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No intolerance of errors: The effect of intolerance of uncertainty on performance monitoring revisited

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ABSTRACT

Errors have been conceptualized as internal forms of threat that can cause harm in unpredictable ways. An index of error processing is the error-related negativity (ERN), an event-related potential reflecting variability in the sensitivity to errors. Prior work has shown the relationship between psychopathology symptoms and the ERN is unclear, and may be moderated by intolerance of uncertainty (IU), a trait that captures how people react to unpredictability. IU includes two subfactors of prospective IU (active seeking of predictability) and inhibitory IU (behavioral paralysis). In the present study, 188 undergraduates performed an Eriksen flanker task designed to elicit the ERN, while brain activity was recorded using electroencephalography (EEG). Participants completed the Intolerance of Uncertainty Scale, Short Form (IUS-12), and other measures of anxiety, depression and worry. Total IU explained 5 % of the variance in correct-response negativity (CRN), but was not associated with the ERN in our sample. In contrast to previous findings, the IU subfactors did not predict the ERN or post-error slowing (PES), nor did total IU and depression interact to predict the ERN. Exploratory analyses also showed that total IU did not moderate the relationship between trait anxiety and the ERN. Small samples may have previously exaggerated the links between self-reported IU and the ERN. As such, further high-powered replications are required to confirm if, and how, they are related.

1. Introduction

1.1. Error processing and psychopathology

Errors are a vital source of information. Learning from our mistakes allows us to guide adaptive behaviors in response to a constantly changing environment. Error processing is part of a higher cognitive function called performance monitoring, which is the ability to scrutinize ongoing performance in order to detect, and therefore correct, mistakes (Dehaene et al., 1994). Despite the adaptive function of error processing, errors themselves have been conceptualized as forms of endogenous threat because their consequences can jeopardize wellbeing (Weinberg et al., 2016). Indeed, several defensive physiological responses observed following errors (e.g., increased startle reflex and pupil dilation, changes in the skin conductance response, increased amygdala activity (Hajcak and Foti, 2008; Hajcak et al., 2003; Pourtois et al., 2010) are similar to other exogenous types of threat, such as predators. However, unlike external threats, errors promote a uniquely interoceptive focus on one's behavior (Weinberg et al., 2016) and may be appraised more negatively by individuals at risk for internalizing psychopathology (Pasion and Barbosa, 2019). Moreover, errors are inherently unpredictable (Proudfit et al., 2013) which means they may be particularly aversive to people who cannot tolerate the unexpected or unknown, regardless of psychopathology status.

An electrophysiological index of error detection is the error-related negativity (ERN), a negative deflection in the event-related potential (ERP) that occurs 50–100 ms after a person makes a mistake in a reaction-time task (Gehring et al., 1993). The ERN represents early and automatic registration of errors (Hoffmann and Falkenstein, 2012) and functions as a signal for increased cognitive control and necessary behavioral adjustments (Weinberg et al., 2016). By contrast, more conscious and explicit processing of errors is indexed by the error

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positivity (Pe) which occurs 200–400 ms after error commission (Hewig et al., 2011). Enhanced ERNs have been observed in patients with obsessive-compulsive disorder (OCD; Endrass et al., 2008), generalized anxiety disorder (GAD; Weinberg et al., 2010) and social anxiety disorder (SAD; Endrass et al., 2008). While the ERN has been found to correlate with trait anxiety in non-clinical samples (Moser et al., 2013; Weinberg et al., 2015a), more recent meta-analytic work has found that the association between trait anxiety (as measured by the State-Trait Anxiety Inventory; Spielberger, 1983) and the ERN was smaller than previously assumed after correcting for publication bias (Saunders and Inzlicht, 2020), with no overall correlation found between anxiety and the ERN across 29 non-clinical volunteer samples.

The relation between depression and the ERN is unclear, with studies finding both enhanced (Weinberg et al., 2012) *and* diminished ERNs (Holmes and Pizzagalli, 2008). One recent meta-analysis found no overall relationship between depression and ERN amplitude (Moran et al., 2017). Another meta-analysis found a negligible unique contribution of depression to the association between internalizing spectrum disorders and increased ERN (Pasion and Barbosa, 2019). Indeed, depression has been found to obscure the relationship between GAD and the ERN, with the enhanced ERN found in those with a single GAD diagnosis not visible in comorbid subjects (i.e., GAD + depression; Weinberg et al., 2015b). In short, the direct relationship between depression and the ERN appears weak at best. Findings suggesting weak associations between the ERN and symptoms are apparent even when considering symptoms at the transdiagnostic level (i.e., anxious-depression; Seow et al., 2020). These relationships might be more adequately explained by considering a potential underlying mechanism: variability in how people react to the unpredictability of threats.

1.2. Intolerance of uncertainty

Intolerance of uncertainty (IU) is a dispositional tendency that reflects an individual's inability to endure ambiguous or uncertain events (Carleton, 2012, 2016; Freeston et al., 1994). Individuals with higher levels of IU then perceive and interpret these events as aversive and threatening (Dugas et al., 2005). IU is present in unselected samples, with higher levels associated with internalizing psychopathology, including GAD, SAD, OCD, panic disorder and depression (Mahoney and McEvoy, 2012). Moreover, IU is also associated with various factors that underlie such internalizing symptoms, such as greater rumination (Liao and Wei, 2011), elevated anxiety sensitivity (Carleton et al., 2007), increased checking behavior (Tolin et al., 2003), difficulties in safety learning (Morriss et al., 2021) and avoidance (Flores et al., 2018). As such, IU is considered a transdiagnostic risk and maintaining factor for internalizing disorders (McEvoy et al., 2019). Further, IU and worry are also highly related, independent of shared variance with anxiety or depression symptoms (Dugas et al., 1997; Freeston et al., 1994). Two subfactors of IU are thought to capture different ways people interpret and respond to uncertainty. Prospective IU (P-IU) reflects a desire for predictability and active seeking of certainty, while inhibitory IU (I-IU) points towards avoidance, behavioral inhibition, and paralysis in the face of uncertainty (Carleton, 2012).

1.3. Relationships between ERN, IU and psychopathology

Studies examining the relationship between IU and the ERN are limited and pose some contradictions. Two studies did not find a direct association between the total score of IU and the ERN (Jackson et al., 2016; Ruchensky et al., 2020). Concerning the IU subfactors, Jackson et al. (2016) observed that both subfactors had opposing associations with the ERN during a flanker task, which requires participants to rapidly discriminate a single centered element (e.g., letter) in a string (Eriksen and Eriksen, 1974). ERN amplitude was enhanced for prospective IU but attenuated for inhibitory IU. The enhanced ERN in P-IU might have indicated an increase in performance monitoring in response

to an error, however, this did not translate into better overall task accuracy for this group (with P-IU and I-IU performing comparably). Relatedly, Jackson et al. (2016) also found behavioral associations with IU subfactors; P-IU was associated with slower post-correct reaction time (RT) and higher I-IU with faster RT for post-corrects and post-errors. Post-error slowing (PES) is the behavioral adaptation characterized by increased RTs on trials that follow error commission compared to correct ones (Schroder et al., 2020), and an indication that processes responsible for cognitive control have responded to the ERN (Weinberg et al., 2016).

These findings of differential ERN amplitude for IU subfactors in Jackson et al. (2016) were not replicated by Sandre and Weinberg (2019) who found no association between the ERN and either subscale during a flanker task. However, Sandre and Weinberg (2019) employed a modified version of the flanker task that also included ambiguous trials (i.e., where they explicitly instructed participants there would be no right or wrong responses). Inhibitory IU was associated with a smaller negative deflection in response to these trials. If this response to ambiguous trials still represents a response to the *possibility* of having made an error, Sandre and Weinberg's evidence of blunted performance monitoring for I-IU under conditions of uncertainty is not completely at odds with the I-IU-ERN attenuation found by Jackson et al. (2016). Still, the exact relationship between the IU subtypes and the ERN remains inconclusive. In the only study examining IU, ERN and psychopathology symptoms, Ruchensky et al. (2020) found that IU moderated the relationship between depression and the ERN. Specifically, depression symptoms were associated with a larger ERN, but only when IU was *low* (with no depression-ERN relationship at high levels of IU). Of note, Ruchensky et al. (2020) found no significant results when examining the subscales of IU. The authors contend that one reason previous research may have failed to find an association between depression and the ERN (e.g., Moran et al., 2017) was by not considering other underlying confounding variables, such as IU.

1.4. The present study

This study aimed to investigate various relationships between intolerance of uncertainty, self-reported symptoms of anxiety/depression, and brain activity associated with error processing. We sought to extend the small body of literature focused on ERN and IU, where evidence has thus far been inconclusive (Jackson et al., 2016; Ruchensky et al., 2020; Sandre and Weinberg, 2019). Importantly, our replication used a large sample of undergraduates ($N = 188$) to ensure high statistical power in order to avoid the presence of chance findings that can occur with smaller samples. High-powered replications are especially needed considering the context of the replication crisis in ERP research (Clayson et al., 2019), whereby low samples can lead to an overestimation of the associations between electrophysiological and self-report variables ("false-positives"; Bernoster et al., 2019; Larson and Carbine, 2017). Our well-powered sample aims to disentangle discrepancies in the literature surrounding our constructs of interest. Specifically, mixed findings regarding the effect of IU subfactors on ERN amplitudes (Jackson et al., 2016; Sandre and Weinberg, 2019), mixed findings for direct relationships between depression error monitoring (Moran et al., 2017), and prior potential overestimation of the relationship between ERN and trait anxiety (Saunders and Inzlicht, 2020). We have attempted an *experimental replication* of Jackson et al. (2016) and Ruchensky et al. (2020), which seeks to verify previous findings using particular variables while adjusting the experimental procedure (Hudson, 2021). By these adjustments it differs from a direct or exact replication, which aims to repeat the identical procedure of its reference. An experimental replication, on the other hand, seeks increased generalizability of findings that were obtained by a specific design. We employed a version of the flanker task that has been previously used with other populations (Andreu et al., 2017; Bernoster et al., 2019; Franken et al., 2007, 2010, 2018; Zijlmans et al., 2021). While it differs

from our replication references (Jackson et al., 2016; Ruchensky et al., 2020) in several features (i.e., the duration of stimulus presentation, type of stimulus, out-of-time feedback inclusion, number of trials and blocks), this modified version does evoke the equivalent behavioral and neural effects related to performance monitoring (Franken et al., 2007, 2010).

The first objective of this study was to examine the relationship between the IU subfactors, ERN amplitudes and post-error slowing. Contradictions exist between Jackson et al. (2016) who found an enhanced ERN for P-IU compared to I-IU, and Sandre and Weinberg (2019), who found no effect. It should be noted that Sandre and Weinberg (2019) *did* find an attenuated ERN for ambiguous (feedback not given) trials, which we have argued could overlap with error trials. Therefore, we expected to replicate the results of Jackson et al. (2016) whereby P-IU would be associated with an enhanced ERN while I-IU would be associated with an attenuated ERN (Hypothesis 1). However, both Ruchensky et al. (2020) and Jackson et al. (2016) found no relationship between ERN and total IU, as such, we also tested if this possible relationship exists in a larger sample (Hypothesis 2). Similar to Jackson et al. (2016) we expected to find an effect of IU subfactor on post-error RT: we expected slower RT in higher P-IU after error trials and faster RT for increased I-IU after error trials. Additionally, we calculated post-error slowing (PES) as difference scores by subtracting average reaction time after correct responses from average reaction time after error responses (Schroder et al., 2020) of which we tested a possible association with IU scores (Ruchensky et al., 2020; Hypothesis 3). The second aim of this study was to perform a replication of the moderation analysis of Ruchensky et al. (2020). As such, we hypothesized that depression and IU would interact to predict the ΔERN (i.e., difference in amplitudes between error trials and correct trials), such that higher levels of depression would be associated with larger ΔERNs at low (but not medium or high) levels of IU.

2. Materials and methods

The hypotheses and methods of this study were pre-registered (https://osf.io/c5yr3). The final sample data and analyses (R script) are publicly available (https://osf.io/4mtn3/).

2.1. Participants

In total, 212 psychology undergraduate students from Erasmus University Rotterdam were invited to the lab session via an online recruitment system. After the data-related (EEG and behavioral responses) exclusion criteria (see below) were applied, a final sample of 188 participants was used for all analyses (age range from 17 to 46 years, $M = 21.23$, $SD = 3.31$, 81 % female, 55 % Dutch). A breakdown of their ethnic self-identification was 74 % for White/Caucasian, 9 % for Asian, 4 % for Middle Eastern, 4 % for Black, 3 % for mixed, 2 % for Latin-American, and around 5 % for Other and non-declared. Participants received course credit and monetary compensation (€5) related to a different task in the same session (see Procedure). A priori exclusion criteria were family history of photic epilepsy, related to another task that used flickering images. Informed consent was obtained before participation, and the research protocol was approved by the ethical board of the Erasmus School of Behavioural and Social Sciences.

2.2. Questionnaires

After finishing the experimental protocol, participants completed the following self-report questionnaires (either in Dutch or English, for Dutch or international students, respectively):

The Intolerance of Uncertainty, Short Form (IUS-12; Carleton et al., 2007) is a shortened version of the original 27-item Intolerance of Uncertainty Scale (Freeston et al., 1994; Dutch translation: de Bruin et al., 2006). It assesses the degree to which individuals find uncertain

scenarios distressing and undesirable, as well as their reactions to them. The items are rated on a five-point Likert scale ranging from 1 (not at all characteristic of me) to 5 (entirely characteristic of me). The scale ranges from 12 to 60, with higher scores indicating higher intolerance of uncertainty. In addition to the total score, two subfactors can be extracted, Prospective IU (7 items) and Inhibitory IU (5 items; McEvoy and Mahoney, 2011). Internal consistency was acceptable – good for the total scale IU ($\alpha = 0.87$) and both subfactors P-IU ($\alpha = 0.81$) and I-IU ($\alpha = 0.77$). Total IU scores in the current sample ranged from 12 to 51 ($M = 29.36$; $SD = 8.05$). Prospective IU ranged from 7 to 31 ($M = 18.21$, $SD = 5.21$) while Inhibitory IU ranged from 5 to 20 ($M = 5.81$, $SD = 7.68$). The distribution of IU total scores was found to be approximately normal (see Supplemental material for density plot). The response patterns/densities in this sample are comparable to community and undergraduate samples reported by Carleton et al. (2012) while being lower than clinical samples, as expected.

The Beck Depression Inventory, Second Edition (BDI-II; Beck et al., 1996; Dutch translation: Does, 2002) is a widely used self-report measure of depressive symptoms, both cognitive and somatic. It consists of 21 items where participants rate items on a four-point Likert scale ranging from 0 to 3 based on perceived severity in the last two weeks. The score ranges from 0 to 63, with higher scores indicating more severe depression symptoms. Internal consistency of this scale was good ($\alpha = 0.94$). Scores in the current sample ranged from 0 to 34 ($M = 5.81$, $SD = 7.68$). The current sample showed less severity of depression scoring on the BDI-II than that of Ruchensky et al. (2020; $M = 11.68$; $SD = 8.51$) who reported 67 % of the sample in the "no depression" range (0–13; compared to our 85 %), 17 % in the "mild depression" range (14–19; compared to our 7 %) and 16 % in the moderate to severe depression range (20–63; compared to our 8 %). Normative data for the BDI-II in undergraduates from the USA ($M = 9.14$, $SD = 8.45$; Whisman and Richardson, 2015) and community samples from The Netherlands ($M = 10.6$, $SD = 10.9$; Roelofs et al., 2013) further illustrates that our sample's mean and standard deviation are lower than that of comparable samples.

The Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990; Dutch translation: Van Rijsoort et al., 1999) is a 16-item self-report questionnaire that measures the trait of worry across the dimensions of excessiveness, generality and uncontrollability. Each item is rated on a five-point Likert scale from 1 (not at all typical of me) to 5 (very typical of me). The scale ranges from 16 to 80 with higher scores indicative of higher levels of trait worry. Internal consistency of this scale was good ($\alpha = 0.83$). Scores in the current sample ranged from 25 to 72 ($M = 44.97$; $SD = 10.82$).

The trait subscale of the State Trait Anxiety Inventory, Form Y (STAI-T; Spielberger, 1983; Dutch translation: Ploeg, 2000) is a self-report questionnaire consisting of 20 items that measure someone's general propensity for anxiety. Respondents rate items based on how they generally feel, using a four-point Likert scale from 1 (almost never) to 4 (almost always), with higher scores indicating greater anxiety (range: 20 to 80). Internal consistency of this scale was acceptable ($\alpha = 0.79$). Scores in the current sample ranged from 24 to 66 ($M = 45.33$; $SD = 8.00$).

2.3. Task and materials

Participants completed an adapted version of the Eriksen flanker task (Franken et al., 2010). The task was programmed in E-prime 3 software (Psychology Software Tools, Pittsburgh, PA). There were four types of a letter string: two congruent (HHHHH and SSSSS) and two incongruent (SSHSS and HHSHH). The target letters array was displayed in white against a black background on a 20-inch PC monitor with a resolution of 1024 × 768 pixels which was placed at a distance of 120 cm from the seated participant. Participants were instructed to answer by pressing either '1' or '5' on a QWERTY keyboard, as fast and accurately as possible, if the middle letter was either H or S. Those number buttons

were associated with H and S in the instructions and the association was counterbalanced among participants.

A schematic depicting the progression of each trial can be seen in Fig. 1. Each trial started with a fixation cross (+) visible for 150 ms, followed by a blank screen of random duration between 100 and 150 ms. The letters were displayed for 50 ms. Participants had 700 ms to respond using both hands. After each trial, participants received feedback for 500 ms in red font (OOO for correct; XXX for incorrect; ! for no answer). The inter-trial interval (ITI) had a duration of 500 ms. Participants completed a practice block of eight trials and the actual task consisted of five blocks of 80 trials with self-administered breaks in between (400 total trials). Participant response type (correct or incorrect) and reaction time (in ms) on every trial was recorded for later analysis.

2.4. Procedure

After arrival at the lab, participants received a verbal description of the experimental procedure from the researcher in charge and provided written informed consent. After this, they were sat in an isolated dimly-lit room where the EEG was attached. The estimated duration of the whole experimental procedure was as follows: first, they performed a Doors task (40 min) followed by a long break (10 min), secondly, there was a Fear Conditioning task (25 min), both tasks not reported here. After a last break (10 min), they performed the Flanker task (15 min). Duration lengths are estimated and vary according to self-administered breaks during the tasks. During the long breaks the researcher checked the overall status of the participant.

2.5. EEG recording and processing

Continuous EEG was recorded using a 32 + 2 electrodes cap arranged according to the 10/20 layout. Electro-oculogram (EOG) was recorded using 4 external electrodes: two above and below the left eye, and another two at the outer canthus of each eye. All electrodes were sintered Ag/AgCl electrodes. Data were recorded using the Active-Two BioSemi system (BioSemi, Amsterdam, Netherlands). This system includes two additional electrodes, CMS (Common Mode Sense) and DRL (Driven Right Leg), that serve as online reference and ground. The EEG data were digitized at a 512 Hz sampling rate and 24-bit A/D conversion. All electrode impedances were kept below 20 kΩ.

Offline EEG data were analyzed with Brain Vision Analyzer 2 (Brain Products, Gilching, Germany). Data were re-referenced to the average voltage at all scalp electrodes (i.e., average reference), band-pass filtered from 0.1 to 35 Hz (Butterworth zero phase filter with a 24 dB/octave roll-off), notch-filtered at 50 Hz, and corrected for eye movement artifacts (Gratton et al., 1983). Data were segmented in epochs of 1200 ms (400 ms before and 800 ms after response). Artifact rejection was applied to all epochs using: (a) an amplitude criterion of ± 75 μV, (b) a gradient criterion of ± 50 μV/ms, (c) a difference criterion of 200 μV and (d) a low activity criterion of 2 μV (interval length: 200 ms). Overall, the percentage of removed epochs by component was for the ERN 4.76 %, for the CRN 4.04 %, for the Pe 4.56 %, and for the Pc, 4.25 %. Baseline correction was applied using the interval from 400 to 200 ms pre-response (Weinberg et al., 2010). From the 212 participants, two participant's questionnaire data was not saved and three participants were removed due to poor EEG data quality. Further, exclusion criteria based on the behavioral performance were: (a) participants who missed >20 % of trials of the flanker task ($n = 3$), or (b) participants who scored <55 % accuracy ($\underline{n} = 10$; Seow et al., 2020). In addition, (c) participants with <10 error commissions were also excluded ($n = 6$; Olvet and Hajcak, 2009). Finally, (d) trials with reaction times below 200 ms were not further considered in the analysis (Sandre et al., 2020; Weinberg et al., 2015b). Altogether, 188 full datasets remained for further analysis after applying these criteria. The average number of error and correct trials in our sample was 50.01 ($SD = 25.29$), and 328.45 ($SD = 37.25$), respectively. Internal consistency of the ERPs and behavioral measures was calculated using odd-even split-half reliability with Spearman-Brown Correction (using SPSS v. 28). The reliability analysis of the ERPs showed good to excellent reliability (ERN: 0.92, CRN: 0.98, Pe: 0.85, Pc: 0.97), and the reliability analysis of the behavioral measures showed excellent reliability for accuracy (0.95) and for reaction times (0.98).

2.6. Power calculations

Power calculations for linear multiple regression were performed using G*Power3 (Faul et al., 2007). For analyses predicting the ERN amplitude from IU subfactors, we entered a minimum effect size of $f^2 = 0.11$ (corresponding to the R^2 of 0.10 found by Jackson et al., 2016), an alpha level of 0.05 and two predictors. A-priori tests showed that a sample size of $N = 90$ was required to achieve a power of 0.80. For our moderation analysis, we entered a minimum effect size of $f^2 = 0.09$ (corresponding to the R^2 of 0.08 found by Ruchensky et al., 2020), an
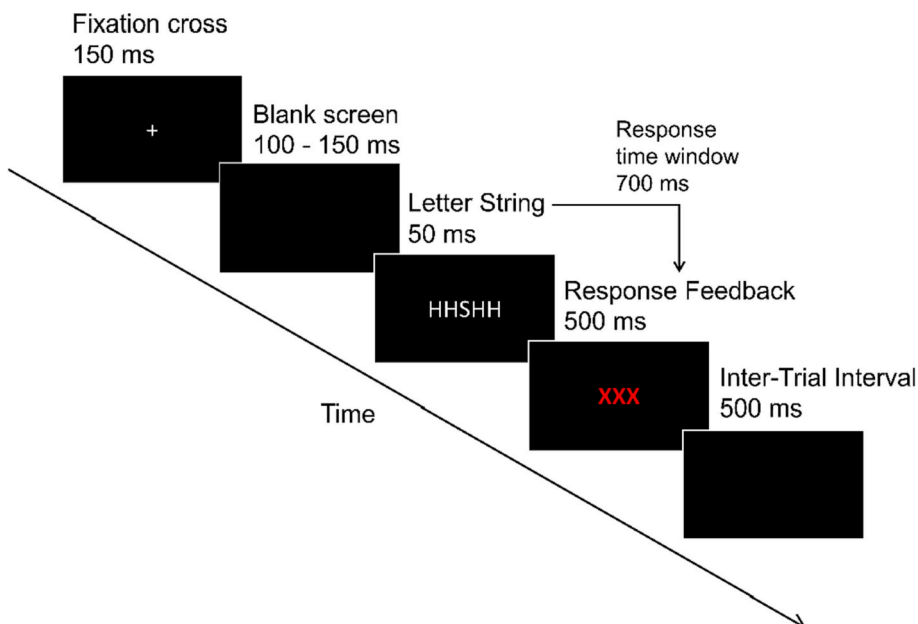
Fig. 1. Schematic of a flanker task trial. A single trial lasted between 1600 and 1650 ms (accounting for the variable duration of the blank screen). A fixation cross was presented for 150 ms, followed by a blank screen for 100–150 ms. The letter string was presented for 50 ms, then the screen remained blank for a further 650 ms (giving participants a 700 ms time frame to indicate their response). Feedback was then shown for 150 ms (XXX for incorrect, OOO for correct, ! for "too late") and the trial ended with a blank screen of 500 ms.

alpha level of 0.05 and two predictors. A-priori tests showed that a sample size of $N = 114$ was required to achieve a power of 0.80. After applying the exclusion criteria, the sample size of this study ($N = 188$) had a power of 0.96 to detect an effect size of $f^2 = 0.09$, and a power of 0.99 to detect an effect size of $f^2 = 0.11$.

2.7. Data analysis

Average ERP waves were calculated for artifact-free trials at each electrode of interest in two conditions (correct and incorrect responses). The ERN and CRN were defined as the mean activity on error and correct trials, respectively, at 0 to 100 ms after the participant's response at Cz electrode, where the signal was maximal (Sandre et al., 2020), see Fig. 2a. The Pe and Pc were defined as the mean activity on error and correct trials, respectively, at 200 to 400 ms after response at both Cz and Pz electrodes (Ruchensky et al., 2020), see Fig. 2b. We also calculated difference potentials to account for individual variation between correct and error trials. This is referred to as ΔERN and ΔPe, calculated as the difference between the respective error potential amplitude and correct potential amplitude (e.g., ERN - CRN).

The behavioral measures extracted from the flanker task were: (1) number of correct trials (accuracy); (2) RT of the correct and incorrect trials; (3) average RTs of the next trial after error and correct feedback (i. e., post-correct and post-error RTs); (4) post-error slowing (PES), as the difference between average RT after error trials and the average RT after correct trials.

To examine the effects of the IU subfactors on performance monitoring, we ran a multiple regression using both IU subfactors (P-IU and I-IU) as simultaneous predictors and the ERN as the dependent variable. Next, we ran a simple linear regression with total IU as predictor as the ERN as the dependent variable. Further exploratory regression analyses were run for several dependent variables (ERN, Pe and RTs) using both total IU (single predictor) and the IU subfactors (simultaneous predictors). To assess the effect that depression and trait anxiety has on the relationship between IU and performance monitoring, we conducted a moderation analysis. Depression and trait anxiety levels were based on the participants' scores in the BDI-II and the trait scale of the STAI, respectively. For this, we conducted a multiple regression with total IU and the interaction term (depression × total IU) entered simultaneously to predict the ΔERN, based on Ruchensky et al. (2020). Predictors were mean centered. Similar exploratory analyses were performed substituting (a) trait anxiety scores in the interaction term and (b) ΔPe as the outcome variable.

All analyses were performed using R Statistical Software (v4.1.2; R Core Team, 2021). The assumptions for multiple linear regression were tested prior to running each confirmatory analysis and can be viewed in the Supplemental material. Our outlier treatment was consistent with the replication references: for the regression analyses we removed univariate outliers from the ERN data (Jackson et al., 2016), while for the moderation analysis neither univariate nor multivariate outliers were removed (Ruchensky et al., 2020). Correlations, *t*-tests, and regressions were performed using the 'stats' package (R Core Team, 2021). The figures were generated using the 'jtools' (Long, 2020) and 'ggplot2' (Wickham, 2016) packages, and a tutorial by Craddock (2016).

3. Results

Table 1 shows means, standard deviations, and correlations for variables of interest which ranged from small (Age and CRN, $r = 0.14$, $p < .05$) to large (P-IU and I-IU, $r = 0.63$, $p < .01$), with the IU subfactors having very large correlations with the total score IU (P-IU and IUS-12, $r = 0.93$, $p < .01$; I-IU and IUS-12, $r = 0.86$, $p < .01$). Both measures of the ERN were uncorrelated with any questionnaire variables. In contrast, the CRN amplitude correlated with total IU and both subfactors (all p values $< .01$), as well as with STAI-T ($p < .05$). Regarding the behavioral measures, both correlated with Pe amplitude (i.e., Correct %: $r = 0.30$, p

$< .01$; PES; $r = 0.17$, $p = .021$), however, PES did not correlate with ΔPe ($r = 0.11$, $p = .128$). The accuracy rate was also correlated with the amplitudes of ΔERN ($r = -0.17$, $p = .019$) and CRN ($r = 0.18$, $p = .014$). Further, both behavioral measures correlated negatively with trait anxiety scores (Correct %: $r = -0.24$, $p < .01$; PES; $r = -0.17$, $p = .019$), while PES was in addition negatively associated with I-IU scores ($r = -0.15$, $p = .038$). Interestingly, accuracy and PES were correlated with each other such that a higher accuracy rate was associated with longer PES ($r = 0.34$, $p \leq 0.01$).[2]

3.1. Behavioral results

Table 2 displays selected behavioral data for the flanker task. Paired-sample *t*-tests revealed behavioral patterns typical for the task. Mean error counts were larger for incongruent trials ($M = 35.32$, $SD = 16.19$), relative to congruent trials ($M = 14.69$, $SD = 10.83$; $t_{187} = -25.86$, 95 % confidence interval (CI) [-22.21, -19.01], $p < .001$, $d = 1.88$). Participants were faster on congruent trials ($M = 445.78$ ms, $SD = 26.90$ ms) versus incongruent trials ($M = 478.73$ ms, $SD = 29.70$ ms; $t_{187} = -31.63$, 95 % CI [-35.00, -30.89], $p < .001$, $d = 2.31$). Participants were also faster on error ($M = 418.52$ ms, $SD = 38.73$ ms) compared to correct trials ($M = 468.95$ ms, $SD = 26.22$ ms; $t_{187} = -25.48$, 95 % CI [-54.33, -46.52], $p < .001$, $d = 1.86$). Finally, post-error mean RTs ($M = 480.47$ ms, $SD = 40.32$ ms) were slower than post-correct mean RTs ($M = 459.83$ ms, $SD = 27.56$ ms; $t_{187} = -9.43$, 95 % CI [-24.96, -16.32], $p < .001$, $d = 0.70$).

3.2. ERPs

Grand average ERP waveforms at electrode Cz are presented in Fig. 3a for the ERN and CRN. Across the sample, the ERN exhibited a mean amplitude of 0.28 μV ($SD = 3.69$ μV) while the CRN had a mean amplitude of 3.55 μV ($SD = 2.56$ μV). A paired sample *t*-test revealed that the ERN was larger (i.e., more negative) than the CRN within the 0–100 ms window ($t_{187} = 14.33$, 95 % CI [2.82, 3.72], $p < .001$, $d = 1.04$).

Grand average ERP waveforms at electrode Cz are presented in Fig. 3b for the Pe and Pc. Across the sample, the Pe exhibited a mean amplitude of 4.00 μV ($SD = 2.57$ μV) while the Pc had a mean amplitude of 0.77 μV ($SD = 1.87$ μV). A paired sample *t*-test revealed that the Pc was larger (i.e., more negative) than the Pe within the 200–400 ms window ($t_{187} = -17.01$, 95 % CI [-3.60, -2.85], $p < .001$, $d = 1.24$).

3.3. Regression analyses

In line with Jackson et al. (2016), the ERN data was screened for univariate outliers using $\pm 1.5 \times$ interquartile range, leading to the removal of eight cases ($n = 180$). A multiple linear regression was performed to predict ERN amplitude via total IU. The results of the regression indicated that total IU was unable to explain any variance in the ERN, ($F_{1, 178} = 3.02$, $p = .084$, $R^2 = 0.02$), nor could total IU predict ERN amplitude ($\beta = -0.05$, $SE = 0.03$, $t = -1.73$, $p = .084$). A linear regression was then performed to predict ERN amplitude using P-IU and I-IU as simultaneous predictors. The two predictors were unable to explain any variance in the ERN, $F_{2, 177} = 1.85$, $p = .160$, $R^2 = 0.02$. Both P-IU ($\beta = -0.08$, $SE = 0.05$, $t = -1.59$, $p = .115$) and I-IU ($\beta = 0.01$, $SE =$

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[2] Because total IU, I-IU and STAI-T intercorrelated with several of our later outcome variables (CRN and PES), we assessed the specificity of IU-related measures above the STAI-T with several partial correlations. The correlation between total IU and CRN remained significant after controlling for STAI-T ($r = -0.19$, $p = .011$). This speaks to the specificity of IU in predicting the CRN, above-and-beyond trait anxiety. However, the significant observed correlation of PES and I-IU did not persist after controlling for STAI-T ($r = -0.09$, $p = .227$).

A ΔERN
0 ms - 100 ms

-3.24 μV   0 μV   3.24 μV

B ΔPe
200 ms - 400 ms

-5.05 μV   0 μV   5.05 μV
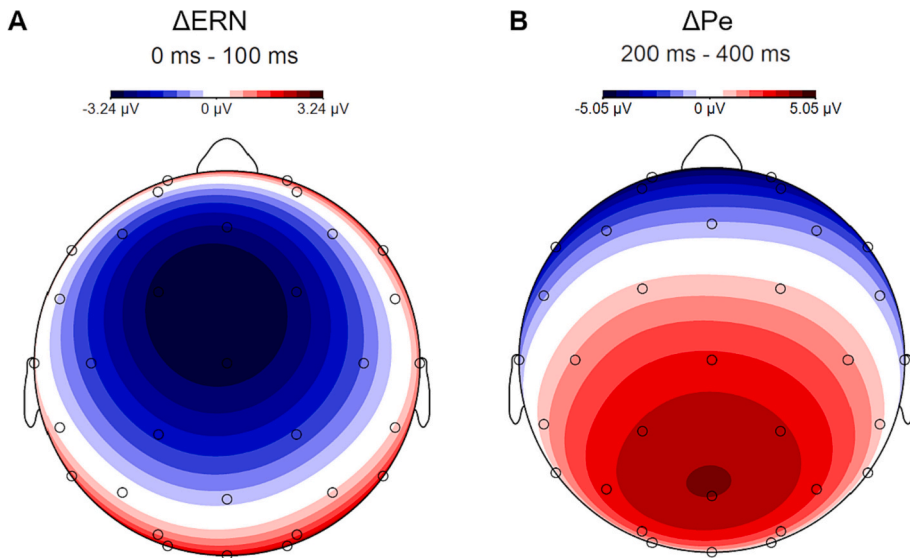
Fig. 2. Topographic map depicting the average difference (μV) between error and correct responses. Scalp maps display the grand average voltage distribution of (A) ΔERN from 0 to 100 ms post-response, and (B) ΔPe from 200 to 400 ms post-response. The voltage distributions are plotted on a minimum-maximum scale going from blue (negative polarity) to red (positive polarity). Compared to other electrodes, the most pronounced negative polarities were seen at Cz for the ΔERN and Pz/Cz for the ΔPe, during their respective time windows. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

0.08, $t = 0.17$, $p = .867$) were unable to predict ERN amplitude.

A multiple linear regression was performed to predict post-error RT using P-IU and I-IU as simultaneous predictors. The results of the regression indicated that two predictors were unable to explain any variance in post-error RT, $F_{2,\ 177} = 0.32$, $p = .728$, $R^2 < 0.01$. Both P-IU ($\beta = -0.29$, $SE = 0.75$, $t = -0.39$ $p = .699$) and I-IU ($\beta = -0.31$, $SE = 1.06$, $t = -0.29$, $p = .772$) were unable to predict post-error RT. Using PES (i.e., mean RT after error minus mean RT after correct) as the dependent variable in the same regression model, the subfactors could not predict any variance in post-error slowing, $F_{2,\ 177} = 2.22$, $p = .112$, $R^2 = 0.02$. I-IU was able to predict post-error slowing ($\beta = -1.56$, $SE = 0.79$ $t = -1.98$, $p = .049$) while P-IU was not ($\beta = 0.39$, $SE = 0.56$, $t = 0.70$, $p = .483$). However, this significant effect of I-IU on post-error RT should be interpreted with caution due to the non-significance of the overall model.

3.4. Moderation analyses

Table 3 presents results for regression analyses predicting the ΔERN. There were no significant results when entering depression and IU into separate regressions to predict the ΔERN. Likewise, there were also no significant results when depression and IU were entered simultaneously to predict the ΔERN. Finally, a model including depression, IU and the interaction term (depression × IU) did not explain any variance in the ΔERN ($F_{3,\ 184} = 1.33$, $p = .265$, $R^2 = 0.02$); the predictors and their interaction were also not statistically significant (see Table 3 and Fig. 4).

There were no significant results when entering trait anxiety and IU into separate regressions to predict the ΔERN (see Table 3). Likewise, there were also no significant results when trait anxiety and IU were entered simultaneously to predict the ΔERN. Finally, a model including trait anxiety, IU and the interaction term (trait anxiety × IU) did not explain any variance in the ΔERN ($F_{3,184} = 1.03$, $p = .380$, $R^2 = 0.02$); the predictors and their interaction were also not statistically significant (see Table 3 and Fig. 5).

There were no significant results when entering worry and IU into separate regressions to predict the ΔERN (see Table 3). Likewise, there were also no significant results when worry and IU were entered simultaneously to predict the ΔERN. Finally, a model including worry, IU and the interaction term (worry × IU) did not explain any variance in the ΔERN ($F_{3,184} = 1.40$, $p = .244$, $R^2 = 0.02$); the predictors and their interaction were also not statistically significant (see Table 3).

3.5. Exploratory regression analyses

Several simple and multiple regressions were run using total IU and the IU subfactors as predictors (see Table 4). Total IU explained 5 % of the variance in correct-response negativity, $F_{1,186} = -10.25$, $p = .002$, $R^2 = 0.05$. Neither total IU nor the IU subfactors could predict any variance in the ΔERN, Pe, Pc or ΔPe.

4. Discussion

Previous research has been inconclusive regarding the relationships between neural indices of performance monitoring, intolerance of uncertainty (including IU subfactors) and self-reported symptoms of anxiety and depression in non-clinical samples (Jackson et al., 2016; Ruchensky et al., 2020; Sandre and Weinberg, 2019; Saunders and Inzlicht, 2020). As such, we used a large sample with high statistical power to further clarify these relationships. As expected, total IU was not associated with the error-related negativity in our sample. However, contrary to our expectations, the IU subfactors did not predict the ERN or post-error slowing (PES), nor did IU and depression interact to predict the ERN. Exploratory analyses also showed that IU did not moderate the relationship between trait anxiety and the ERN; further, IU was not associated with the error positivity (Pe). Overall, our sample shows no evidence for the relationship between intolerance of uncertainty and the registration of errors at the implicit or conscious level (i.e., ERN and Pe, respectively; Hewig et al., 2011). Nevertheless, our findings should not be misinterpreted as conclusive proof of "no effect", but rather considered as a contribution to a small existing literature. Further high-powered replications as well as alternative statistical approaches (e.g., equivalence testing, Bayesian approaches; Harms and Lakens, 2018) are needed to determine whether the effect of IU on the ERN is meaningful. Moreover, the precise relationship between IU and general performance monitoring may be more nuanced than we have accounted for presently.

Our observation that total IU and the ERN were not associated is in line with Jackson et al. (2016) and Ruchensky et al. (2020). Regarding the additional explanatory value of the IU subfactors compared to the total measure, Jackson et al. (2016) had previously found that the IU subfactors were able to predict the ERN in different directions (i.e., enhanced ERN for P-IU; attenuated ERN for I-IU). However, we were not able to replicate this; our null finding instead aligns with two other studies showing no association between the IU subfactors and the error-related negativity (Ruchensky et al., 2020; Sandre and Weinberg, 2019). A priori power calculations recommended a sample of $N = 90$, meaning

Table 1
Means, standard deviations, and correlations of study variables.

Column label	M	SD	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. ΔERN (μV)	-3.17	3.12	1	.	.	.	.	.	.	.	.	.	.	.	.	.
2. ΔPe (μV)	3.44	2.39	0.23**	1	.	.	.	.	.	.	.	.	.	.	.	.
3. ERN (μV)	0.32	3.57	0.73**	0.27**	1	.	.	.	.	.	.	.	.	.	.	.
4. CRN (μV)	3.49	2.48	-0.17*	0.11	0.55**	1	.	.	.	.	.	.	.	.	.	.
5. Pe (μV)	4.12	2.5	0.03	0.74**	0.33**	0.44**	1	.	.	.	.	.	.	.	.	.
6. Pc (μV)	0.68	1.74	-0.28**	-0.38**	0.07	0.44**	0.34**	1	.	.	.	.	.	.	.	.
7. IUS-12	29.38	7.93	0.10	-0.09	-0.11	-0.23**	-0.11	-0.03	1	.	.	.	.	.	.	.
8. P-IU	18.23	5.14	0.05	-0.09	-0.01	-0.22**	-0.09	-0.00	0.93**	1	.	.	.	.	.	.
9. I-IU	11.15	3.65	0.14	-0.06	-0.03	-0.19*	-0.10	-0.06	0.86**	0.63**	1	.	.	.	.	.
10. STAI-T	45.35	7.91	0.09	-0.11	-0.03	-0.16*	-0.19*	-0.10	0.36**	0.26**	0.42**	1	.	.	.	.
11. BDI-II	5.76	7.71	0.08	-0.04	-0.04	-0.14	-0.10	-0.08	0.28**	0.19*	0.34**	0.40**	1	.	.	.
12. PSWQ	45.11	10.81	-0.04	-0.06	-0.12	-0.13	-0.13	-0.09	0.49**	0.39**	0.52**	0.54**	0.61**	1	.	.
13. Correct %	82.19	9.34	-0.17*	0.29**	-0.02	0.18*	0.30**	0.01	-0.10	-0.08	-0.11	-0.24**	-0.06	-0.06	1	.
14. PES (ms)	20.38	30.28	0.01	0.11	-0.02	-0.05	0.17*	0.08	-0.10	-0.05	-0.15*	-0.17*	-0.07	-0.10	0.34**	1
15. Age	21.21	3.32	-0.19**	-0.24**	-0.06	0.14*	-0.12	0.17*	-0.12	-0.08	-0.16*	-0.08	-0.19*	-0.12	0.18*	-0.04

Note. $N = 188$. ERN = Error related negativity; Pe = Error positivity; Pc = Correct positivity; CRN = Correct response negativity; P-IU = Prospective IU; I-IU = Inhibitory IU; STAI-T = Trait subscale of the State-Trait Anxiety Inventory; BDI-II = Beck Depression Inventory; IUS-12 = Intolerance of Uncertainty, Short Form; PSWQ = Penn State Worry Questionnaire; Correct % = percentage of correct responses; PES = PES traditional [Mean Post-Error RT – Mean Post-Correct RT]. Pearson product-moment correlation coefficients are given.

* Indicates $p < .05$.

** Indicates $p < .01$.

Table 2
Descriptive statistics of flanker task behavioral data.

	M	SD	Minimum	Maximum
Accuracy (n)				
Correct	328.45	37.25	229	382
Error	50.01	25.29	12	128
Missed	19.10	14.78	2	79
Average RT (ms)	462.08	27.32	387.19	531.92
Correct	468.95	26.22	396.26	541.89
Error	418.52	38.73	304.70	517.18
Post-correct	459.83	27.56	389.89	538.46
Post-error	480.47	40.32	335.81	599.08

Note. $N = 188$. Participants completed 400 trials. The average accuracy rate was 82.11 %, the average inaccuracy rate was 12.50 % and the average rate of missed responses was 4.77 %.

both Ruchensky et al. (2020; $N = 100$) and the present study ($N = 188$) were adequately powered to detect this association, should it exist. Previous studies with comparatively smaller samples may have run the risk of having spurious findings due to insufficient statistical power (i.e., "false positive"; Button et al., 2013). While further replications are required, the lack of effect of IU subfactors on ERN amplitude suggests that studies previously reporting these associations should be interpreted with caution.

Regarding post-error slowing (PES), we did not replicate the observations of an effect of IU subfactors on post-error RT (Jackson et al., 2016). In their original paper this analysis is referred to as PES, however, this approach does not accurately index PES because it considers post-accuracy RTs (i.e., error and correct responses) separately, rather than a comparison of both in one index (i.e., how much is the next correct trial delayed by a previous mistake compared to the response after a correct trial). Accordingly, two approaches have been defined for calculating PES: (a) PES defined as the difference between average RT after error trials and the average RT after correct trials, and, (b) PES as the mean of the differences from the RT of the correct trial after an error minus the RT of the correct trial before that error (Dutilh et al., 2012; Schroder et al., 2020). In line with Ruchensky et al. (2020), using the former so-called traditional PES, we also obtained a null result, whereby total IU could not predict PES in our sample (Ruchensky et al., 2020). In sum, we did not find evidence for an association of IU and behavioral measures. It is important that studies investigating behavioral adjustments after error commission consider improved estimates of PES to ensure reliability and internal consistency as recommended by Schroder et al. (2020) and Sandre et al. (2020).

Our study also found no relationship between error processing and symptoms of depression or anxiety. We found no main effect of depression on the ERN which is in line with other research showing no relation (Moran et al., 2017; Ruchensky et al., 2020). We also failed to find an association between trait anxiety and the ERN. Previous studies have advanced the ERN as a potential biomarker for anxiety (Meyer, 2016), but the meta-analysis of Saunders and Inzlicht (2020) found that publication bias may have exaggerated the relationship between trait anxiety and the ERN, with no overall correlation for volunteer non-clinical samples. Our null results from a sample of undergraduates support this notion and relate to other work that has shown that trans-diagnostic anxiety-depression symptom dimensions are not related to ERN magnitude (Seow et al., 2020). While meta-analytic work has shown a small effect for increased ERN amplitude in internalizing disorders (Pasion and Barbosa, 2019), the effects of attenuated ERNs in externalizing disorders are larger overall (Lutz et al., 2021; Pasion and Barbosa, 2019). As such, investigations into error processing in externalizing disorders and its relationship to intolerance of uncertainty deserve attention, particularly given findings showing that the desire to remove uncertainty is implicated in impulsive risky behaviors, such as substance abuse (Sadeh and Bredemeier, 2021).

Based on our lack of direct effects, it was conceivable that the broad
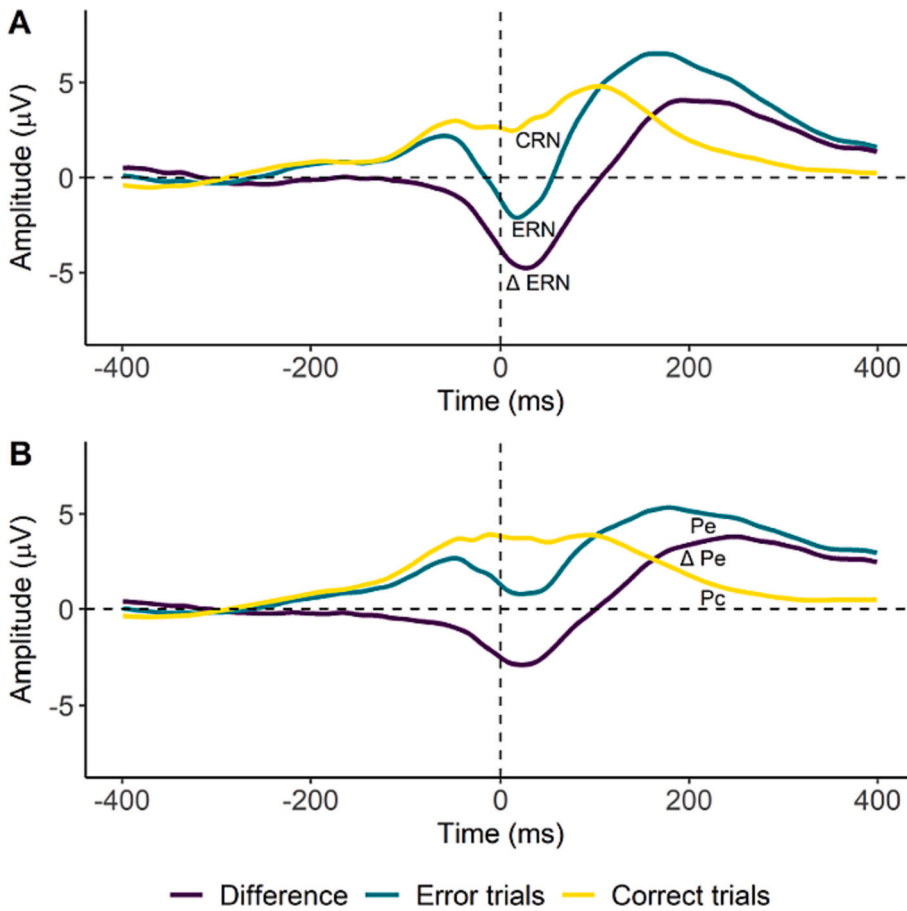
Fig. 3. Response-locked grand average waveforms showing the ERN and Pe. Panel A shows grand average activity for error and correct trials at Cz. Panel B shows grand average activity for error and correct trials averaged across Cz and Pz. 0 ms represents response commission. Event-related potentials are labelled, indicating the peak of each wave between 0 and 100 ms post-onset (panel A) and 200–400 ms post-onset (panel B). ERN = error-related negativity; CRN = correct-related negativity; Pe = error-related positivity; Pc = correct-related positivity; Difference = grand average amplitude of error trials minus average amplitude correct trials.

Table 3
Standardized beta coefficients (β) for regression analyses predicting the ΔERN.

|	Model 1	Model 2	Model 3
Predictors (depression)			
Depression	0.03	0.02	0.03
Total IU	0.04	0.03	0.04
Depression × IU	–	–	<−0.01
Predictors (trait anxiety)			
Trait anxiety	0.04	0.03	0.02
Total IU	0.04	0.03	0.03
Trait anxiety × IU	–	–	<−0.01
Predictors (worry)			
Worry	−0.01	−0.03	−0.03
Total IU	0.04	0.05	0.06
Worry × IU	–	–	<−0.01

Note. $N = 188$. Model 1 = separate predictors; model 2 = simultaneous predictors; model 3 = simultaneous predictors plus interaction term. No coefficients were significant at $p < .05$. These null results still held when using the absolute ERN amplitude as an outcome variable (see Supplemental material).

Fig. 4. Relationship between depression and the ΔERN at different levels of IU. The relationship between depression and ΔERN at low (red line; −1 SD), medium (blue line; mean) and high (green line; +1 SD) levels of IU. There were no main effects or an interaction between any variables in predicting the outcome. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

measure of total IU might moderate the relationship between depression and the ERN, such that depression and the ERN are only related at low levels of IU (Ruchensky et al., 2020). However, this was not the case for our sample. Moreover, our exploratory analysis showed no interaction between trait anxiety and total IU in explaining the ERN. The fact that total IU or IU subfactors were not associated with symptoms or the ERN/Pe in our models was surprising given that errors have been conceptualized as inherently uncertain threats (Weinberg et al., 2016). Despite our null results, approaching errors as endogenous forms of threat does remain valid, particularly considering the neural and behavioral commonalities shared by errors and exogenous threats (e.g., heightened skin conductance response and pupil dilation, increased amygdala activity;
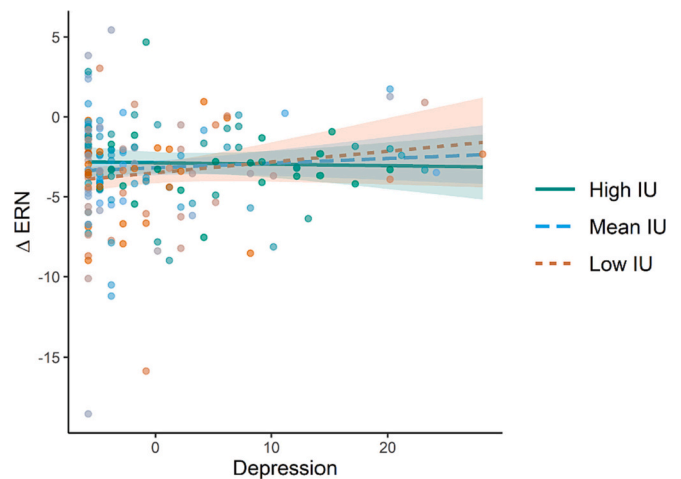
Hajcak and Foti, 2008; Hajcak et al., 2003; Pourtois et al., 2010). The idea that the IU subfactors should differentially predict ERN amplitudes is elegant because these subfactors capture different ways people respond to unpredictability, either by engaging cognitive control (P-IU) or by inhibition of action (I-IU). However, these constructs overlapped somewhat in our sample ($r = 0.61$) which raises questions about their
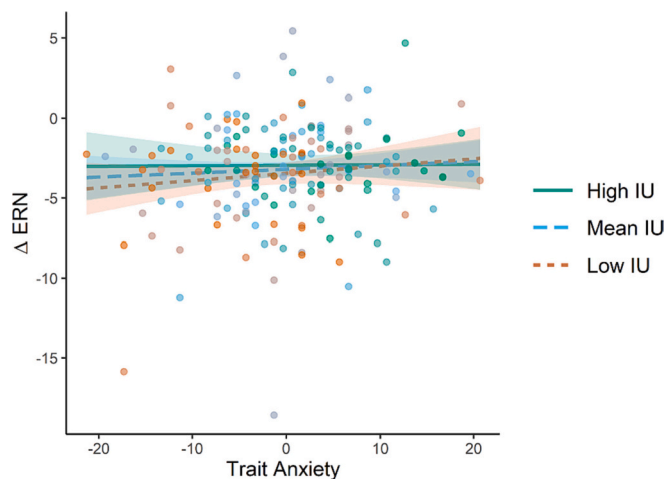
Fig. 5. Relationship between trait anxiety and the ΔERN at different levels of IU. The relationship between trait anxiety and ΔERN at low (red line; −1 SD), medium (blue line; mean) and high (green line; +1 SD) levels of IU. There were no main effects or an interaction between any variables in predicting the outcome. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 4
Standardized beta coefficients (β) for exploratory regression analyses.

Predictors	Outcome				
	CRN	ΔERN	Pe	Pc	ΔPe
Total IU	−0.07**	0.04	−0.03	−0.01	−0.03
Simultaneous predictors					
P-IU	−0.08	−0.03	−0.03	0.02	−0.04
I-IU	−0.06	0.15	−0.05	−0.05	<−0.01

Note. $N = 188$. P-IU = Prospective intolerance of uncertainty; I-IU = Inhibitory intolerance of uncertainty.

** Indicates $p < .01$.

usefulness relative to the total IUS-12 score.

Questions about the validity of the IUS-12 subfactors appear in the extant literature, with support being found for bifactor models with a high loading on a general IU factor (Bottesi et al., 2019; Hale et al., 2016; Lauriola et al., 2016; Shihata et al., 2018; Yao et al., 2021). However other groups have shown discriminant validity for P-IU and I-IU relating to psychopathology (Boelen et al., 2015; McEvoy and Mahoney, 2011; Penney et al., 2020; Saulnier et al., 2019). While considering the IU subfactors as separate constructs may be useful for distinguishing psychopathology (Penney et al., 2020; Yao et al., 2021), research designs employing additional neural, behavioral and psychophysiological approaches become complex. In particular, associations are harder to find when measurement levels exist at different levels of abstraction (Brandt and Mueller, 2022), such that trait personality variables (e.g., IU) can exist at highly abstracted levels of feeling and behavior and therefore may struggle to predict specific contextualized neurobiological variables (e.g., ERN). Moreover, adjacent research from our lab involving impulsivity has shown that self-report and EEG measurement levels can have within- but not between-level correlations (Bernoster et al., 2019). Consequently, more research is needed before linking IU to ERP measures of neurocognitive processes. Studies that validate the assessment of trait IU via self-report measures as well as other units of analysis should be prioritized, especially with regard to accumulating evidence that the IU subfactors do not link well with the ERN.

Any critique of the IU subfactors is tempered by the fact that total IU was also not a good predictor in the current study: Total IU could not predict the ERN, Pe or Pc. The only significant regression result in this study was the finding that total IU (but not subfactors) predicted a larger

(i.e., more negative) CRN. This effect was independent of the relationship of total IU and trait anxiety, showing the unique contribution of IU to elevated CRN. This finding is somewhat consistent with a previous study showing that prospective and inhibitory IU were associated with a larger CRN (Sandre and Weinberg, 2019), although they did not report on total IU. High IU constitutes a risk and maintaining factor for OCD (Tolin et al., 2003), and IU has been shown to predict unique variance in OCD symptoms above other cognitive processes such as perfectionism (Fergus and Wu, 2010). It has been posited that a desire to reduce uncertainty may underlie the compulsions in the disorder (particularly repetitive checking behavior; Tolin et al., 2003). Our finding supports the notion of Sandre and Weinberg (2019) that enhanced CRNs may reflect the salience of correctness, and may explain why hypervigilant checking behavior persists despite a registration of having performed correctly. Such associations between IU and the CRN reflect the need for more research and replications in clinical populations, perhaps integrating disorder-specific forms of IU that have been distinguished from dispositional IU (Shihata et al., 2017). Task-elicited uncertainty may also prove relevant given that some conceptualizations of the CRN regard it as sensitive to uncertainty of responses *in general*, rather than merely indexing commission of correct ones (Pailing and Segalowitz, 2004). This suggests the CRN could be relevant to performance monitoring under conditions of uncertainty. Along this line Sandre and Weinberg (2019) found relationships between I-IU and flanker trials where correctness was ambiguous. Such adaptions to the flanker task and other paradigms may help understand if the relationship between performance monitoring and IU depends on the task, with the possibility for divergent relations across different forms of psychopathology. As noted by Sandre and Weinberg (2019), it is possible that IU is more related to the recruitment of cognitive control under uncertain conditions, rather than performance monitoring per se.

While adaptions to the flanker task may prove fruitful in clarifying relationships, we do rule out the role of paradigm considerations in explaining our null results. In line with others, we assume that errors can be considered as endogenous and unpredictable forms of threat (Proudfit et al., 2013; Weinberg et al., 2016). However, errors committed during a flanker task may only elicit a relatively mild level of threat compared to other paradigms employing external threats. Individual differences in threat responding (e.g., fear-potentiated startles, skin conductance responses) have been observed in fear-conditioning tasks employing various stressors (e.g., electrical shocks, bursts of white noise/air; see Lonsdorf et al., 2017). An attenuated level of threat/ uncertainty generated by errors compared to external threats may explain our null results; however, there are also good reasons for assuming that even the mild uncertainty evoked by errors can moderate responses in high dispositional IU. This has been argued by and shown in the earlier studies using the flanker paradigm (Jackson et al., 2016; Ruchensky et al., 2020), underlying that IU/uncertainty distress need not necessarily be evoked or manipulated directly at the task-level.

Our null results were against expectations meaning several general explanations must be considered. Our sample was adequately powered to find a small-to-medium effect if one existed, indicating that sample size was not an issue; indeed, this study had almost triple the sample size of Jackson et al. (2016) and Sandre and Weinberg (2019) whom we sought to replicate. Nevertheless, our power was based on effect sizes from published results which may have been exaggerated. The self-report measures employed were reliable, with internal consistency estimates ranging from good to excellent for all measures; the distribution of IUS-12 scores reflected other undergraduate and community samples (Carleton et al., 2012; see Supplemental material). Moreover, our data reflected the typical behavioral and electrophysiological patterns expected for a flanker task. The use of the ΔERN (i.e., ERN minus CRN) has been criticized because subtraction-based difference scores remain highly correlated with their constituent signals, meaning the ΔERN is not independent of the CRN (Meyer et al., 2017). As such, the use of a difference score may not isolate neurophysiological activity specific to

error processing and can also lower between-subject variance. Based on this, our analyses were additionally performed using the absolute ERN/Pe (instead of ΔERN/ΔPe) as a predictor; all effects were all non-significant, suggesting that the use of a difference score did not bear on the null results observed in this study. This approach of comparing different methods of calculating the results is aligned with the objective of the experimental replication (Hudson, 2021), by testing the variables in different but equivalent ways we can ensure that the effects found are there (or not, as in our case), and do not depend on the specific experimenter choice either in design or analysis steps. Finally, our data fulfilled the assumptions for the various statistical tests which points to the robustness of our null results.

4.1. Limitations

A couple of limitations of our study need to be mentioned. First, the whole data collection took place during the COVID-19 pandemic, which in itself constitutes a situation of permanent uncertainty. Especially when investigating anxiety-related constructs, we need to acknowledge that the lab situation before and during the pandemic cannot be compared. This may be a general issue for clinical research. Second, we used a slightly different experimental design (e.g., letters instead of arrows and faster stimuli presentation) and EEG analysis approach (e.g., EEG signal re-referenced to average instead of mastoids and ERN calculated with single electrode instead of pool of electrodes) compared to the original studies, which however is supported both by previous research using the same experimental design of a flanker task to assess performance monitoring (e.g., Franken et al., 2010) and methodological research testing different analytical pipelines of the flanker task (Sandre et al., 2020). Third, due to the diversity in our sample, we had participants answering the questionnaires in their native language (Dutch, $n = 103$), while international participants received the questionnaires in English, which for most of them was their second language ($n = 85$). However, internal consistency in our sample was good for both versions (see Supplemental material). Fourth, to keep our variables comparable with our reference papers, we did not use a more robust index of the PES. Finally, the BDI-II scores were skewed towards the lower end of the scale (i.e., less severe symptoms) compared to our replication reference (Ruchensky et al., 2020; see Questionnaires). Contrary to our sample, the means and variance of Ruchensky et al. (2020) are comparable to the norms of undergraduates in both the USA (Whisman and Richardson, 2015) and the Netherlands (Roelofs et al., 2013). Thus, our relative lack of depression symptoms could have obscured the relationships between depression and other study variables.

4.2. Implications and conclusions

Altogether, our study shows the need for high-powered replication studies in clinical and individual differences (EEG) research, which is currently underway in different EEG Many Labs replication attempts (Pavlov et al., 2021). Our high-powered replication failure of linking intolerance of uncertainty to an electrophysiological index of error-monitoring underlines the need for further replicability and reproducibility within psychophysiology. Attempts should be made to replicate studies with small samples. Recommendations for transparent and open-science practices in psychophysiology have been discussed extensively elsewhere (Garrett-Ruffin et al., 2021) and will contribute to the viability of replication attempts while making sure findings are disseminated regardless of statistical significance. In a field that is particularly time- and resource- intensive, efforts are better directed at theory-testing related to uncertainty as opposed to discovery-oriented approaches which run the risk of Type 1 errors and non-replicability (Brandt and Mueller, 2022). While the ERN and IU have trans-diagnostic potential to cut across multiple forms of psychopathology, further rigorous and incremental work is needed to see if, and how, they are related.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijpsycho.2022.07.001.

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