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Disentangling the effects of trait and state worry on error-related brain activity: Results from a randomized controlled trial using worry manipulations

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Abstract

Enhanced amplitudes of the error-related negativity (ERN) have been suggested to be a transdiagnostic neural risk marker for internalizing psychopathology. Previous studies propose worry to be an underlying mechanism driving the association between enhanced ERN and anxiety. The present preregistered study focused on disentangling possible effects of trait and state worry on the ERN by utilizing a cross sectional observational and a longitudinal randomized controlled experimental design. To this end, we examined the ERN of $n = 90$ students during a flanker task (T0), which were then randomly assigned to one of three groups (worry induction, worry reduction, passive control group). Following the intervention, participants performed another flanker task (T1) to determine potential alterations of their ERN. Manipulation checks revealed that compared to the control group, state worry increased in the induction but also in the reduction group. ERN amplitudes did not vary as a function of state worry. An association of trait worry with larger ERN amplitudes was only observed in females. Furthermore, we found larger ERN amplitudes in participants with a current or lifetime diagnosis of internalizing disorders. In summary, our findings suggest that the ERN seems to be insensitive to variations in state worry, but that an elevated ERN is associated with the trait-like tendency to worry and internalizing psychopathology, which is consistent with the notion that the ERN likely represents a trait-like neural risk associated with anxiety.

KEYWORDS

anxiety, anxious apprehension, EEG, ERN, error monitoring, worry

1 | INTRODUCTION

Goal-directed behavior is a fundamental ability allowing humans to adjust to their environment. Cognitive control, especially performance monitoring, plays a crucial role for goal-directed behavior. By monitoring errors (i.e., potential harm), humans can improve upcoming behavior through cognitive, motivational, and behavioral adjustments (Botvinick et al., 2001; Cavanagh & Shackman, 2015; Proudfit et al., 2013; Simons, 2010; Weinberg et al., 2012).

A commonly studied event-related potential (ERP) associated with performance monitoring is the error-related negativity (ERN; Gehring et al., 1993); or originally called the error negativity (N_E; Falkenstein et al., 1991). The ERN is assumed to constitute the neural representative of error monitoring and is observable as a fronto-central negative peak approx. 50 ms after committing an error. The monitoring of correct responses elicits a similarly timed but smaller negative peak after a correct response: the correct-response negativity (CRN; Vidal et al., 2003, 2000). Various hypotheses have been postulated about the functional role of the ERN, ranging from signaling the mismatch between representations of required and actual responses (Falkenstein et al., 1991; Gehring et al., 1993), over indicating the conflict of two simultaneously active response tendencies (Botvinick et al., 2001; Yeung et al., 2004), to allowing reinforcement learning to modify performance on a task at hand by signaling whenever an outcome was not as predicted (Holroyd & Coles, 2002). All of these hypotheses suggest that the ERN is crucial for cognitive control and a prerequisite to adjusting behavior to the requirements of the task and improving future performance.

Regarding the clinical utility of error monitoring (Hajcak et al., 2019), increased or decreased ERN amplitudes have been discussed in the literature as an endophenotype for the development and maintenance of psychopathological symptoms (e.g., Manoach & Agam, 2013; Olvet & Hajcak, 2008; Riesel, Klawohn, et al., 2019). An endophenotype is defined as a measurable (mostly, but not necessarily, biological) component on the complex pathway between the genotype and a psychopathological phenotype, informative of the specific mechanisms that lead to a complex mental disorder (Gottesman & Gould, 2003). A marker qualifies as an endophenotype (a) when it is associated with the illness, (b) when it is heritable, (c) when it is primarily state-independent, (d) when the endophenotype and the illness co-segregate within families, and (e) when the endophenotype is also more prevalent in nonaffected family members compared to individuals of the general population (Gottesman & Gould, 2003).

In fact, there is convincing meta-analytical evidence for an association of the ERN with psychopathology (criterion a): ERN variations were found along the lines of internalizing and externalizing mental disorders (Lutz et al., 2021; Pasion & Barbosa, 2019) with enhanced ERN amplitudes for anxiety (Moser et al., 2013; Saunders & Inzlicht, 2020) and obsessive-compulsive disorders (Riesel, 2019) on the one hand, and attenuated ERN amplitudes for substance use disorder (Luijten et al., 2014) and attention deficit hyperactivity disorder (Shiels & Hawk, 2010) on the other hand. The ERN also fulfills criterion (b), as it has been found to be heritable from one generation to the other (Anokhin et al., 2008; Suor et al., 2021), and criterion (c), since a successful cognitive-behavioral therapy decreasing psychopathological symptoms has no effect on the ERN (Gorka et al., 2018; Hajcak et al., 2008; Kujawa et al., 2016; Ladouceur et al., 2018; Riesel et al., 2015). Lastly, corresponding alterations of the ERN have also been found in unaffected individuals with a family history of anxiety disorders, obsessive-compulsive disorders, and substance use disorder (Carrasco et al., 2013; Riesel et al., 2011; Riesel, Klawohn, et al., 2019), implying co-segregation within families (criterion d) and higher rates in first-degree relatives (criterion e). In addition, enhanced ERN amplitudes predict the onset of anxiety disorders (Meyer et al., 2015), all together supporting the notion that increased ERN amplitudes represent a trait-like neural risk marker or endophenotype for anxiety.

As mentioned above, the endophenotype approach aims at identifying specific mechanisms that lead to psychopathology. Unfortunately, the identification of mechanisms leading to pathological anxiety is complicated by the complex and heterogeneous nature of anxiety that likely overshadows specific associations between neural functioning and psychopathology. Regarding different dimensions of anxiety, the link between anxiety and elevated ERN amplitudes seems to be driven by worry as opposed to anxious arousal. This is supported by previous studies (Hajcak et al., 2003; Lin et al., 2015; Moran et al., 2012; Moser et al., 2012; but see Härpfer, Carsten, Spychalski, et al., 2020) as well as meta-analyses (Moser et al., 2013; Saunders & Inzlicht, 2020). Specifically, enhanced error monitoring in clinical populations such as generalized anxiety disorder (GAD; Meyer et al., 2012; Weinberg et al., 2010), social anxiety disorder (SAD; Endrass et al., 2014), health anxiety (Riesel et al., 2017), or obsessive-compulsive disorder (OCD; see Mathews et al., 2012; Riesel, 2019, for reviews and a meta-analysis), has been assumed to be related to transdiagnostically shared worry symptoms. Studies of healthy subjects and meta-analyses suggest that this relationship is evident across the full spectrum of worry symptoms including subclinical individuals. However, this assumption has been challenged in more recent studies, which tend to suggest that the association may be stronger in clinical

populations (Saunders & Inzlicht, 2020). In addition, gender seems to be an important moderator, and especially women show the expected association between worry and ERN (Moser et al., 2016).

Although the ERN is often discussed as an endophenotype, studies that experimentally manipulated state affect (e.g., Nigbur & Ullsperger, 2020; Wiswede, Münte, Goschke, et al., 2009), attentional biases (Klawohn, Hajcak, et al., 2020; Nelson et al., 2015), or the consequences of an error (i.e., punishment; Meyer & Gawlowska, 2017; Riesel et al., 2012) found that the ERN is susceptible for intra-individual variation. Utilizing the strengths of a causal intervention study, a recent approach that focused on the experimental reduction of worry (Schroder et al., 2018) showed that emotional expressive writing was associated with attenuated ERN amplitudes, which points to state worry as an affective state associated with ERN variations. This central and important study used a between group design, limiting conclusions because of possible confounds introduced by preexisting between-group variations. Thus, longitudinal within-between comparisons are important and promising to extend this line of research.

In summary, a variety of findings point to an association between ERN and worry. Based on this, the compensatory error-monitoring hypothesis (CEMH; Moser et al., 2013) postulates that anxious individuals need to employ compensatory effort, as reflected by an increased ERN, in order to overcome processing inefficiency that is caused by the distracting effects of worry on working memory capacity. As a result, the compensating effort leads to comparable levels of task performance. Another influential approach (Proudfit et al., 2013) assumes that enhanced error monitoring of anxious individuals is caused by a pronounced trait-like sensitivity to uncertain threats (e.g., errors): This threat sensitivity temporally increases defensive motivation in an uncertain and potentially threatening situation, making errors motivationally more relevant and leading to greater error monitoring. In this view, worrying is a by-product that has developed as a maladaptive coping strategy of anxious individuals associated with heightened threat sensitivity. In accordance with the endophenotype approach, the authors argue that ERN variations are due to differences in threat sensitivity—a stable trait—not due to state-dependent temporarily efforts to compensate for the distracting effects of worries. Overall, both approaches converge on the idea that anxiety is associated with an increased ERN, although they differ in their assumptions on the underlying mechanisms of this relationship.

This preregistered study (Härpfer et al., 2020) aimed at disentangling the relationships between trait and state worry and error-related brain activity by utilizing both a cross sectional observational design (similar to many previous studies) as well as longitudinal randomized controlled experimental design manipulating state worry (allowing causal inferences). We wanted to examine whether worry interventions cause alterations in neural signals of performance monitoring. To this end, we assessed the baseline ERN of 90 participants (T0), which were then randomly assigned to one of three groups (two experimental groups with either a worry induction or reduction; one passive control group with no worry intervention). Following the intervention, participants performed another flanker task to determine potential alterations of their ERN (T1). Our overall research question targeted the relationship between the ERN and trait worry as well as state worry, i.e., whether this link can be found in both cross sectional and longitudinal comparisons. As preregistered, we expected that trait worry would be associated with increased ERN amplitudes at T0 and that state worry would cause alterations in ERN amplitudes in such way that, relative to the control group, ERN amplitudes between T0 and T1 would increase in the worry induction group and decrease in the worry reduction group. Likewise, we exploratorily investigated associations of trait and state worry and the CRN cross-sectionally and longitudinally. In a first meta-analysis, there was no evidence for a link between anxiety and the CRN (Moser et al., 2013). In contrast, a more recent meta-analysis found a small but significant association of anxiety and the CRN, but this was not moderated by the type of anxiety, such as worry (Saunders & Inzlicht, 2020).

2 | METHOD

2.1 | Participants

When preregistering the hypotheses and methods of our study (Härpfer et al., 2020), an a priori sample size calculation was conducted using G*Power, version 3.1.9.7 (Faul et al., 2009, 2007). Based on the results of a previous study (Schroder et al., 2018), we assume to find medium-sized effects of the worry interventions. Paired comparisons of subgroups (two-sided dependent *t*-tests) can detect medium-sized effects (Cohen's $d > 0.60$) with a sample size of $n = 24$ per group, a power of 80%, and an alpha of 0.05 (Cohen, 1992). Specifications of the sample size calculation can be found in the supplementary materials (Figure S1). As preregistered, participants were recruited until $n = 30$ complete and evaluable data sets per group were collected ($n = 4$ were excluded and replaced, for details see section 'Electrophysiological Recording and Processing'). In light of the mixed previous findings, this enabled detecting possible smaller effects. Therefore, our final sample consisted of $N = 90$ right-handed university students (66 identified as

female) aged 18 to 30 years ($M = 23.50$, $SD = 3.12$). They received either course credit or monetary compensation for their participation.

Participants were required to speak German as a native language, to have normal or corrected-to-normal vision, and to be able to provide written informed consent. Exclusion criteria for all subjects included a history of any neurological disorder, current or lifetime diagnosis of a substance-related disorder, schizophrenia spectrum disorder, bipolar disorder, and use of benzodiazepines during the last week or of neuroleptic medication during the last three months. At the time of participation, $n = 15$ participants were currently medicated with at least one drug including oral contraceptives ($n = 7$), antidepressants ($n = 4$), dermatological drugs against acne ($n = 3$), and thyroid hormones ($n = 2$).

Regarding clinical status, $n = 14$ participants had a current or lifetime diagnosis for at least one mental disorder including a major depressive episode ($n = 5$ lifetime), anorexia nervosa ($n = 3$ lifetime), bulimia nervosa ($n = 3$ lifetime), specific phobia ($n = 2$ current, $n = 1$ lifetime), panic disorder ($n = 2$ lifetime), obsessive-compulsive disorder ($n = 1$ lifetime), pain disorder ($n = 1$ lifetime), and posttraumatic stress disorder ($n = 1$ lifetime). Note, that information on clinical status was missing for seven participants.

2.2 | Procedure

Participants received verbal and written information of the objectives and methods of the study and gave written informed consent. Mental disorders were assessed by trained personnel using the Structured Clinical Interview for DSM-5—clinical version (SCID-5-CV; Beesdo-Baum et al., 2019; First et al., 2016). During the laboratory assessment (Figure 1), participants were asked to identify at least three worry topics with high personal relevance that could be used at a later point in time. They were given a list of possible content domains as examples (Arch & Craske, 2006; Boehnke et al., 1998) including social relations, achievement/work, money/economics, health, and safety, but they could also write down any other ideographic worry topic. This identification procedure was adapted from previous worry intervention studies (Arch & Craske, 2006; Oathes et al., 2008; Vasey & Borkovec, 1992; Verkuil et al., 2009).

After that, participants completed several questionnaires, performed a flanker task (T0), and had a four-minute resting state assessment (T0). The resting state served for another research question and results will be reported elsewhere. Next, participants were randomly assigned to one of three groups (induction, reduction, control) and parallelized across groups regarding gender. This randomization procedure ensured a balanced design with equally sized groups. The experimental groups received either an eight-minute worry induction or reduction; the passive control group did not receive any intervention and paused for an equivalent amount of time. The interventions were informed by previous literature. We aimed at selecting interventions that wereas potent and standardized as possible. Regarding the length of the interventions, we considered eight minutes as the best tradeoff between sufficiently inducing worries in the induction group, yet preventing participants to start habituating, and reducing worry effectively in the reduction group by allowing them enough time to reflect.

The worry induction was consistent with previous studies using a classical induction paradigm to create a worrisome and ruminative state (e.g., Arch & Craske, 2006; Borkovec & Inz, 1990; Fisher & Newman, 2013; Lyonfields et al., 1995; McLaughlin et al., 2007; Oathes et al., 2008; Ray et al., 2009; Ruscio & Borkovec, 2004; Thayer et al., 1996; Vasey & Borkovec, 1992; Verkuil et al., 2009). In accordance with the Catastrophizing Interview Technique (Vasey & Borkovec, 1992), participants were instructed to worry as intensively as they can until the experimenter asked them to stop. Participants should think about worst-case scenarios, the consequences for themselves as well as for significant others, and how badly they would feel if their worries became reality. They were allowed to switch back and forth between worry topics to facilitate rumination; but they were instructed to return to the chosen worry topics if their thoughts drifted away.

The reduction paradigm was also based on previous research on emotional expressive writing (e.g., Baddeley & Pennebaker, 2011; Gortner et al., 2006; Pennebaker & Beall, 1986; Pennebaker & Francis, 1996; Ramirez & Beilock, 2011; Sayer et al., 2015; Schroder et al., 2018). Participants were asked to write as openly as possible about their thoughts and feelings regarding their worry topics until the experimenter asked them to stop. Participants were encouraged to explore their thoughts and feelings in a completely non-judgmental manner and they were informed that they could keep their essay to ensure confidentiality. Subsequently, all participants performed another flanker task (T1), followed by a two minute booster session of the intervention or pause, and another four-minute resting state (T1).

Manipulation checks were implemented at several points throughout the assessment (i.e., before the T0 and T1 flanker, before the T0 and T1 resting state, before the intervention/pause, and after the T1 resting state) in order to track intraindividual changes of participants'

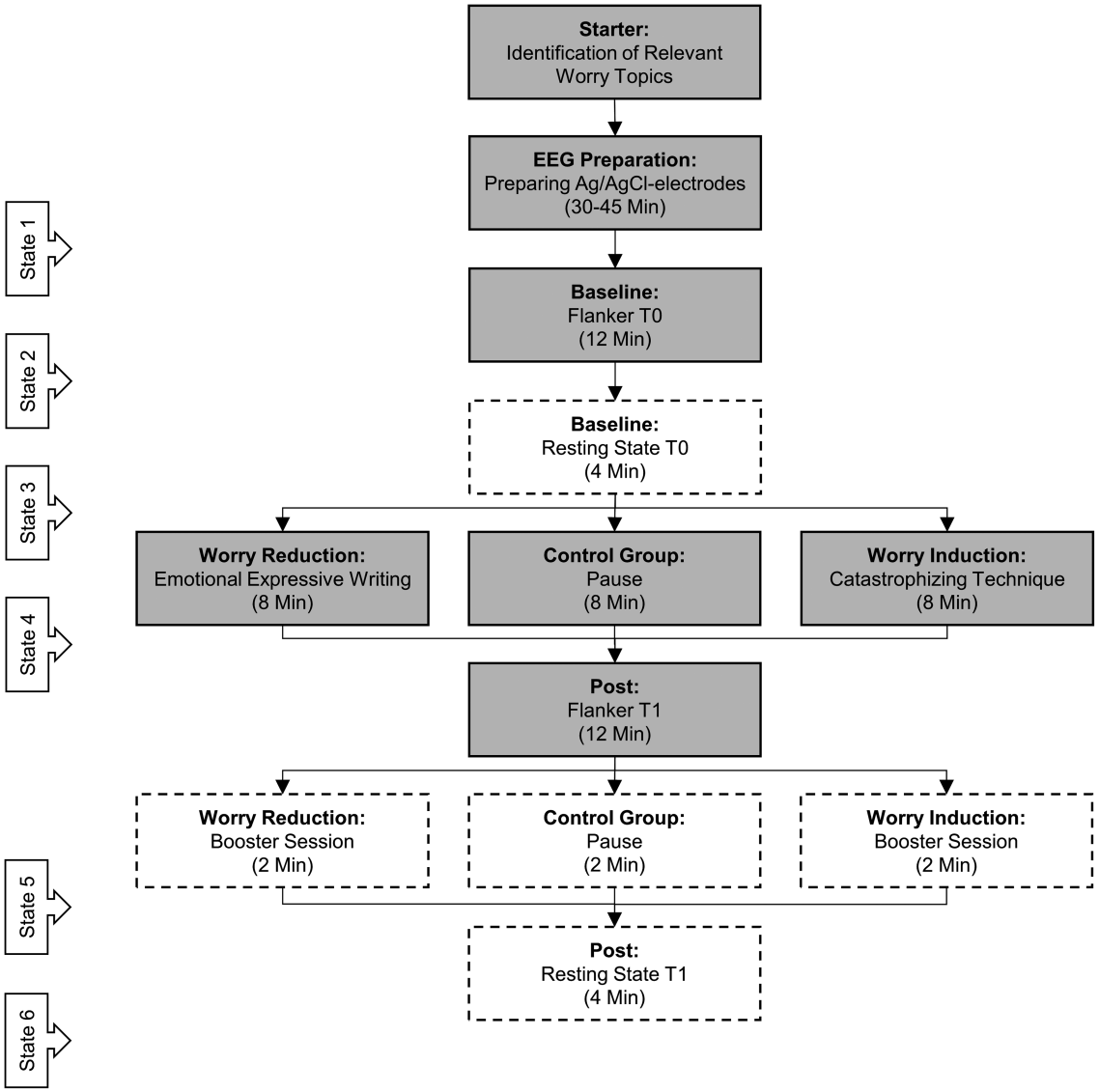

FIGURE 1 Flowchart of the study procedure. Boxes with surrounding dashed lines were part of another research question whose results will be presented elsewhere. Duration of each task is approximative. State measurements throughout the study ensured the tracking of fluctuations in state worry, state arousal, and state affect

mood over time. These short manipulation checks were introduced by 'At this moment…' and consisted of three domains with each two items: worry ('… how worried are you?', '… how much do you ruminate?'), arousal ('… how aroused are you?', '… how tensed are you?'), and affect ('… how many positive feelings do you feel [e.g., joyful, enthusiastic, active]?', 'how many negative feelings do you feel [e.g., angry, sad, anxious]?'). Participants rated each item on a on a forced-choice visual analogue scale ranging from 0 to 100. Scores of the three domains were aggregated by averaging the respective two items (the item for negative affect was first inverted before averaging). The local ethics committee approved that the study procedure is in accordance with the Declaration of Helsinki (World Medical Association, 2013).

2.3 | Questionnaires

During laboratory assessment, following questionnaires were administered: Trait worry was measured by the Penn State Worry Questionnaire (PSWQ; 16 items, 5-point Likert scale 1–5; $\alpha = .93$; Glöckner-Rist & Rist, 2014; Meyer et al., 1990), anxious arousal by the respective subscale of the Mood and Anxiety Symptom Questionnaire (MASQ-AA; 17 items, 5-point Likert scale 1–5; $\alpha = .87$; Watson & Clark, 1991; Watson et al., 1995), trait anxiety by the respective subscale of the State-Trait-Anxiety Inventory (STAI-T; 20 items, 4-point Likert scale 1–4; $\alpha = .89$; Laux et al., 1981; Spielberger et al., 1970), obsessive-compulsive symptoms by the Obsessive-Compulsive Inventory Revised (OCI-R; 20 items, 5-point Likert scale

0–4; $\alpha = .85$; Foa et al., 2002; Gönner et al., 2007), depression symptoms by the Beck Depression Inventory (BDI-II; 21 items, 4-point Likert scale 0-3; $\alpha = .92$; Beck et al., 1996; Hautzinger et al., 2006), alcohol consumption by the Alcohol Use Disorders Identification Test (AUDIT; 10 items, 5-point Likert scale 0–4; $\alpha = .75$; Babor et al., 2001), and handedness by the modified Edinburgh Handedness Inventory (EHI; 10 items, 5-point Likert scale −10 to +10; $\alpha = .75$; Loffing et al., 2014; Oldfield, 1971).

2.4 | Task

Participants sat in a dimly lit, electrically shielded cabin approx. 24 inches in front of a 19-inch LCD monitor with a resolution 1920 × 1080 pixels and a refresh rate of 120 Hz. A speeded arrowhead version of the flanker task (Eriksen & Eriksen, 1974) with a set of five horizontally aligned arrows (one target, four flankers) was displayed using Presentation Software (Neurobehavioral Systems, Inc., Albany, California). The set of arrows was approx. 6.2° in width and approx. 1.0° in height with an equal number of trials pointing pseudo randomly either into same (<<<<< or >>>>>) or opposite directions (<<><< or >><>>). Each trial included fixation (200–1200 ms), presentation of arrow stimuli (100 ms), and response (max. 800 ms). Participants were instructed to indicate the direction of the center arrow by pressing a key with their respective left or right index finger as quickly and accurately as possible, which was practiced in 20 trials before the first flanker task. Each of the five blocks consisted of 80 trials, which equals 400 trials in total. After each block, participants received a performance feedback asking them to respond faster, irrespective of their actual response times. This procedure was used for three reasons: First, to achieve sufficient error trials in both flanker tasks; second, a reasonable length of the tasks; and third, there is evidence that neural differences between OCD patients and healthy control participants are pronounced under speed conditions (Riesel, Kathmann, et al., 2019).

Accuracy was defined as the percentage of correct responses of the response trials, response times as the time difference between the onset of arrows and the respective correct or incorrect response, and post-error slowing (PES) was quantified using the robust measurement method (i.e., the average response time difference between the last correct trial before an error and the first correct trial after an error; Dutilh et al., 2012).

2.5 | Electrophysiological recording and processing

The setup of recording and processing parameters of the electrophysiological data were mostly as preregistered. Whenever we deviated from the preregistration, we clearly state which other parameters were chosen. As preregistered, using 61 passive Ag/AgCl-electrodes mounted on a cap with equidistant and concentric electrode sites (Easycap, Herrsching, Germany) and two 32-channel BrainAmp amplifiers (Brain Products GmbH, Gilching, Germany), EEG signals were recorded with a band-pass filter of 0.01 to 250 Hz and digitized continuously at a sampling rate of 1000 Hz. Recording reference was located between AF3 and Fz, the ground electrode between AF4 and Fz. External electrodes were placed at the left infraorbital site for vertical eye movements and at the neck for the electrocardiogram. Impedances were always kept below 5 kΩ, however, we did not preregister a maximum impedance threshold.

Processing of the EEG data was performed in Brain Vision Analyzer (Brain Products GmbH, Gilching, Germany). First, a band-pass filter with a low cut-off of 0.1 Hz and a high cut-off of 30 Hz (24 dB/oct roll-off) as well as a notch filter of 50 Hz was applied to continuous EEG data. Subsequently, ocular artifacts were corrected by using an independent component analysis (ICA; Jung et al., 2000), whereby relevant components were semi automatically identified and manually checked by visual inspection of the scalp topography, the component activation, and the inspection of the corrected EEG signal. Continuous data were then re-referenced to the average of all scalp electrodes and segmented into response-locked epochs (−500 to 800 ms). Segments containing artifacts (i.e., absolute voltage range exceeding 200 μV, voltage step exceeding 50 μV between consecutive data points, or maximum voltage difference of less than 0.5 μV within 100 ms intervals) were removed. Due to equipment failure ($n = 1$) and low data quality (i.e., more than 25% of all trials containing artifacts; Luck, 2014; $n = 3$) participants were excluded and replaced before data analysis. In the final sample, artifact rejection caused minimal data loss in both the first ($M = 0.99\%$, $SD = 0.02\%$, Max. = 13.00%) and the second flanker ($M = 0.68\%$, $SD = 0.02\%$, Max. = 10.40%). None of participants was falling below a threshold of less than six usable error segments in either flanker task (Olvet & Hajcak, 2009). We did not preregister a fixed baseline interval but specified a visual inspection procedure to identify a baseline that ensures aligned waveforms in the pre-response interval. Consequently, segments were corrected for the baseline interval of −500 to −300 ms and averaged separately for correct and erroneous responses.

The preregistered procedure to determine the electrode at which the ERN and CRN were quantified was also based on visual inspection. At FCz, the signal was maximal after inspecting the grand-averaged waveforms and topographical distributions. As preregistered, quantification was based on peak-to-peak amplitudes, which

is the difference between the most negative peak in the 0–100 ms post-response interval and the most positive peak in a −100 to 0 ms pre-response window. ERPs revealed good psychometric properties for both the ERN (r_{T0} = .85; r_{T1} = .85) and CRN (r_{T0} = .96; r_{T1} = .92) as indicated by Spearman-Brown corrected correlations of odd- and even-numbered trials. In order to ensure the robustness of result patterns and to account for the potential influence of methodological choices in ERP research (Klawohn, Meyer, et al., 2020; Sandre et al., 2020), we decided in our preregistration to report results of our main analyses for other commonly used scoring strategies (Tables S1 and S2 in the Supporting Information), including mean amplitude between 0 and 100 ms after response, adaptive mean amplitude around the most negative peak (±20 ms) in the 0–100 ms post-response interval, and the difference measure (i.e., ΔERN) using mean amplitude between 0 and 100 ms after correct and erroneous responses.

2.6 | Data analysis

As preregistered, frequentist analyses were conducted using IBM SPSS Statistics, version 25.0 (SPSS, Inc., Chicago) with a significance level of $\alpha = .05$. Differences in demographic (age, education, handedness) and clinical characteristics (all questionnaires) between the three groups were tested by one-way analysis-of-variance (ANOVAs) with group (induction, reduction, control) as between-subject factor. For the manipulation checks, 3 × 6 mixed-measures ANOVAs with group (induction, reduction, control) as between-subject factor and time (State 1 to 6) as within-subject factor were performed separately for worry, arousal, and affect. Note, that the manipulation check analysis was not specified in the preregistration.

Cross sectional hypotheses on ERPs (ERN and CRN) and behavioral data (accuracy, response times for correct and incorrect response, and PES) were analyzed, as preregistered, using separate multiple linear regression models including the predictors gender, PSWQ, and gender × PSWQ to investigate the association of worry and ERPs as well as the role of gender as a potential moderator of the relationship between worry and ERPs (Moran et al., 2012; Moser et al., 2016). Exploratively, we also conducted separate independent samples *t*-tests between participants that had any current or lifetime diagnosis and those who were not diagnosed with a disorder to investigate whether these two groups differ in ERN or CRN amplitudes.

Longitudinal hypotheses on ERPs (ERN and CRN) and behavioral data (response times for correct and incorrect response) were analyzed, as preregistered, using separate 2 × 2 × 3 mixed-measures ANOVAs with time (T0, T1) and response (correct, incorrect) as within-subject factors as well as group (induction, reduction, control) as between-subject factor. Accuracy and PES were tested by 2 × 3 mixed-measures ANOVAs with time (T0, T1) as within-subject factor and group (induction, reduction, control) as between-subject factor. Greenhouse-Geisser correction was applied if the assumption of sphericity was violated. Follow-up analyses were conducted if results revealed significant interactions. Mirroring the frequentist analyses, we also conducted explorative Bayesian statistical analyses using JASP, version 0.15.0.0. (JASP Team, 2021) allowing the quantification of evidence for the null hypothesis (i.e., the absence of an effect of worry interventions on ERPs; Keysers et al., 2020). Complementary mixed-measures Bayesian ANOVAs were performed with weakly informative priors (r scale fixed effects = 0.5; random effects = 1; covariates = 0.354; van Doorn et al., 2021) resulting in a Bayes factor (BF) that quantifies the evidence for the alternative hypothesis over the null hypothesis (BF_{10}) or for the null hypothesis over the alternative hypothesis ($BF_{01} = 1/BF_{10}$) of a specific model that includes the predictor of interest. For example, the $BF_{10} = 20$ implies that the alternative hypothesis (i.e., there is an effect) is 20 times more likely than the null hypothesis (i.e., the absence of an effect) in light of the data. The Bayes factor included (BF_{Incl}) reflects the inclusion probability of a predictor across all models excluding this specific predictor. Although the Bayes factor is a continuous metric, we refer to a heuristic (Raftery, 1995) when interpreting the BF_{10} and BF_{Incl}: Evidence can be weak (BF = 1–3 or 0.33), positive (BF = 3–20 or 0.33–0.05), strong (BF = 20–150 or 0.05–0.0067), or very strong (BF > 150 or 0.0067–0).

3 | RESULTS

3.1 | Demographic and clinical data

Group-specific means and standard deviations of demographical and clinical data can be found in Table 1. As preregistered, we examined potential group differences in these data. However, groups displayed no significant differences regarding demographic and clinical data. In an additional explorative analysis, no differences between groups were found regarding current or lifetime clinical status, χ^2 (2) = 0.06, p = .969.

3.2 | Manipulation check

Results of the manipulation checks revealed significant differences of participants' mood throughout the study

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TABLE 1 Demographical and clinical data of all groups

	Sample ($n = 90$)		Control ($n = 30$)		Induction ($n = 30$)		Reduction ($n = 30$)		Group Comparison			
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*	*df*	η_p^2	*p*
Demographical												
Gender (f/m)	66/24		22/8		22/8		22/8					
Age	23.50	3.12	23.57	3.02	23.20	3.34	23.73	3.07	0.23	2, 87	0.01	.799
Education	12.34	0.60	12.33	0.66	12.47	0.51	12.23	0.63	1.14	2, 87	0.03	.326
Clinical												
PSWQ	45.73	10.93	43.17	9.97	46.83	11.12	47.20	11.54	1.26	2, 87	0.03	.290
MASQ-AA	27.14	8.46	27.77	9.21	25.70	8.04	27.97	8.16	0.66	2, 87	0.01	.522
STAI-T	38.33	8.28	37.33	7.22	37.30	7.73	39.97	9.72	0.89	2, 87	0.02	.415
OCI-R	12.86	8.53	12.77	8.30	14.33	9.36	11.47	7.90	0.85	2, 87	0.02	.432
BDI-II	5.72	6.82	6.40	7.85	4.30	4.56	6.47	7.55	0.98	2, 87	0.02	.379
AUDIT	4.94	3.39	5.00	3.30	4.20	2.89	5.63	3.87	1.36	2, 87	0.03	.263
EHI	81.11	16.21	81.83	15.40	82.00	15.29	79.50	18.21	0.22	2, 87	0.01	.804

Note. Gender (f = female, m = male); age and education in years.

Abbreviations: AUDIT, alcohol use disorders identification test; BDI-II, Beck Depression Inventory II; EHI, Edinburgh Handedness Inventory (Modified); MASQ-AA, Mood and Anxiety Symptom Questionnaire; OCI-R, Obsessive-Compulsive Inventory; PSWQ, Penn State Worry Questionnaire; STAI-T, State-Trait-Anxiety Inventory (Trait Subscale).

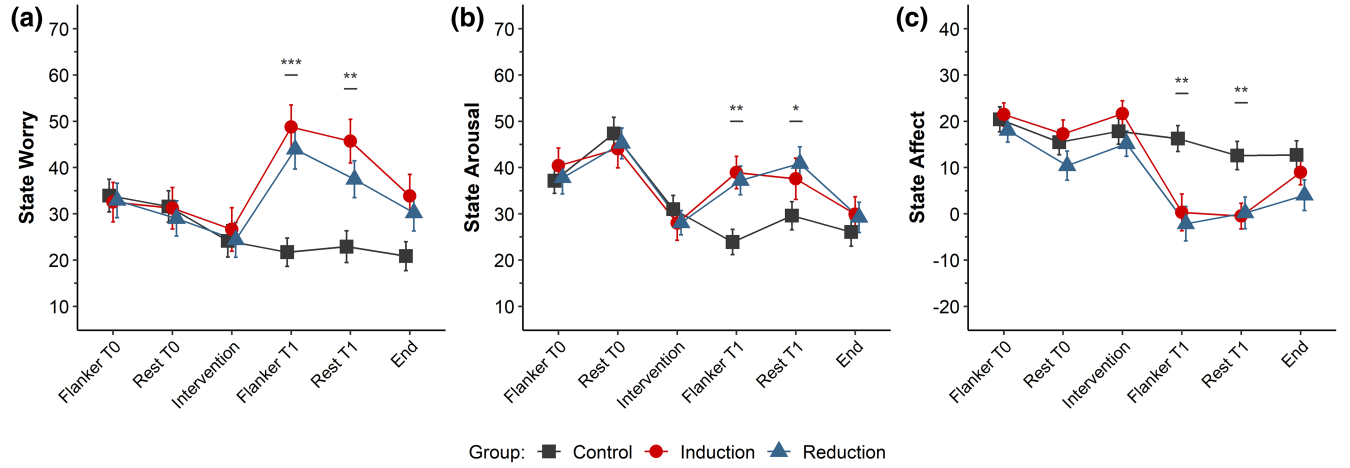

FIGURE 2 State measurements of state worry, state arousal, and state affect. State measurements tracked fluctuations of state worry (a), state arousal (b), and state affect (c) throughout the study and were assessed before each task. Each scale consisted of two items that were rated on a visual analogue scale. For state worry and state arousal, the potential range was 0 to 100, for state affect it was -50 to 50. Error bars represent one standard error. Asterisks indicate significant differences between groups at each time point: ***$p < .001$. **$p < .01$. *$p < .05$

(Figure 2). There was no main effect of group, $F(2, 87) = 2.34$, $p = .102$, $\eta_p^2 = 0.05$, but a significant main effect of time, $F(3.60, 312.96) = 16.58$, $p < .001$, $\eta_p^2 = 0.16$, and a significant interaction effect of group × time, $F(7.19, 312.96) = 10.46$, $p < .001$, $\eta_p^2 = 0.19$, in the ANOVA testing state worry. Follow-up one-way ANOVAs indicated that groups did not differ regarding state worry, neither before the first flanker, $F(2, 87) = 0.04$, $p = .965$, $\eta_p^2 = 0.00$, nor before the intervention, $F(2, 87) = 0.12$, $p = .890$, $\eta_p^2 = 0.00$. However, state worry differed between groups after the intervention, $F(2, 87) = 12.38$, $p < .001$, $\eta_p^2 = 0.22$, indicating that both the induction and the reduction group reported higher levels of state worry compared to the control group (Figure 2). In summary, the induction and reduction group did not differ from the control group regarding state worry before the intervention, but they did so after the intervention during the second flanker and resting state assessment. Consequently, the worry induction successfully increased levels of state worry in the induction group. However, instead of lower levels of state worry in the reduction group, participants reported higher levels of state worry after the emotional expressive writing, which was the opposite pattern of that expected.

Similar patterns were found for state arousal (Figure 2), where there was no main effect of group, $F(2, 86) = 0.73$, $p = .487$, $\eta_p^2 = 0.02$, but a significant effect of time, $F(4.25, 365.22) = 27.28$, $p < .001$, $\eta_p^2 = 0.24$, and group × time, $F(8.39, 365.22) = 3.59$, $p < .001$, $\eta_p^2 = 0.08$, indicating an increase in arousal as a result of both interventions. State affect also varied as a function of group and time (Figure 2) with no main effect of group, $F(2, 86) = 2.82$, $p = .065$, $\eta_p^2 = 0.06$, but a significant effect of time, $F(3.67, 315.36) = 38.64$, $p < .001$, $\eta_p^2 = 0.31$, and group × time, $F(7.33, 315.36) = 5.39$, $p < .001$, $\eta_p^2 = 0.11$. As with state worry and arousal, state positive affect decreased as a result of the worry induction and reduction.[1] Taken together, our interventions not only altered levels of state worry, but also of state arousal and state affect, such that the induction and reduction group did not differ from the control group before the intervention, but did so after the intervention during the second flanker and resting state assessment.

3.3 | Event-related potentials

Group-specific means and standards deviation of the ERN and CRN at T0 and T1 can be found in Table 2.

3.3.1 | Cross-sectional analyses of trait worry and ERPs

The preregistered cross-sectional analyses for the ERN (T0) revealed a significant main effect of gender, $b = 2.65$, $SE = 1.05$, $p = .014$, and an interaction effect of gender × PSWQ, $b = 0.32$, $SE = 0.10$, $p = .002$, but no main effect of PSWQ, $b = -0.06$, $SE = 0.04$, $p = .221$, $R^2 = .13$, $F(3, 86) = 4.23$, $p = .008$. As indicated by Figure 3, higher levels of trait worry were associated with larger (i.e., more negative) ERN amplitudes in female participants, whereas in male participants, higher levels of trait worry were associated with smaller (i.e, more positive) ERN amplitudes. Regarding the CRN (T0), we observed a main effect of

[1]The reason why there is one degree of freedom less in the analyses for arousal and affect was missing data of one participant due to a technical malfunction.

TABLE 2 Group-specific electrophysiological and behavioral data

	Control ($n = 30$)				Induction ($n = 30$)				Reduction ($n = 30$)			
	T0		T1		T0		T1		T0		T1	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
ERPs												
ERN (μV)	−7.38	4.33	−8.17	4.67	−7.76	4.72	−8.08	5.25	−6.69	3.30	−6.77	4.07
CRN (μV)	−3.13	2.37	−2.83	2.36	−3.31	2.44	−2.22	2.20	−2.65	1.59	−2.28	1.69
Behavior												
n missing	23.80	54.54	7.03	10.65	15.77	38.63	14.60	42.16	11.70	17.65	6.27	9.33
n correct	329.10	61.54	346.90	44.14	328.63	63.60	327.30	62.18	337.60	34.31	339.73	36.68
n incorrect	47.10	37.62	46.07	40.70	55.60	37.01	58.10	43.32	50.70	29.25	54.00	34.86
Accuracy (%)	87.46	9.51	88.21	10.54	84.72	11.80	84.66	12.51	86.92	7.55	86.27	8.90
RT correct (ms)	437.83	55.41	419.06	51.09	425.65	50.49	397.85	59.59	430.49	53.96	413.82	59.47
RT incorrect (ms)	382.50	58.91	371.18	60.46	365.11	56.58	351.23	66.71	368.09	72.61	362.56	72.41
PES (ms)	39.10	28.45	27.88	30.99	44.56	25.63	25.82	28.42	43.84	24.34	27.47	20.01

Abbreviations: CRN, correct-response negativity; ERN, error-related negativity; ERPs, event-related potentials; PES, post-error slowing; RT, response time.

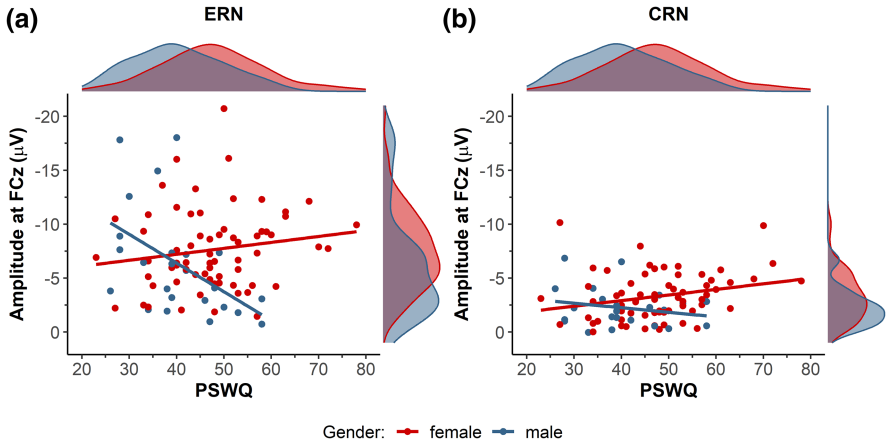

FIGURE 3 Scatter plots depicting the relationship between trait worry and ERN (a) and CRN (b). CRN, correct-response negativity; ERN, error-related negativity; PSWQ, Penn State Worry Questionnaire. Data points, regression lines, and densities were grouped by gender.

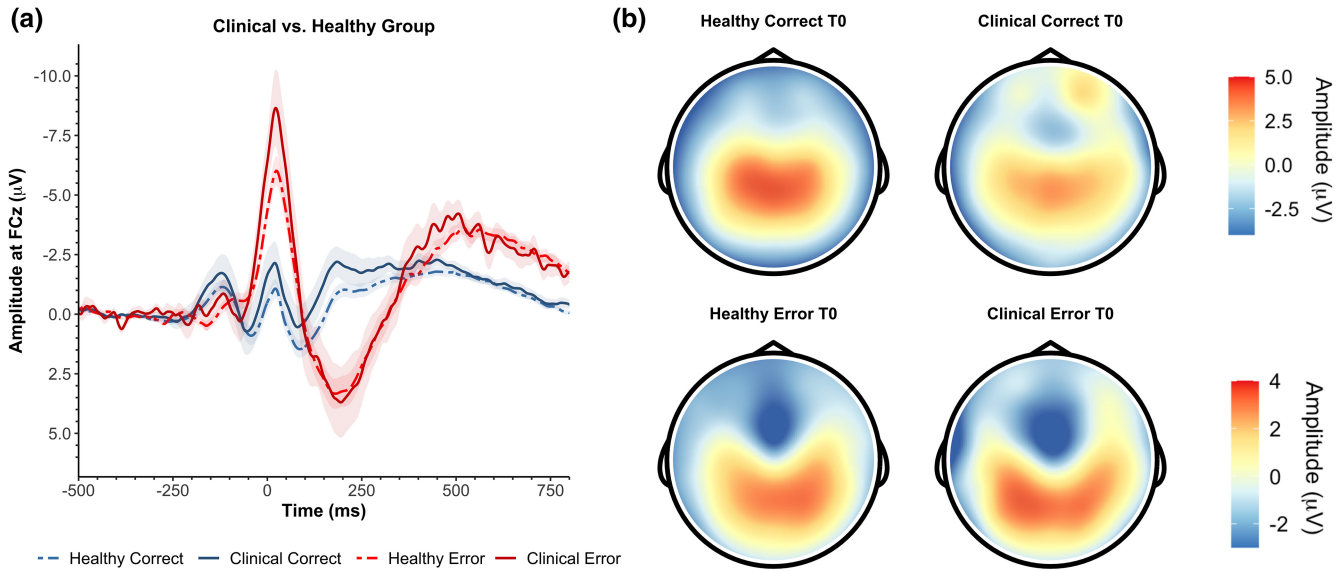

FIGURE 4 Response-locked grand-averaged waveforms of correct and erroneous trials at baseline (T0) for the clinical group with a history of internalizing disorders and the unaffected healthy group (a). Corresponding topographic head maps of the ERN and CRN (b)

gender and PSWQ: Females compared to males showed a higher CRN, $b = 1.20$, $SE = 0.56$, $p = .034$. Furthermore, increasing levels of trait worry were associated with larger CRN amplitudes, $b = -0.05$, $SE = 0.02$, $p = .031$, irrespective of gender since no gender × PSWQ interaction was observed, $b = 0.09$, $SE = 0.05$, $p = .075$, $R^2 = .11$, $F(3, 86) = 3.44$, $p = .020$.

In addition, we exploratively examined the impact of current or lifetime psychopathology on ERPs: An independent samples t-test compared participants who had any current or lifetime diagnosis of at least one internalizing disorder ($n = 14$) and those who were not diagnosed with a disorder ($n = 69$). Results indicated a larger ERN amplitude in the clinical group ($M = -9.96$, $SD = 4.86$) compared to the healthy group ($M = -6.81$, $SD = 3.68$), $t(81) = 2.76$, $p = .007$, $d = 0.81$. No evidence emerged for CRN associations with lifetime clinical status, $t(81) = 1.45$, $p = .151$, $d = 0.43$ (Figure 4).

3.3.2 | Longitudinal analyses of state worry and ERPs

In the preregistered longitudinal analyses for the ERN and CRN (Figure 5), we found larger amplitudes for erroneous compared to correct responses, $F(1, 87) = 155.49$, $p < .001$, $\eta_p^2 = 0.64$. Further, we found a significant interaction of response × time, $F(1, 87) = 8.92$, $p = .004$, $\eta_p^2 = 0.09$, driven by a reduction of the CRN at T1, while no evidence emerged for ERN differences between T0 and T1. Importantly, neither a main nor interaction effect including group were observed, suggesting the absence of evidence for worry interventions modulating ERN or CRN. Further, none of the other main or interaction effect reached significance (all $ps > .45$). Detailed statistics are summarized in Table 3.

To further trace the missing intervention effect, we performed non-preregistered Bayesian analyses. These complementary Bayesian analyses yielded very strong

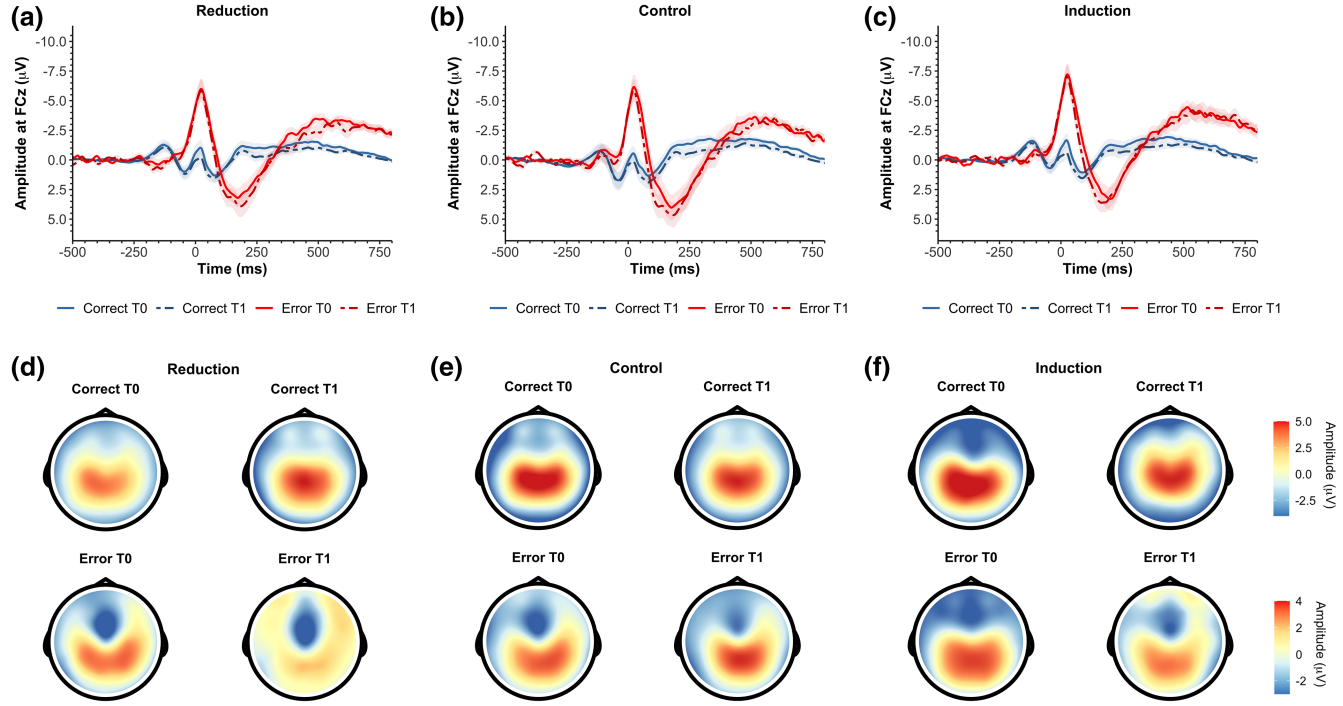

FIGURE 5 Response-locked grand-averaged waveforms of correct and erroneous trials at baseline (T0) and after the intervention (T1) for the worry reduction group (a), passive control group (b), and worry induction group (c). Corresponding topographic head maps of the ERN and CRN for the worry reduction group (d), passive control group (e), and worry induction group (f)

	F	*df*	η_p^2	*p*	$BF_{Incl.}$
Response	155.49	1, 87	0.64	**<.001**	∞
Response × Time	8.92	1, 87	0.09	**.004**	0.182
Response × Group	0.46	2, 87	0.01	.632	0.047
Response × Time × Group	0.77	2, 87	0.02	.465	0.000
Time	0.21	1, 87	0.00	.648	0.078
Time × Group	0.80	2, 87	0.02	.453	0.006
Group	0.80	2, 87	0.02	.453	0.084

Note. Bayes factor included (BF_{Incl}), correct-response negativity (CRN), error-related negativity (ERN), group (induction, reduction, control), response (correct, incorrect), time (T0, T1). Significant $p < .05$ printed in bold.

TABLE 3 Results of frequentist and Bayesian ANOVA for ERN and CRN with factors response, time, and group

evidence for response type since all models including response type compared to the null model were more likely given the data (BF_{10} > 2.61e+39), with the most likely model encompassing response type alone (BF_{10} = 8.91e+43). Furthermore, there was positive to strong evidence against including group, time, or an interaction involving one of them into the model (Table 3) suggesting that neither ERN nor CRN varied as a function of state worry interventions.

Past research suggests that the link between anxiety and larger ERN amplitudes is larger for females (Moser et al., 2016). Therefore, in an explorative approach, we analyzed potential ERN variations of only female participants in our sample (n = 22 per group) using a 2 × 3 mixed-measures ANOVA with time (T0, T1) as a within-subject factor and group (induction, reduction, control) as a between-subject factor. Results indicated no significant effect of group, $F(1, 63) = 0.57$, $p = .567$, $\eta_p^2 = 0.18$, time, $F(1, 63) = 2.92$, $p = .093$, $\eta_p^2 = 0.04$, or time × group, $F(2, 63) = 0.81$, $p = .451$, $\eta_p^2 = 0.03$. Taken together, in the female sample, we could not find evidence for ERN variations from T0 to T1 by our state worry interventions.

3.4 | Behavioral data

Group-specific means and standard deviations of behavioral data at T0 and T1 can be found in Table 2.

3.4.1 | Cross-sectional analyses of trait worry and behavioral data

As preregistered, we also investigated whether behavioral data of the flanker (T0) varied as a function of trait worry and gender. None of the predictor significantly predicted accuracy; neither gender, $b = -0.01$, $SE = 0.03$, $p = .735$, PSWQ, $b = 0.00$, $SE = 0.00$, $p = .855$, nor the interaction of gender × PSWQ, $b = -0.00$, $SE = 0.00$, $p = .074$, $R^2 = .05$, $F(3, 86) = 1.37$, $p = .259$.

Response times were also not significantly predicted by gender, $b = -24.03$, $SE = 13.75$, $p = .084$ (correct trials), $b = -18.86$, $SE = 16.28$, $p = .250$ (incorrect trials), PSWQ, $b = 1.04$, $SE = 0.59$, $p = .079$ (correct trials), $b = 1.12$, $SE = 0.69$, $p = .111$ (incorrect trials), nor the interaction of gender × PSWQ, $b = -0.50$, $SE = 1.29$, $p = .646$ (correct trials), $b = 0.78$, $SE = 1.53$, $p = .609$ (incorrect trials), $R^2 = .09$, $F(3, 86) = 2.90$, $p = .040$ (correct trials), $R^2 = .10$, $F(3, 86) = 3.06$, $p = .032$ (incorrect trials).

However, we found an effect of gender on PES: Males compared to females showed less slowing after committing an error, $b = -20.55$, $SE = 6.72$, $p = .003$. No effect on PES emerged for PSWQ, $b = -0.06$, $SE = 0.29$, $p = .847$, or the interaction of gender × PSWQ, $b = -0.39$, $SE = 0.63$, $p = .537$, $R^2 = .10$, $F(3, 86) = 3.26$, $p = .025$.

When comparing participants with and without a current or lifetime diagnosis of a mental disorder in explorative analyses, we found no differences regarding accuracy, $t(81) = 0.10$, $p = .923$, $d = 0.03$, nor response times, $t(81) = -0.06$, $p = .953$, $d = -0.02$ (correct trials), $t(81) = 0.51$, $p = .611$, $d = 0.15$ (incorrect trials). However, PES was significantly longer in the clinical group compared to the healthy group, $t(81) = -2.19$, $p = .031$, $d = -0.64$.

3.4.2 | Longitudinal analyses of state worry and behavioral data

Following our preregistration, we examined the behavioral data across both sessions and groups. Across groups (induction, reduction, control) and sessions (T0, T1), accuracy was comparable, as indicated by the absence of an effect of group, $F(2, 87) = 0.30$, $p = .446$, $\eta_p^2 = 0.02$, or time, $F(1, 87) = 0.00$, $p = .986$, $\eta_p^2 = 0.00$, in the mixed measures ANOVA. The interaction of group × time was also not statistically significant, $F(2, 87) = 0.28$, $p = .756$, $\eta_p^2 = 0.01$. For response times, we observed a main effect of response type and session: Participants showed faster responses for incorrect compared with correct trials, $F(1, 87) = 296.79$, $p < .001$, $\eta_p^2 = 0.77$, and were faster at T1 compared to T0, $F(1, 87) = 34.83$, $p < .001$, $\eta_p^2 = 0.29$. No main effect of group and no interaction with group were observed, indicating a comparable training effect in all groups. In addition, the response × time interaction was significant, $F(1, 87) = 9.18$, $p = .003$, $\eta_p^2 = 0.10$, reflecting a larger decrease in response times for correct trials. A training effect of time was also found for the PES, where the PES was smaller at the T1, $F(1, 87) = 26.55$, $p < .001$, $\eta_p^2 = 0.24$, independent of group, $F(2, 87) = 0.07$, $p = .930$, $\eta_p^2 = 0.00$, or the interaction of group × time, $F(2, 87) = 0.52$, $p = .599$, $\eta_p^2 = 0.01$.

4 | DISCUSSION

Previous studies suggested that the link between anxiety and neural correlates of performance monitoring may be driven by worry. However, most of these studies used cross-sectional research designs that preclude causal inferences. The present study aimed at disentangling the effects of trait and state worry on ERN and CRN in a mainly subclinical sample. To this end, we performed cross-sectional as well as longitudinal analyses in a randomized controlled trial. First, $n = 90$ university students completed a flanker task, after which they were randomly assigned to either a worry induction, a worry reduction, or a passive control group. Afterwards, they performed a second flanker task to assess potential alterations of performance related ERPs attributable to the worry interventions. Manipulation checks showed that compared to the control group, state worry increased in the induction group, but also in the reduction group. However, this marked increase in state worry in both groups had no effects on ERN or CRN amplitudes. Across all groups, CRN amplitudes decreased over time, possibly reflecting either training effects or task disengagement. Cross-sectional analyses of the baseline ERPs found larger ERN and CRN amplitudes in females. In addition, higher levels of trait worry were associated with larger CRN amplitudes (irrespective of gender) and larger ERN amplitudes in females only, whereas in males, higher levels of trait worry were associated with smaller ERN amplitudes. Participants with a current or lifetime diagnosis of internalizing disorders showed larger ERN amplitudes compared to participants without a lifetime diagnosis.

In terms of our preregistered hypotheses, we only found partial support for our first hypothesis proposing an association of trait worry with larger ERN amplitudes: Only females showed an increase of ERN with growing levels of trait worry. This finding is in line with meta-analytical evidence suggesting a gender-specific link which was attributed to larger inferences of subvocal rehearsal and gonodal hormones in females on task performance and cognitive control (Moser et al., 2016). Males, in comparison, showed the opposite direction of effects with smaller ERN amplitudes in participants with

increasing trait worry—an unexpected finding that has not been reported before and that warrants further investigation. Nonetheless, we want to emphasize that our sample consisted of only $n = 24$ male participants, limiting the generalizability of male-specific associations. Irrespective of gender, CRN amplitudes were larger in participants with larger trait worry, which might imply that the link to neural correlates of performance monitoring is not specific to error monitoring but translates to performance monitoring in general. This is in line with recent meta-analytical evidence suggesting a small relationship between anxiety and CRN amplitudes (Saunders & Inzlicht, 2020). Our results support this pattern and refine this relationship by pointing to a specific association of trait worry and the CRN. Contradicting previous findings (Fischer et al., 2016; Härpfer, Carsten, Spychalski, et al., 2020; Larson et al., 2011), females showed larger ERN and CRN amplitudes compared to males. As mentioned before, there were only few male participants in our sample limiting the generalizability of the present gender-related results. Future studies targeting research questions of gender-specific effects of performance associated ERPs should recruit more equally distributed samples with large and comparably sized gender groups including participants across the whole worry spectrum, to deliver more informative and reliable evidence for the suggested link. Altogether, our cross-sectional findings support the hypothesized relationship between neural correlates of performance monitoring and worry in females, and suggest that this relationship may not be error-specific.

Because we aimed at disentangling the possible effects of trait and state worry on performance monitoring, we experimentally manipulated state worry by targeted interventions. Because both interventions resulted in a significant increase in state worry, we can only make conclusions about the induction of state worry. No empirical support could be found for our second preregistered hypothesis that higher levels of state worry lead to an increased ERN. This null finding was backed by explorative Bayesian analyses that allow the quantification of evidence for the null hypothesis, yielding positive to strong evidence against an association of state worry and the ERN. In addition, we could also not find evidence for ERN variations due to the state worry interventions in an all-female subsample. Our second hypothesis was derived from the CEMH assuming that the link between error monitoring and worry is the product of compensatory effort of the brain to overcome processing inefficiency due to the workload that worries put on working memory (Moser et al., 2013). However, our results do not support that ERN amplitudes increase when state worry does, as the CEMH predicts.

It is important to note that there is an ongoing debate in the literature whether the ERN is a trait-like neural risk maker or whether it reflects symptom states. It is also discussed whether the ERN can be altered by interventions (Moser et al., 2013; Proudfit et al., 2013). On the one hand, an error-specific training (Meyer et al., 2020) as well as an attentional bias modification (Klawohn, Hajcak, et al., 2020; Nelson et al., 2015; but see Carlson et al., 2021) successfully reduced ERN amplitudes, suggesting that the ERN can be modulated and might thus be state related to a certain degree. On the other hand, cognitive behavioral therapy decreasing psychopathological symptoms has no effect on the ERN (Gorka et al., 2018; Hajcak et al., 2008; Kujawa et al., 2016; Ladouceur et al., 2018; Riesel et al., 2015), unaffected first-degree relatives and their affected family members show similar aberrant error monitoring (Carrasco et al., 2013; Riesel et al., 2011; Riesel, Klawohn, et al., 2019), and an enhanced ERN is predictive for the onset of anxiety disorders (Meyer et al., 2015). These studies further emphasize the stable, trait-like properties of the ERN but still show that state manipulations that alter error sensitivity also lead to ERN variations.

In light of these previous findings, together with the results of the current study showing ERN variation due to interindividual differences (i.e., trait worry, lifetime internalizing psychopathology) but not intraindividual differences in emotional state (i.e., state worry), we conclude that increased ERN amplitudes are not a consequence or a correlate of worry, but rather may reflect a more stable general disposition (i.e., trait-like) underlying increased anxiety and worry. This conclusion is in line with the assumption that the ERN likely represents a trait-like risk marker or endophenotype for anxiety (Olvet & Hajcak, 2008; Proudfit et al., 2013; Riesel, Klawohn, et al., 2019). However, the fact that the ERN does not seem to be related to state worry does not imply that the ERN per se is insensitive to other emotional states but that the trait-like properties might limit the range of state-associated variability of the ERN. Previous research has found positive affect (Bakic et al., 2014; Larson et al., 2006; Nigbur & Ullsperger, 2020; but see Larson et al., 2013) and negative affect (Pfabigan et al., 2013; Unger et al., 2012; Wiswede, Münte, Goschke, et al., 2009; Wiswede, Münte, & Rüsseler, 2009; but see Larson et al., 2006, 2013) to be associated with elevated ERN amplitudes when manipulating experimental conditions using affective pictures, negative feedback, or mathematical reasoning tasks. Our study design did not aim to directly manipulate positive or negative affect, but as the manipulations checks show, affect was also influenced by the interventions (but to a lesser extent than state worry). The fact that no alterations of the ERN and CRN were observed contradicts earlier findings, but could also be due to an insufficient potency of the intervention. At the same time, the non-specificity

of the effects also illustrates the large overlap between different anxiety-associated emotions.

The interpretation of the present results must be seen in light of certain limitations. Not only was there an unbalanced distribution of gender in our sample, we also recruited mainly participants with subclinical levels of worry. The link between ERN and anxiety was less reliably demonstrated in subclinical samples (Saunders & Inzlicht, 2020) and males (Moser et al., 2016) which might have been the reason why the power of the present study was not large enough to detect small sized effects. However, Bayesian analyses provided positive to strong evidence against an effect of state worry on the ERN.

In addition, we cannot make any statement on the effects of a worry reduction as our intervention to reduce worry levels did not work as intended but instead, increased state worry almost to the same magnitude as the worry induction did. Although there is meta-analytical evidence for emotional expressive writing to improve a broad spectrum of psychological and physiological outcomes (Frattaroli, 2006; Zachariae & O'Toole, 2015), its specific effect on reducing worries has gained less attention. However, there are primary studies showing that expressive writing significantly reduces worries and test anxiety (Goldman et al., 2007; Wolitzky-Taylor & Telch, 2010). Meta-analyses also suggest that effect sizes are larger with increasing duration and number of writing sessions (Reinhold et al., 2018; Travagin et al., 2015). In our study, participants received a very short eight-minute intervention that might have not allowed them to adapt to the exposed worry topics. Another reason might be that we did not pre-select a sample of chronic worriers like Schroder et al. (2018) did. Individuals without chronic worries might not need to reduce or offload their minds from the distracting effects of worries. Therefore, they cannot benefit from such an intervention. However, we have chosen this approach because of the advantage to directly compare effects of a worry induction and reduction within one study.

Regarding the worry induction paradigm, we successfully induced worry, but did not see alterations in the ERN or CRN. This might be due to an absence of state-related influences on these ERPs, but might also be due to an insufficiently potent intervention. In fact, it is still unclear, how potent a worry intervention would have to be in order to alter performance monitoring associated ERPs. A statistically significant increase of state worry must not be equaled with a (clinically) relevant increase. Further, our findings and conclusion are restricted to mainly subclinical populations. Individuals with clinically relevant psychopathologies would be interesting participants for future studies investigating the effects of worry interventions. The effects of the interventions were also not worry-specific but also altered state affect and state arousal. Another difference to previous studies (Ramirez & Beilock, 2011; Schroder et al., 2018) is that our interventions were not linked to the upcoming testing situation. As a result, the interventions targeted worries that were unrelated to the performance of the flanker task and unrelated worries might have played a subordinate role for participants' performance monitoring at that task. A previous study showed that error monitoring increases with the greater relevance of error commission, such that the ERN is larger when a punishment is related to errors but not when punishment is unrelated (Meyer & Gawlowska, 2017). This might apply to related and unrelated worries as well. Yet, a link to ideographic and task-unrelated worries with personal relevance would imply increased ecological validity of findings and facilitate clinical translation.

In summary, the present study investigated the effects of trait and state worry on performance monitoring associated ERPs (i.e., ERN and CRN) in a mainly subclinical sample. We could only partially replicate an association of trait worry with larger ERN amplitudes: Only females showed larger ERN amplitudes with increasing trait worry. Furthermore, in line with previous studies, we found larger ERN amplitudes in participants with a current or lifetime diagnosis for an internalizing disorder. Concerning the CRN, amplitudes were larger with increasing trait worry (irrespective of gender) but did not differ between participants with or without a lifetime diagnosis of internalizing disorders. Our worry interventions successfully manipulated levels of state worry, but only the worry induction showed effects into the intended direction, whereas the worry reduction also increased state worry. Nevertheless, neither ERN nor CRN amplitudes were altered due to any of the interventions that led to marked increases in state worry. In the face of the current findings, we find tentative support for an association between trait worry and ERN and CRN, with gender being an important moderator. At the same time, we find no evidence for a causal role of state worry influencing the ERN or CRN in our subclinical sample. Instead, consistent with previous findings and assumptions, we demonstrate the stability and independence of the ERN from state worry, as assumed for a trait-like neural risk marker or endophenotype, as which the ERN has been repeatedly conceptualized.

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AUTHOR CONTRIBUTIONS

Kai Härpfer: Conceptualization; data curation; formal analysis; investigation; methodology; project administration; resources; software; visualization; writing – original draft; writing – review and editing. **Hannes Per Carsten:** Conceptualization; investigation; methodology; project administration; validation; writing – review and editing. **Kim Löwisch:** Formal analysis; investigation; validation; visualization; writing – review and editing. **Nele Westermann:** Formal analysis; investigation; validation; visualization; writing – review and editing. **Anja Riesel:** Conceptualization; funding acquisition; methodology; project administration; supervision; writing – review and editing.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

FIGURE S1 Output with specifications of the a priori sample size calculation using G*Power, version 3.1.9.7

TABLE S1 Effects of gender, PSWQ, and Gender × PSWQ on the ERN and CRN using different scoring approaches

TABLE S2 Results of frequentist ANOVA for ERN and CRN varying as function of time and group using different scoring approaches

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