerability* for anxiety across different diagnoses (Olvet and Hajcak, 2008; Riesel et al., 2015; Riesel et al., 2011).

However, the phenotype that corresponds most closely with increased ERN amplitudes is still a subject of debate. There is meta-analytic evidence for a greater effect size for the relationship between the ERN and anxious apprehension specifically, compared to more general or mixed anxiety measures (Moser et al., 2013; Saunders & Inzlicht, Preprint). Alternatively, some have proposed that the magnitude of the ERN reflects the degree to which errors are evaluated as threatening (Weinberg et al., 2012; Weinberg et al., 2016). Further Uncertainty (Cavanagh and Shackman, 2014) and checking (Weinberg et al., 2015b) have also been proposed to be associated with increased ERN amplitudes. Importantly, all explanations suggest that enhanced ERN amplitudes reflect a transdiagnostic phenomenon related to anxiety that is not limited to specific categorical diagnoses.

Taken together, variation in psychophysiological responses to errors seems to be shaped by both genetic and contextual influences. Further, a substantial portion of the variance in error monitoring can also be attributed to broader environmental factors. For instance, punitive and/or overprotective parental behaviors have been linked to the development of increased neural error signals (Banica et al., 2019; Brooker and Buss, 2014; Meyer et al., 2019; Meyer et al., 2015). However, these studies tend to rely on rather global assessments of parenting, and cannot describe whether the effects are due to specific parenting behaviors in response to mistakes, or whether they reflect stress associated with early experiences of an adverse environment (Khan et al., 2018; Lackner et al., 2018). Additionally, the effects of parenting in these studies might be confounded with genetic influences on the ERN—that is, parents who behave in a more punitive and/or overprotective way with their offspring might themselves have a larger ERN. However, another possibility is that individuals who are genetically predisposed to anxiety might then be more *susceptible* to environmental and situational influences that make the consequences of errors more grave. A better understanding of the mechanisms that can lead to hyperactive error monitoring is of crucial importance to gain better insights into the pathways to anxiety.

In order to better understand these pathways, we demonstrated in a previous study that learning experiences that altered the significance of errors (i.e., punishment) can lead to short-lasting adaptations in the ERN (Riesel et al., 2012). In this study, errors were punished in some blocks of a flanker task but not others. We observed that the ERN was enhanced in blocks in which errors could be punished and, moreover, that this effect persisted after punishment had stopped. Further, trait anxiety influenced the effects of punishment, such that high anxious individuals showed stronger punishment-based modulations in ERN amplitude. The effect of punishment on the ERN was replicated and extended in a study by Meyer and Gawlowska (2017) that showed that the ERN was increased when errors were punished but not when the punishment was incidental to the commission of errors. Again this effect was modulated by trait anxiety, and group differences were only found when errors are punished. Together, these data suggest that pre-existing vulnerabilities can make some individuals more susceptible to the effects of punishment on the ERN. However, both previous punishment studies were conducted over the course of a single lab session, and it is thus unclear whether these effects persist for long enough that punishment might represent a viable mechanism in the pathway to anxiety.

The current study therefore intends to replicate and extend the effects of punishment on error monitoring. We extended the design of the Riesel et al. (2012) study in two important aspects. First, we aimed to examine the duration of punishment-based modulations on error monitoring over a longer time period. To that end, we added a second extinction phase 24 h after the acquisition phase. Second, in order to increase power to assess the effects of anxiety, we oversampled individuals high on trait anxiety. We hypothesized that punishment would increase the ERN amplitude and that this effect would persist after the punishment had stopped, up to 24 h later. Further, we expected that the effects of punishment would be strongest in the high anxiety group.

## 2. Methods

### 2.1. Participants

Individuals with elevated levels of anxiety were taken from a large sample of 1145 individuals that completed an online version of the Spielberger State-Trait Anxiety Inventory (STAI, Spielberger et al., 1983) and were recruited through local online advertisements. The STAI is a self-report questionnaire measuring individual differences in state and trait anxiety. The trait subscale (STAI-T) was used to recruit participants, and measures relatively stable individual differences in anxiety proneness.

Individuals with scores in the lowest (STAI-T $\leq$ 38) and highest third (STAI-T $\geq$ 47) of the distribution of trait anxiety scores of the large screening sample were invited to participate in the experiment. The sample included 50 participants (34 female) with STAI scores that ranged from 26 to 65 ($M$ = 43, $SD$ = 12.03). Those participants were split up into a low anxious group ($n$ = 23, STAI-T $M$ = 32.39, $SD$ = 7.26) and a high anxious group ($n$ = 27, STAI-T $M$ = 48.89, $SD$ = 10.62). After exclusion of 6 participants (high anxiety group: $n$ = 5, low anxiety group: $n$ = 1), due to either data quality (high anxiety group: $n$ = 1) or insufficient error numbers (fewer than 6, Olvet and Hajcak, 2009b) in at least one condition (high anxiety group: $n$ = 4, low anxiety group: $n$ = 1), a final sample of 44 participants (27 female) was retained: 22 individuals with elevated levels of trait anxiety and 22 low anxious comparison participants (see Table 1 for demographic and clinical characteristics). All participants were free of a current psychiatric disorder according to the Structured Clinical Interview for DSM-IV (SCID-I, German version; Wittchen et al., 1997).

All participants were Caucasian/European and between 18 and 65 years old ($M$ = 35.86 years, $SD$ = 13.31), had normal or corrected-to-normal vision and reported no history of head trauma or neurological disease. Groups were matched with regard to gender, age and level of education (see Table 1 for further characteristics). The participants received verbal and written information about the aims and procedure of the study. Written consent was obtained and the participants received either course credit or 10 € per hour. Study procedures

**Table 1**
Demographic and clinical characteristics of high and low anxious individuals.

| | High | Low | $t/\chi^2$ | df | $p$ |
|---|---|---|---|---|---|
| Demographics | | | | | |
| N (female:male) | 22 (14:8) | 22 (13:9) | 0.1 | 1 | 0.76 |
| Age | 22.86 (14.23) | 20.5 (14.26) | 0.55 | 42 | 0.59 |
| Clinical | | | | | |
| STAI-T | 54.14 (4.57) | 31.86 (3.93) | 17.33 | 42 | < **0.001**** |
| STAI-S – day 1 | 41.23 (9.24) | 32.59 (5.69) | 3.73 | 42 | < **0.001**** |
| STAI-S – day2 | 42.27 (8.67) | 31.73 (8.21) | 4.14 | 42 | < **0.001**** |
| OCI-R | 12.27 (8.33) | 5.73 (5.28) | 3.11 | 42 | < **0.01**** |
| BDI-II | 12.77 (8.72) | 3.18 (4.09) | 4.67 | 42 | < **0.001**** |
| FEE – emotional warmth | 10.64 (5.08) | 14.23 (5.85) | 2.17 | 42 | < **0.05*** |
| FEE – control and overprotection | 5.23 (3.14) | 5.29 (4.45) | 0.06 | 42 | 0.95 |
| FEE – rejection and punishment | 3.48 (3.02) | 3.79 (4.35) | 0.28 | 42 | 0.78 |

Note. STAI-T = Spielberger State-Trait Anxiety Inventory - Trait anxiety, STAI-S = Spielberger State-Trait Anxiety Inventory – State anxiety, OCI-R = Obsessive-Compulsive Inventory-Revised, BDI-II = Beck Depression Inventory–II, FEE = *Fragebogen zum erinnerten elterlichen Erziehungsverhalten*, The Questionnaire of Recalled Parental Rearing Behaviour. Significant values are printed in bold.

* $p < 0.05$.

** $p < 0.01$.

were in accordance with the Declaration of Helsinki and approved by the local ethics committee. We confirm that we have reported all measures, conditions, and data exclusions.

### 2.2. Measures

Participants were recruited based on scores on the Spielberger State-Trait Anxiety Inventory (STAI; Spielberger et al., 1983). The STAI is a 40-item self-report questionnaire measuring individual differences in state and trait anxiety with scores ranging from 20 to 80, with higher scores indicating more anxiety. Twenty items assess trait anxiety (STAI-T), which refers to feelings of worry and stress experienced daily across situations. In addition, 20 items assess state anxiety (STAI-S), which refers to nervousness and arousal triggered by perceived threats. The STAI and its German version (Laux et al., 1981) have demonstrated good internal consistency, test-retest reliability, as well as construct and concurrent validity (Spielberger et al., 1983; Spielberger et al., 1983). In addition to trait anxiety, we measured obsessive-compulsive symptoms with the Obsessive-Compulsive Inventory-Revised (OCI-R, Foa et al., 2002) in a German version (Gonner et al., 2008). Further, depressive symptoms were assessed with the German version of the Beck Depression Inventory–II (BDI-II, Beck et al., 1996, Hautzinger et al., 2006). Parenting behavior was assessed using a German questionnaire of Recalled Parental Rearing Behaviour (*Fragebogen zum erinnerten elterlichen Erziehungsverhalten*, FEE, Schumacher et al., 1999). The German version of the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders (DSM) fourth edition (SCID-I; First et al., 1996; Wittchen et al., 1997) was administered to determine participant DSM-IV diagnosis status. The SCID-I is a semi-structured, well-validated interview used to guide the diagnosis of DSM-IV disorders. The SCID-I has demonstrated moderate to high inter-rater agreement (Lobbestael, Leurgans, & Arntz, 2011). In the first step, participants completed an online screener based on SCID questions. During the telephone recruitment, a follow-up interview was conducted by a clinically trained student to determine eligibility.

### 2.3. Task and procedure

An arrowhead version of the flanker task (Eriksen & Eriksen, 1974; Kopp, Rist, & Mattler, 1996) was administered using Presentation software (Neurobehavioral Systems, Inc., Albany, Calif.). On each trial five horizontally aligned arrowheads were presented and participants were instructed to respond with their left or right index finger in accordance with the direction of the central arrowhead. Half the trials were compatible (e.g., flanker arrows and target point in the same direction) and half were incompatible (e.g., flanker arrows and target point in the opposite directions), displayed in a pseudorandomized order. At a viewing distance of approximately 65 cm, the set of arrows filled 0.9° of visual angle vertically and 7.5° horizontally.

The Flanker task was administered on two consecutive days and included three experimental phases (acquisition, extinction 1 and extinction 2). In each phase, there were two conditions (control and punishment condition). There were blocks of trials in which errors were punished (punishment condition), and blocks of trials in which errors were never punished (control condition). The punishment and control conditions were distinguished by the color of the arrows presented, which were either blue or yellow. The experimental setup is illustrated in Fig. 1. The assignment of color to control versus punishment condition was counterbalanced across participants. The conditions (control vs. punishment) varied blockwise in an alternating way, whereby the order was counterbalanced across participants. In the beginning of the experiment, participants were explicitly instructed that errors in one color condition would sometimes be followed by a loud sound. However, they were not informed which color would be punished. Thus, the experimental design combined elements of both instructed and associative learning. The reinforcement schedule during the acquisition block changed from continuous (i.e., the first five errors in the punishment condition were punished) to intermittent (50% of errors in the punishment blocks were punished subsequently). This reinforcement schedule was used to ensure both fast and stable learning. The acquisition phase was followed by two extinction phases. During the extinction phases, errors were never followed by a loud sound in either condition (formerly punished and control). Extinction phase one directly followed acquisition. The second extinction phase was recorded the following day 24 h later.

Each experimental phase consisted of eight blocks of 64 trials per block (1536 trials total in all three phases). Prior to the experiment, the participants performed a practice block containing 20 trials, half presented in blue and half in yellow. All trials started with the presentation of a central fixation cross (Intertrial interval, ITI) for 900 to 1500 ms (duration varied randomly). Afterward the flanker stimuli were presented for 200 ms, followed by a response interval that lasted until a response was made with a maximal duration of 800 ms. Then a new trial started. Thus the total duration of a trial varied between 1300 ms (for very fast response with 200 ms reaction time and a 900 ms ITI) to maximal 2500 ms. The timing and structure were different for punishment trials: Such that during acquisition phase, the commission of an error in the punishment condition was punished by presenting an aversive loud sound (100 db) with a 1-s duration via two speakers to the right and left of the monitor. The high-pitched sound (3500 Hz) was

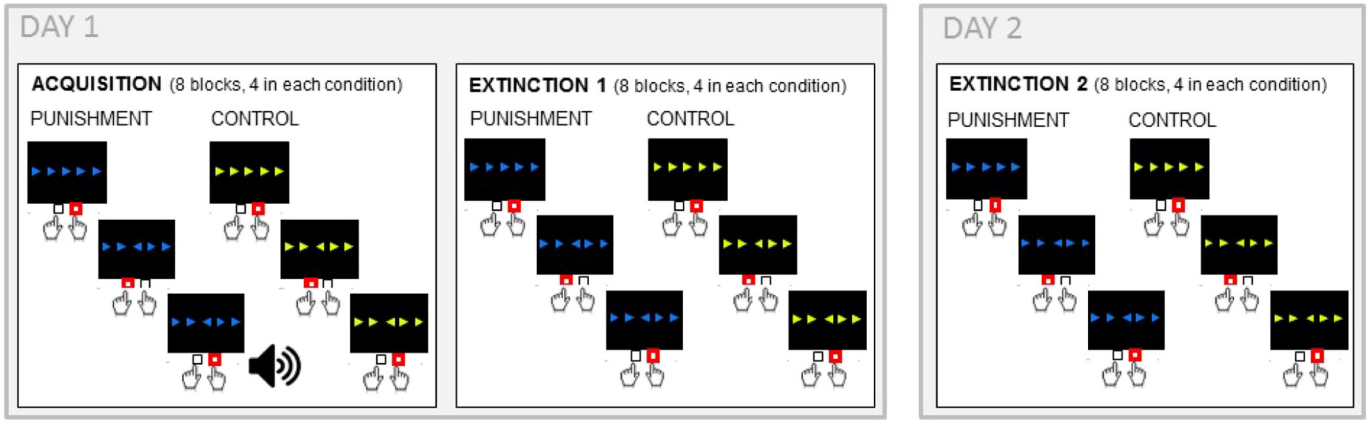


**Fig. 1.** Schematic depiction of the experimental setup of the modified Flanker Task. (For interpretation of the references to color in this figure, the reader is referred to the web version of this article.)

presented 1 s after the error was committed to prevent a potential influence on response-locked ERPs. After a punishment sound was presented, the original ITI (900 to 1500 ms) was increased by 1500 ms, such that it varied between 2400 and 3000 ms before the next trial started. Thus the total duration of a punished trials was 3500 ms sec longer (1000 ms pause after an error before the sound was presented, 1000 ms sound presentation and 1500 ms longer ITI) and varied between 4800 ms (for very fast errors with 200 ms reaction time) to maximal 6000 ms. The number of punished errors varied across participants, depending on their error rate and on average 11.48 errors were punished ($SD$ = 5.44). Groups did not differ in the amount of punished errors ($t(1,42) = 1.55$, $p = 0.13$). At the end of the experiment, participants were asked whether and when they realized that the punishment had stopped.[1] Fifty-seven percent of the participants stated that they were aware midway through the experiment that errors in the punishment condition were no longer punished. After 60% of the experiment, 86% of participants indicated that punishment stopped. High and low anxious participants did not differ in their ratings when punishment stopped (high anxiety group: $M$ = 49.05%, $SD$ = 18.68, low anxiety group: $M$ = 49.05%, $SD$ = 18.95, $t(1,40) = 0.00$, $p = 1$).

Throughout the experiment, participants were encouraged via written and verbal feedback to be both fast and accurate in their performance. Performance-based feedback was presented at the end of each block. If performance accuracy was below 75%, a message appeared instructing participants to respond more accurately. When the performance was above 90%, participants were instructed to respond faster. Accuracy rates between 75% and 90% were followed by the feedback, “You're doing a great job.” The total duration of the task was approximately 45 min on day one and 20 min on day 2. In addition to the flanker task, the Emotional Interrupt paradigm (Mitchell et al., 2006) was collected from the same participants on day 2 after extinction 2. These results are still being analyzed and will be reported elsewhere.

### 2.4. Psychophysiological recording, data reduction, and analysis

The continuous EEG was recorded with sintered Ag/AgCl electrodes from 64 sites from a concentric and equidistant EasyCap montage (Easycap, GmbH, Herrsching, Germany). Additional electrodes were placed at the following four locations: approximately 2 cm below each eye, nasion and neck. The ground electrode was attached to the right cheek below T1. All electrodes were referenced to Cz and were kept below 5 kΩ during recording. The EEG was digitized with a sampling rate of 500 Hz. Offline, the data was referenced to the average of all electrodes, and bandpass filtered with low and high cutoffs of 0.1 and 30 Hz (slope 24db/octave), respectively and a Notch filter of 50 Hz. Using the software Brain Vision Analyzer 2.0 (Brainproducts, München) raw data were preprocessed and eye movement artifacts were corrected with an Independent Component Analysis (ICA). Response-locked epochs with duration of 1500 ms including a 500-ms prestimulus interval were extracted. A semiautomatic procedure was used to detect and reject artifacts. Epochs containing a voltage step of > 50 mV between sample points, a voltage difference of 300 mV within a trial, and a maximum voltage difference of < 0.50 mV within 100-ms intervals were rejected. In addition, visual inspection of the data was conducted to detect and reject any remaining artifacts. In addition to the ERN, a smaller negative component, the correct-related negativity (CRN; Ford, 1999; Vidal et al., 2000) with a similar time-course, topography and source was examined following correct responses. Response-locked ERPs were averaged separately for each participant, each experimental condition and phase, and for incorrect and correct responses. Trials with response times < 100 ms and > 700 ms were excluded from averaging. The interval from −100 ms to the response served as a baseline. The response-locked negativities were evaluated as the difference between the most negative peak occurring in a 150 ms epoch following the response and the preceding positive peak (between −50 and 30 ms relative to the response). Response-locked ERPs were analyzed at electrode FCz, where error-related brain activity was maximal (e.g., Riesel et al., 2011). Grand averages were filtered with a 15-Hz low-pass filter for visual presentation.

Statistical analyses were conducted using SPSS (Version 25.0). A 2 (*response*: error, correct) × 2 (*condition*: punishment, control) × 3 (*phase*: acquisition, extinction 1, extinction 2) repeated measures analysis of variance (ANOVA) with *group* (high, low) as between-subjects factor was used to analyze reaction times and ERN amplitude. For all ERP analyses, only subjects that had at least six artifact-free error trials were analyzed (Olvet and Hajcak, 2009b). Error rate and post-error slowing were statistically analyzed by using a 2 (*condition*: punishment, control) × 3 (*phase*: acquisition, extinction 1, extinction 2) ANOVA with *group* (high, low) as between-subjects factor. Paired *t*-tests were performed for follow-up post hoc tests. The significance level was $\alpha = 0.05$, two-tailed. Greenhouse-Geisser correction was used for all comparisons with more than two within-subject levels and ε is reported. Correlational analyses were performed to examine the retest-reliability for the ERN and CRN across the three phases separately for the control and punishment condition and both groups. Related results are included in the data supplement.

[1] Note that due to technical problems rating data of one participant of the high anxiety group and one in the low anxiety group is missing.

## 3. Results

### 3.1. Behavioral results[2]

Behavioral data across the different conditions and phases in both anxiety groups are presented in Table 2. Error rates differed between the experimental phases, as reflected in a main effect of phase ($F(2,82) = 5.81$, $p < 0.01$, $\eta_P^2 = 0.12$). Post-hoc tests indicated that error rates increased between the learning phase and extinction phase 1 ($t(1,42) = 3.10$, $p < 0.01$) as well as extinction phase 2 ($t(1,42) = 3.64$, $p < 0.01$). The two extinction phases did not differ in error rate ($t(1,42) = 0.35$, $p = 0.73$). No main effect of *condition* was observed ($F(1,41) = 3.37$, $p = 0.07$, $\eta_P^2 = 0.08$) and no interactions between *condition* and *phase* ($F(2,82) = 1.27$, $p = 0.29$, $\eta_P^2 = 0.03$, $\varepsilon = 0.97$). Error rates did not differ between the high and low *anxiety group* ($F(1,41) = 0.86$, $p = 0.36$, $\eta_P^2 = 0.02$) and no interactions between *condition* and *group* ($F(1,41) = 0.75$, $p = 0.39$, $\eta_P^2 = 0.02$) or between *phase*, *condition* and *group* were observed ($F(2,82) = 2.73$, $p = 0.07$, $\eta_P^2 = 0.06$, $\varepsilon = 0.97$).

Post-error slowing (i.e., average difference in reaction time between the post-error trial and the associated pre-error trial, Dutilh et al., 2012) varied significantly across the phases of the experiment ($F(2,82) = 22.30$, $p < 0.001$, $\eta_P^2 = 0.36$). No main effect of *condition* was observed ($F(1,41) = 2.72$, $p = 0.11$, $\eta_P^2 = 0.06$). A significant interaction between *condition* and *phase* ($F(2,82) = 3.21$, $p < 0.05$, $\eta_P^2 = 0.07$, $\varepsilon = 0.98$) emerged. Post-hoc tests indicated that post-error slowing was significantly larger in the punishment compared to the control condition in the learning phase only ($t(1,42) = 2.72$, $p < 0.01$), whereas the two conditions did not differ during extinction 1 ($t(1,42) = 0.62$, $p = 0.54$) and extinction 2 ($t(1,42) = 0.09$, $p = 0.93$). Post-error slowing did not differ between the high and low anxiety *group* ($F(1,41) = 0.27$, $p = 0.61$, $\eta_P^2 = 0.006$) and no interactions between *condition* and *group* ($F(1,41) = 1.96$, $p = 0.17$, $\eta_P^2 = 0.05$), *phase* and *group* ($F(2,82) = 0.47$, $p = 0.63$, $\eta_P^2 = 0.01$) or *phase*, *condition* and *group* ($F(2,82) = 0.29$, $p = 0.75$, $\eta_P^2 = 0.007$, $\varepsilon = 0.98$) were observed.

The results of the ANOVA for reaction times are presented in Table 3. A main effect of *response type* reflected that errors were faster compared to correct responses. A main effect of phase was observed such that reaction times decreased from the first day to second day (learning vs. extinction 2: $t(1,42) = 6.37$, $p < 0.001$, extinction 1 vs. extinction 2: $t(1,42) = 6.54$, $p < 0.001$) but did not differ between learning and extinction 1 ($t(1,42) = -0.77$, $p = 0.44$).

### 3.2. Error-related brain activity

Fig. 2 presents the response-locked ERP waveforms for ERN and CRN in the punishment and control condition in the three experimental phases separately for high and low anxious participants. Results for the 2 (*response*) × 2 (*condition*) ×2 (*group*) × 3 (*phase*) ANOVA for ERN and CRN are presented in Table 4. To further decompose results that included several interactions with the factor *response*, we conducted a 2 (*condition*) × 2 (*group*) × 3 (*phase*) ANOVA separately for ERN and CRN.

#### 3.2.1. Error-related negativity

No significant main effect of *anxiety group* on the magnitude of the ERN was observed ($F(1,42) = 0.36$, $p = 0.55$, $\eta_P^2 = 0.009$). Nor was there a significant main effect of *condition* ($F(1,42) = 2.27$, $p = 0.14$, $\eta_P^2 = 0.05$) reflecting that in the full sample the punishment and control conditions did not differ. A significant main effect of *phase* was observed ($F(2,84) = 8.46$, $p < 0.001$, $\eta_P^2 = 0.17$, $\varepsilon = 0.94$) indicating that ERN amplitude changed across phases. Across both groups, the ERN was reduced in the extinction phase 1 compared to the learning phase ($t(1,43) = 3.79$, $p < 0.001$) but recovered on day two and did not differ between learning phase and extinction 2 ($t(1,43) = 1.21$, $p = 0.23$). Thus, the ERN was smaller for extinction 1 compared to extinction 2 ($t(1,43) = 3.23$, $p < 0.01$). The phase effects were not modulated by group or condition (*condition* × *phase*: $F(2,84) = 0.16$, $p = 0.86$, $\eta_P^2 = 0.004$, $\varepsilon = 0.99$; *phase* × *group*: $F(2,84) = 0.09$, $p = 0.91$, $\eta_P^2 = 0.002$, $\varepsilon = 0.94$).

Importantly, anxiety modulated the effects of punishment on the

**Table 2**
Performance data of high and low anxious individuals.

| | | High Anxiety group[a] | Low Anxiety group |
|---|---|---|---|
| Behavior | | | |
| Error rate | | | |
| Acquisition | Control | 7.28 (3.27) | 8.01 (5.89) |
| | Punishment | 5.98 (2.30) | 7.54 (4.95) |
| Extinction 1 | Control | 8.49 (4.07) | 9.75 (9.18) |
| | Punishment | 8.71 (4.17) | 9.34 (7.92) |
| Extinction 2 | Control | 7.92 (3.77) | 10.38 (8.33) |
| | Punishment | 7.61 (3.37) | 9.23 (6.01) |
| Reaction time for errors in ms | | | |
| Acquisition | Control | 382 (54) | 375 (49) |
| | Punishment | 377 (44) | 393 (65) |
| Extinction 1 | Control | 394 (58) | 391 (62) |
| | Punishment | 389 (47) | 404 (76) |
| Extinction 2 | Control | 366 (47) | 363 (46) |
| | Punishment | 371 (39) | 365 (51) |
| Reaction time for correct responses in ms | | | |
| Acquisition | Control | 434 (45) | 437 (49) |
| | Punishment | 439 (48) | 437 (49) |
| Extinction 1 | Control | 433 (47) | 426 (53) |
| | Punishment | 431 (47) | 428 (56) |
| Extinction 2 | Control | 413 (48) | 402 (48) |
| | Punishment | 412 (47) | 404 (47) |
| Post-error slowing in ms | | | |
| Acquisition | Control | 40 (26) | 41 (25) |
| | Punishment | 60 (30) | 52 (36) |
| Extinction 1 | Control | 23 (28) | 29 (33) |
| | Punishment | 21 (30) | 20 (32) |
| Extinction 2 | Control | 25 (23) | 20 (35) |
| | Punishment | 29 (29) | 20 (23) |

[a] Performance data of one participant of the high anxiety group is missing because of technical problems.

**Table 3**
Results of the 2 (response) × 2 (condition) × 2 (group) × 3 (phase) ANOVA for reaction times.

| Effect | df | *F* | *p*-Value | $\eta_P^2$ |
|---|---|---|---|---|
| Condition | 1, 41 | 1.72 | 0.20 | 0.04 |
| Condition × Group | 1, 41 | 3.53 | 0.07 | 0.08 |
| Phase | 2, 82 | 25.27 | < **0.001**** | 0.38 |
| Phase × Group | 2, 82 | 0.49 | 0.61 | 0.01 |
| Response | 1, 41 | 257.98 | < **0.001**** | 0.86 |
| Response × Group | 1, 41 | 1.35 | 0.25 | 0.03 |
| Condition × Phase | 2, 82 | 0.44 | 0.64 | 0.01 |
| Condition × Phase × Group | 2, 82 | 1.80 | 0.17 | 0.04 |
| Condition × Response | 1, 41 | 0.84 | 0.36 | 0.02 |
| Condition × Response × Group | 1, 41 | 5.88 | < **0.05*** | 0.13 |
| Phase × Response | 2, 82 | 12.32 | < **0.001**** | 0.23 |
| Phase × Response × Group | 2, 82 | 0.36 | 0.70 | 0.01 |
| Condition × Phase × Response | 2, 82 | 0.01 | 0.99 | 0.00 |
| Condition × Phase × Response × Group | 2, 82 | 5.95 | < **0.01**** | 0.13 |
| Group | 1, 41 | 0.002 | 0.96 | 0.00 |

Note. df = degrees of freedom.
Significant values are printed in bold.
* $p < 0.05$.
** $p < 0.01$.

[2] Note that due to technical problems during recording behavioral data of one participant of the high anxiety group is missing.

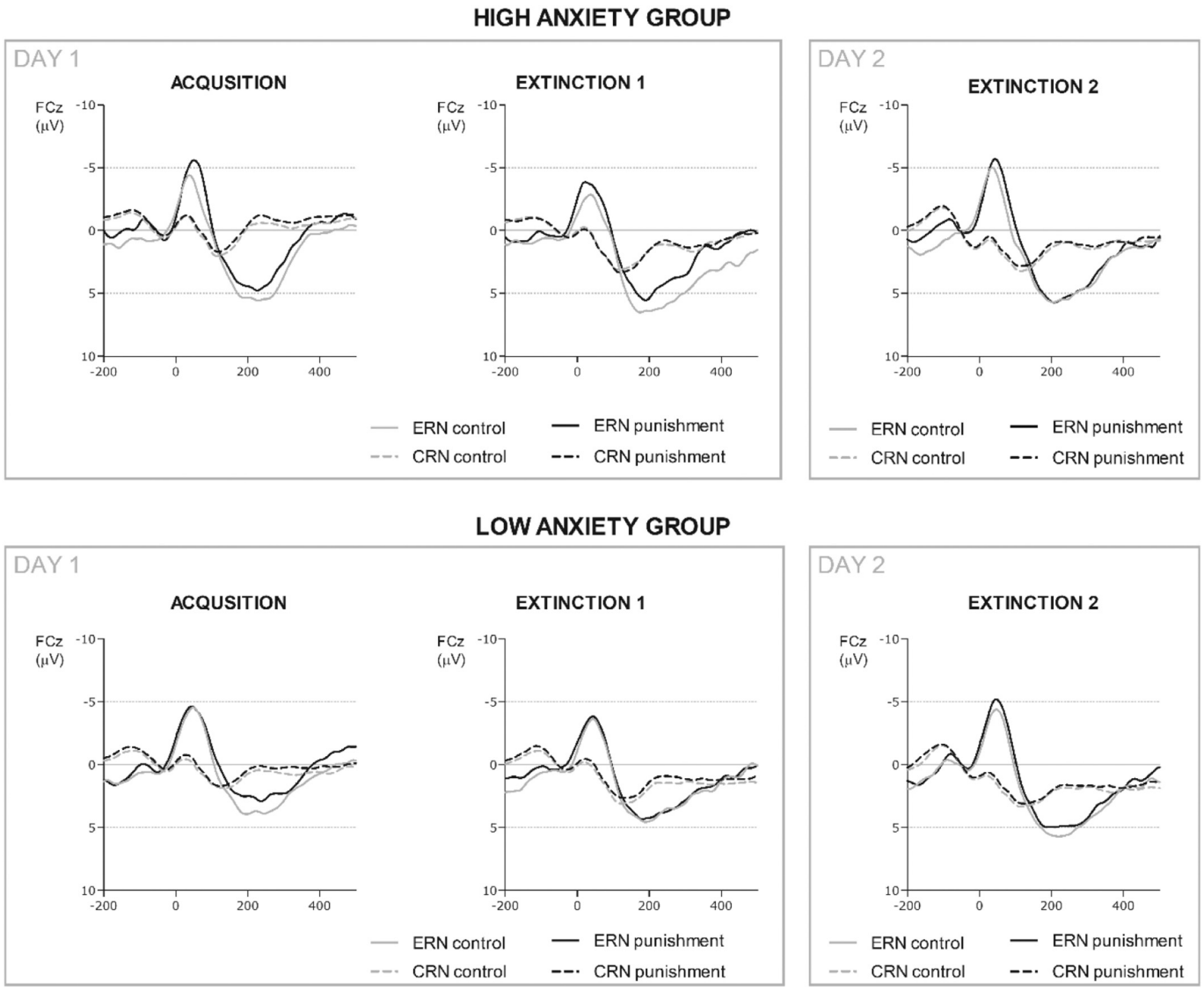


**Fig. 2.** ERN (solid) and CRN (dashed) in the punishment (black) and control condition (grey) during acquisition, extinction 1 and extinction 2 in the high (upper panel) and low anxiety group (lower panel).

**Table 4**
Results of the 2 (response) × 2 (condition) × 2 (group) × 3 (phase) ANOVA for ERPs.

| Effect | df | *F* | *p*-Value | $\eta_P^2$ |
|---|---|---|---|---|
| Condition | 1, 42 | 3.29 | 0.08 | 0.07 |
| Condition × Group | 1, 42 | 6.68 | < **0.05*** | 0.14 |
| Phase | 2, 84 | 14.23 | < **0.001**** | 0.25 |
| Phase × Group | 2, 84 | 0.38 | 0.69 | 0.01 |
| Response | 1, 42 | 123.79 | < **0.001**** | 0.75 |
| Response × Group | 1, 42 | 0.02 | 0.88 | 0.001 |
| Condition × Phase | 2, 84 | 0.47 | 0.63 | 0.01 |
| Condition × Phase × Group | 2, 84 | 0.67 | 0.51 | 0.02 |
| Condition × Response | 1, 42 | 2.51 | 0.12 | 0.056 |
| Condition × Response × Group | 1, 42 | 11.41 | < **0.01**** | 0.21 |
| Phase × Response | 2, 84 | 4.58 | < **0.05*** | 0.10 |
| Phase × Response × Group | 2, 84 | 0.43 | 0.65 | 0.01 |
| Condition × Phase × Response | 2, 84 | 0.31 | 0.73 | 0.007 |
| Condition × Phase × Response × Group | 2, 84 | 1.12 | 0.33 | 0.03 |
| Group | 1, 42 | 0.91 | 0.35 | 0.02 |

Note. df = degrees of freedom.
Significant values are printed in bold.
* $p < 0.05$.
** $p < 0.01$.

**Table 5**
ERN for high and low anxious subjects divided by phase and condition.

| | | Control | Punishment |
|---|---|---|---|
| High anxiety | Learning | −7.02 (3.53) | −8.79 (4.11) |
| | Extinction 1 | −5.75 (3.94) | −6.82 (3.58) |
| | Extinction 2 | −6.77 (3.17) | −7.74 (3.16) |
| | Overall | −6.51 (2.99) | −7.78 (3.13) |
| Low anxiety | Learning | −7.74 (3.53) | −6.88 (3.94) |
| | Extinction 1 | −5.92 (2.76) | −5.39 (2.75) |
| | Extinction 2 | −6.82 (3.85) | −7.06 (3.87) |
| | Overall | −6.83 (2.89) | −6.44 (2.88) |

Note. Mean (Standard Deviation).

ERN as reflected in a significant interaction between *condition* and *group* ($F(1,42) = 7.90$, $p < 0.01$, $\eta_P^2 = 0.16$). No three-way interaction between *condition* × *phase* × *group* ($F(2,84) = 1.30$, $p = 0.28$, $\eta_P^2 = 0.03$, $\varepsilon = 0.99$) was observed, indicating that the interaction between condition by group was not modulated by the different experimental phases (see Fig. 2 and Table 5). Post-hoc *t*-tests revealed that the ERN averaged across the three phases in the punishment condition was larger (i.e., more negative) compared to the ERN in the control

condition in high anxious individuals (high anxiety group: ERN punishment: Mean = −7.78, SD = 3.13, ERN control: Mean = −6.51 μV, SD = 2.99, $t(1,21) = 3.41$, $p < 0.01$). In the low anxiety group, no difference between the punishment and control condition was observed (low anxiety group: Mean = −6.44, SD = 2.88, ERN control: Mean = −6.83 μV, SD = 2.89, $t(1,21) = -0.84$, $p = 0.41$).

#### 3.2.2. Correct-related negativity

A significant main effect of *phase* ($F(2,84) = 15.22$, $p < 0.001$, $\eta_P^2 = 0.27$) emerged. The CRN was larger in the learning phase and decreased to extinction 1 ($t(1,43) = 4.67$, $p < 0.001$) and the delayed extinction phase 2 ($t(1,43) = 4.57$, $p < 0.001$). The two extinction phases did not differ in CRN magnitude ($t(1,43) = 0.21$, $p = 0.84$). The CRN was not modulated by *condition* ($F(1,42) = 0.38$, $p = 0.54$, $\eta_P^2 = 0.009$) and no interaction between *condition* and *phase* was observed ($F(2,84) = 0.76$, $p = 0.47$, $\eta_P^2 = 0.02$). Further, no main effect of *anxiety group* on the magnitude of the CRN was observed ($F(1,42) = 0.92$, $p = 0.34$, $\eta_P^2 = 0.02$) and no interactions between *group* and *condition* ($F(1,42) = 1.35$, $p = 0.25$, $\eta_P^2 = 0.03$), *group* and *phase* ($F(2,84) = 2.25$, $p = 0.11$, $\eta_P^2 = 0.05$) or *group*, *phase* and *condition* ($F(2,84) = 0.41$, $p = 0.67$, $\eta_P^2 = 0.01$) were observed.

## 4. Discussion

The present study examined the duration of the effects of punishment on neural responses to errors in high and low anxious individuals. Consistent with our hypotheses and previous work (Meyer and Gawlowska, 2017; Riesel et al., 2012), we observed that errors in a punished condition elicited a larger ERN than errors in an unpunished condition. Further, this effect was only evident in highly anxious individuals—indeed, in individuals low on trait anxiety, punishment did not significantly modulate the magnitude of the ERN. Additionally, the present study extends previous work by demonstrating the lasting nature of punishment-induced changes in the ERN. An enhanced ERN in the formerly-punished condition in high anxious individuals was still evident even after punishment had stopped—both in an immediate extinction phase, and in a delayed extinction phase 24 h later.

### 4.1. Punishment-based modulation of error processing

The finding of punishment-based modulation of error processing in high anxious individuals adds to the body of evidence suggesting that not only is error-related brain activity subject to genetic influence (Anokhin et al., 2008; Burwell et al., 2016), but the ERN can also be modulated by situational contexts (Hajcak et al., 2005; Riesel et al., 2019a) and learning experiences (Meyer and Gawlowska, 2017; Riesel et al., 2012), that change the motivational and affective value of an error. Moreover, the present results suggest that these learning-related modulations can be enduring. As reviewed in the introduction, an enhanced ERN has consistently been observed in anxiety disorders, and has therefore been proposed as a viable candidate for a genetically-mediated risk marker or endophenotype for anxiety-related psychopathologies (Riesel, 2019; Riesel et al., 2019b; Weinberg et al., 2015a; Weinberg et al., 2012). In addition to genes, environmental risk factors play an important role in the process by which genetic risk is translated into dysfunction and psychopathology. Consistent with this, the results of the present study add to a growing body of work linking punishment (Meyer and Gawlowska, 2017; Riesel et al., 2012) or punitive and/or overprotective parental behaviors more broadly (Banica et al., 2019; Brooker and Buss, 2014; Meyer et al., 2019; Meyer et al., 2015)—each of which is thought to be an etiological factor in the development of anxiety disorders (Hicks et al., 2009; Hirshfeld-Becker et al., 2008)—to alterations in the way the brain processes errors. Moreover, there is evidence that the ERN mediates the association between harsh parenting and the development of anxiety (Meyer et al., 2015). In addition, differences in trait anxiety, one of the most commonly-examined vulnerability factors for clinical anxiety, have also been linked to alterations in learning and conditioning processes (Craske et al., 2018; Lonsdorf and Merz, 2017). More specifically, facilitated fear acquisition and slowed extinction have been proposed as potential etiological factors in the development of anxiety (Barrett and Armony, 2009; Lissek et al., 2005; Otto et al., 2007; Sehlmeyer et al., 2011). In line with this, our results suggest that a predisposition to anxiety facilitates learning about errors, thereby increasing the possibility of acquiring and maintaining hyperactive error monitoring. This study therefore provides an experimental model of the ways in which punishment experiences might sensitize individuals to the commission of errors, thereby influencing the magnitude of the ERN.

### 4.2. Group differences in error-related negativity between anxiety groups

In the present study, we recruited individuals high and low on trait anxiety in order to increase power for the analyses of interest. However, even though the punishment-based modulation in ERN was larger/only present in the high anxiety groups, it is worth noting that these two groups did not differ in the magnitude of their ERN. This is in contrast to several previous studies linking anxiety to increased ERN amplitudes (Aarts and Pourtois, 2010; Hajcak et al., 2003b; Meyer et al., 2012). Though recruited based on anxiety levels, our HA group was also screened to be free from current psychiatric disorders—it is possible baseline group differences would emerge with greater symptom severity. In addition, regarding the relationship of the ERN with measures of anxiety, a threefold larger effect size has been found with measures of anxious apprehension compared to mixed anxiety measures (Moser et al., 2013; Saunders & Inzlicht, Preprint). Thus, using the STAT-T as a broad measure of mixed anxiety symptoms to recruit the samples may have also limited the power to detect baseline group differences in ERN.

Another possibility is related to the fact that group differences in error monitoring are sensitive to task differences (Gründler et al., 2009; Kaczkurkin, 2013; Riesel, 2019). Previous work comparing healthy controls to OCD patients suggest that group differences (i.e., larger ERN amplitudes in OCD compared to healthy individuals) vanish in conditions that increase error significance, such as monetary punishment for errors (Endrass et al., 2010) or accuracy instruction (Riesel et al., 2019a). It may be the case that, under conditions in which it is adaptive to upregulate error monitoring and that require a cautious response strategy, healthy individuals display an ERN that is comparable to the one observed in OCD. Similarly, the introduction of a punishment condition in the experiment may have altered the significance of errors throughout the experiment for the low anxiety group, resulting in an up-regulation and thereby masking existing group differences. Additional research will be necessary to explore this possibility and to examine whether alterations in the neurocognitive adaptability of error monitoring to different instructions and contexts can also be seen in anxiety.

### 4.3. "What is cognitive control without affect?" The role of affective influences on error processing

In the context of the present special issue "*What is cognitive control without affect?*" our results highlight motivational and affective influences that shape the way an individual processes errors and the way an individual executes cognitive control more broadly. Affective and motivational influences on error processing have been operationalized as individual differences in anxiety that have been found to be associated with differences in learning about the value of errors. Thus, variation in psychophysiological responses to errors may reflect meaningful and stable individual differences that reflect the subjective value of errors based on context, genes, personality, and learning history. In this regard, affective variables influence the way an individual processes errors. However, regardless of individual differences, error-processing is not affectively neutral, in that it is accompanied by negative emotions

(Saunders et al., 2017) and leads to cognitive and affective adaptations to avoid future negative outcomes (Cavanagh and Shackman, 2014; Shackman et al., 2011). Taken together, we believe that a dynamic interaction between affective variables and cognitive control may be precisely what leads to a system that is flexible and adaptive to a complex and changing environment.

### 4.4. Limitations and implications for future research

Further limitations of the study suggest future directions. For instance, the design of our study combines elements of instructed and associative learning. Participants were explicitly instructed that errors in one condition would be punished, though they were not told which color would be associated with punishment. Previous work suggests that instructed and associative learning have similar effects on psychophysiological indices of fear learning (Olsson & Phelps, 2004). However, it might be useful to differentiate discrete learning processes in future studies. Similarly, future studies over longer time periods are needed to gain more insights in the temporal dynamics of the observed effects.

Additionally, our study did not include a condition in which threat was independent of the commission of errors—it may be the case that, rather than a direct association between punishment and error processing, there is an indirect effect, whereby punishment in general increased anxiety, thereby increasing the magnitude of the ERN. However, we would note here that previous research suggests this would not be the case. For instance, general threat-related increases in anxiety (i.e., the presence of a tarantula, for spider phobic individuals) do not appear to increase the ERN (Moser, Hajcak, & Simons, 2005). Consistent with this, in a previous study in which participants experienced electric shock either at random or as a consequence of errors, the ERN was specifically enhanced in the condition in which errors were punished, and not under conditions in which the punishment was incidental to the commission of errors (Meyer and Gawlowska, 2017).

In addition, one important question for future studies relates to the generalizability of these effects across different tasks and contexts, and how this is modulated by anxiety. In order to better understand the pathways to anxiety and the contribution of genes, learning experiences, and gene-environment interactions, it is crucial to investigate individuals at increased familial risk for anxiety at an age stage that is known to be sensitive is for the development of anxiety symptoms (e.g., childhood and adolescence). Finally, it has to be noted that the observed effects of punishment on the ERN varies substantially across participants in both groups. Thus, future studies are needed to replicate this effect and to get a better understanding of potential moderators in addition to anxiety such as emotion regulation or coping style (Kessel et al., 2019).

### 4.5. Conclusion

The present results indicate that punishment leads to an increase of ERN magnitude in high anxious individuals and that this effect persists after the punishment has stopped in an immediate and delayed extinction phase (24 h later). We have previously proposed that individual differences in anxiety and learning history interact to influence the ERN—and that the ERN could therefore be used to better understand trajectories of *risk* for anxiety disorders. The results of this study point to a mechanism by which punitive learning environments contribute to the development of an increased ERN, particularly in individuals with greater dispositional anxiety, thereby further increasing risk for anxiety disorders. This may indicate a pathway by which trait vulnerability, evident in a heightened predisposition to anxiety, translates into psychopathology via interactions with the environment and its influences on error processing. By measuring the impact of both trait and learning factors, these data may therefore provide a foundation for future studies that seek to unravel the complex influences of genes and environment on neural indices of error monitoring.

## Declaration of competing interest

None.

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## Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijpsycho.2019.09.014.

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