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# Associations between GAD symptom severity and error monitoring depend on neural quenching variability

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## Abstract

Generalized anxiety disorder (GAD) has been inconsistently associated with exaggerated threat monitoring measured via the error-related negativity (ERN). This suggests the need to consider whether the link between GAD and ERN is influenced by additional processes, such as cognitive inhibition of non-threat. The current study explored this possibility by employing a novel, trait-like measure of cognitive processing inhibition, neural quenching (NQ). Electroencephalography was recorded while 16 adults diagnosed with GAD and 14 age-matched healthy controls viewed angry and neutral faces prior to individual trials of a flanker task. NQ was generated to aversive (angry) and non-aversive (neutral) facial primes, and the ERN was generated to incorrect and correct responses on the flanker task. We tested the hypothesis that higher GAD symptom severity would be associated with larger-magnitude ERN when NQ to non-aversive was enhanced (higher levels of non-aversive processing inhibition), but with blunted ERN when NQ to non-aversive was also blunted (lower levels of non-aversive processing inhibition). Overall, greater NQ to non-aversive faces was associated with larger-magnitude ERNs. As predicted, higher GAD symptom severity was associated with blunted ERN when accompanied by blunted NQ to non-aversive. Findings suggest that exaggerated threat processing is not uniform in GAD and may depend on individual differences in the ability to inhibit processing of non-aversive and other types of information.

**Keywords** Neural quenching · Error related negativity · Anxiety · Overgeneralized Fear

Generalized anxiety disorder (GAD) is among the most commonly diagnosed anxiety disorders (Ballenger et al., 2001) affecting an estimated 5.7% of the adult population in their lifetime (Eisenberg & Lipson, 2017; Kessler et al., 2012; Kroenke et al., 2007). A range of cognitive processing disruptions appear to underlie the emergence and progression of GAD symptoms, including exaggerated response monitoring (e.g., Weinberg et al., 2010, 2012, 2015) in which processing of aversive rather than non-aversive experiences (e.g., errors) is prioritized. For example, disruptions in response monitoring in GAD include exaggerated error monitoring and processing that can be captured by neural measures (e.g., faster, more salient detection of an error response; Van Veen & Carter).

Exaggerated response monitoring can be measured using a response-locked event-related potential (ERP) called the error-related negativity (ERN; Gehring et al., 1990). The ERN is a sharp negative deflection measured in anterior EEG electrodes peaking within about 100 ms after an incorrect response, and with neural generators identified in the anterior cingulate cortex (Brázdil et al., 2005; Holroyd et al., 1998; Pizzagalli et al., 2006; Stemmer et al., 2004; Yeung et al., 2004). Functionally, the ERN reflects the relatively rapid and automatic detection and monitoring of making an error (Falkenstein et al., 2000; Van Veen & Carter, 2002). Importantly, errors are considered intrinsically aversive relative to a correct response (e.g., Aarts et al., 2012, 2013), and thus the ERN has been utilized as a metric of the processing of aversive, threat-relevant information.

Some studies have found that, relative to healthy controls, adults diagnosed with GAD show larger-magnitude ERNs following the commission of an error relative to a correct

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response (Weinberg et al., 2010, 2012, 2015). Yet, other studies document that GAD is associated with broader disruptions in error-related brain activity, including exaggerated processing of correct responses that are non-aversive (Denefrio et al., 2019; Endrass et al., 2010; Xiao et al., 2011). For example, in one study, adults diagnosed with GAD, compared to healthy controls, showed greater magnitude error positivity to non-aversive correct responses, suggesting overgeneralized threat monitoring at a temporal window slightly later than the ERN (Denefrio et al., 2019).

Although methodological differences, including diagnostic status, time-scale of measurement, and level of analysis (brain versus behavior), may help explain this heterogeneity of findings (see also, Amir et al., 2009; Nitschke et al., 2009), it remains unclear whether individual differences in anxiety-related overgeneralized threat monitoring (Dymond et al., 2015; Lissek et al., 2014) influence the magnitude of the ERN. In other words, if a study fails to detect an exaggerated ERN in GAD, it may be due to disruptions in processing inhibition of *non-aversive* stimuli during threat monitoring. In this case, threat processing would extend to the processing of non-aversive stimuli as *potentially aversive*, thus blunting the ERN (because it is a metric of differential processing of aversive relative to non-aversive responses). The goal of the present study was therefore to test whether the association between GAD symptom severity and ERN was sensitive to individual differences in processing inhibition of non-aversive stimuli more broadly.

A novel measure, derived from EEG, called neural quenching (NQ) provides a temporally sensitive measure of processing inhibition that would allow testing of this hypothesis. Neurophysiological activity in the human brain is highly variable over time (Arazi, Censor, et al., 2017; Arazi, Gonen-Yaacovi, et al., 2017; Arieli et al., 1996; Faisal et al., 2008; Goris et al., 2014). This trial-by-trial neural variability changes across contexts and perceptual tasks, such that it is relatively large in pre-stimulus period and relatively small (quenched) soon after a stimulus is presented (within ~ 200 ms). This phenomenon of NQ suggests that sensory stimulation reduces ("quenches") ongoing neural variability (e.g., Churchland et al., 2010), which in turn allows the brain to flexibly amplify or inhibit perceptual signals (Garrett et al., 2013).

Notably, NQ is a highly stable, trait-like individual difference (e.g., Arazi, Censor, et al., 2017; Arazi, Gonen-Yaacovi, et al., 2017), and shows strong associations with behavioral and perceptual performance. For example, individuals evidencing potentiated NQ during a behavioral task show superior performance (e.g., faster reaction times; Arazi et al., 2019) and facilitated perceptual discrimination (e.g., smaller contrast discrimination thresholds; Ayelet et al., 2016). Because greater NQ has been correlated with inhibition of broadband neural oscillations, NQ is thought to boost task performance because it facilitates gain modulation by making neural activity less variable and thus more reproducible across trials (Ayelet et al., 2016), by reducing trial-level response variability of single neurons (Mitchell et al., 2007) and by coordinating responses across neuronal populations (Cohen & Maunsell, 2009; Mitchell et al., 2009).

Since greater NQ is associated with inhibition of neural activity (Garrett et al., 2013), it is thought to reflect general efficiency of processing inhibition. For example, in a recent study, Arazi and colleagues found that when attention control was experimentally amplified, NQ was increased and participants showed greater performance benefits of competing attention cues (Arazi et al., 2019). In the present study, we expected that greater NQ to non-aversive stimuli may boost inhibition of such processing, thereby facilitating targeted response monitoring of aversive errors, leading to larger-magnitude ERNs.

The goal of the present study was to test whether the association between GAD symptom severity and ERN was sensitive to individual differences in processing inhibition measured via NQ. To do so, we reanalyzed data from a previously published study examining the ERN in adults diagnosed with GAD (i.e., Denefrio et al., 2019). We quantified NQ to non-aversive (neutral faces) and aversive (angry faces) stimuli presented prior to flanker arrays on each trial of a speeded flanker task, from which ERN to incorrect versus correct responses were generated. We were particularly interested in quantifying the NQ to these "task-irrelevant" stimuli (the faces), because it allowed us to test for broader disruptions in the processing inhibition of non-aversive stimuli, as predicted. In order to capture the full spectrum of anxiety severity, we used a continuous measure of GAD symptom severity (GAD-Q; Newman et al., 2002) instead of using clinical diagnostic group, which was used in the prior study. We tested the hypothesis that greater GAD symptom severity would be associated with greater error monitoring (larger-magnitude ERN) when NQ to non-aversive stimuli is enhanced (greater processing inhibition) but would be associated with blunted error monitoring when NQ to non-aversive stimuli is blunted.

## Method

### Participants

Potential participants were recruited and screened to participate from Hunter College campus and community. Participants consisted of a group of adults with a primary diagnosis of GAD and a group of aged-matched healthy controls. Part of the data used for this manuscript were previously published, but used to test distinct hypotheses (Denefrio et al., 2019). The Structured Clinical Interview for DSM-IV

Disorders (SCID-I/P; First & Spitzer, 2002) was used to screen for elevated anxiety and generalized anxiety disorder (GAD). The interviews were conducted by trained personnel, and the reliability was determined using the clinician severity rating (CSR) from the Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV; DiNardo et al., 1994). Reliability was examined in 60% (9/15) of participants, and with 100% agreement indicating CSR ratings were at or above the clinical level of severity ($k = 1.00$, $p < 0.001$; see Denefrio et al., 2019 for details). Individuals who met criteria for a current primary GAD diagnosis were included. Comorbidities with additional anxiety disorders and mood-related disorders were allowed given the high comorbidity between GAD and these disorders (Ballenger et al., 2001). The exclusion criteria were (1) current major depressive episodes, (2) suicidality, or (3) psychotic symptoms. After the screening procedure, 40 participants were invited to complete the study. Out of these individuals, nine participants were excluded from analysis due to committing fewer than four errors per experimental block, and one participant was excluded from analysis due to exhibiting subclinical symptom of GAD after review at the consensus meeting. All GAD participants were not taking medication and the control participants did not meet criteria for any Axis I disorder at the time of participation (see Denefrio et al., 2019). The final sample consisted of 30 participants ($M_{age} = 22.2$, $SD_{age} = 5.03$; 24 [80%] females). Self-reported race/ethnicity was as follows: 14 (46.7%) Asian, 4 (13.3%) White, 2 (6.7%) Black or African-American, 1 (3.3%) reported other, and 9 (30%) opted to not report this information. Further, 8 (26.7%) participants were Hispanic or Latinx and 22 (73.3%) were not Hispanic or Latinx (see Denefrio et al., 2019 for detail). As reported in the original paper, we used G*Power (Faul et al., 2007) to compute the achieved power range as 94%—99% power with our alpha criteria ($p < 0.05$), sample size ($N = 30$), and medium to large effect sizes detected for behavioral measures of performance ($\eta_p^2 = 0.12$) and neural measures ($\eta^2 = 0.14$–$0.17$).

## Materials and procedures

Following consent procedures, participants completed a self-report questionnaire to assess levels of anxiety and anxiety-related symptoms. In addition, the SCID was conducted by a trained lab personnel, followed by a computerized modified flanker task that included emotional faces as primes while EEG was continuously recorded. Each study session lasted approximately two and a half hours.

## Self-report measure

*Generalized Anxiety Disorder Questionnaire- IV* (GAD-Q IV; Newman et al., 2002). The GAD-Q IV is a revised edition of the Generalized Anxiety Disorder Questionnaire (GAD-Q; Roemer et al., 1995) which is comparable to an interview for clinical diagnosis of GAD (Roemer et al., 1995). The self-reported questionnaire consisted of nine questions assessing all symptoms of GAD as listed in the DSM-IV with high reliability in this study (Cronbach's Alpha = 0.83). The total sum scores are created by adding up the first four items that are coded into "1" for "yes" and "0" for "no" assessing the presence of symptoms (e.g., "Do you experience excessive worry"). Items 5 and 7 ask for a list of topics of most frequent worry and anxiety-related physical symptoms respectively, with each topic or symptom is given 1 point up to 6 points per item, which is then divided by 3. Lastly, questions 8 and 9 assess the degree of distress and interference, with each score divided by 4. Total scores ranged from 0 to 12, higher scores indicating higher GAD symptom severity.

### Modified flanker task

All participants completed a modified version of flanker task with facial primes (aversive and non-aversive) and without a face prime (no face). Participants were instructed to identify the direction (right versus left) of the central arrow that was "flanked" by four arrows presented in pairs on each side of the central arrow. Arrows were facing either the same direction (i.e., congruent trial) or the opposite direction (i.e., incongruent trial; Eriksen & Eriksen, 1974). The entire task consisted of three different blocks (non-aversive, aversive, and control), during which either one of the two face types (modified flanker task) was presented, or no facial stimulus (original flanker task) was presented.

Faces displaying emotions were used as stimuli based on previous literature probing anxiety-related attention and processing of threat or aversion. The stimuli ($177 \times 228$ pixels) were taken from the NimStim database (Tottenham et al., 2009), and were presented in grayscale against a white background. Stimuli consisted of 32 facial expressions of 16 actors (equally divided between males and females) portraying angry (aversive) and neutral (non-aversive) expressions, and each stimulus was presented 30 times. The order of presentation of the blocks was fixed (no face, non-aversive face, then aversive face). Each block consisted of 480 trials, with 240 incongruent and 240 congruent flanker trials. On each trial, participants were presented with a face (500 ms), fixation period (variable 100–300 ms), flanker (congruent or incongruent for 100 ms), response time (up to 1700 ms), and intertrial interval (varied 1700–2300 ms).

### EEG recording and data reduction

Continuous EEG was recorded using BioSemi system (Biosemi; Amsterdam, Netherlands) via 64 Ag/AgCl scalp

electrodes. Electrodes were applied to an EEG cap and arranged in accordance with the international 10–20 system, and the activity was recorded at a sampling rate of 512 Hz. Preamplification of EEG signal was done for each electrode for improvement of the signal-to-noise ratio. Eye movement artifacts were monitored by electro-oculogram (EOG) using facial electrodes placed one centimeter above and below the left eye (for vertical eye movements) and one centimeter on the outer corner of each eye (for horizontal eye movements). During EEG acquisition, the voltage from each electrode was referenced online to the common mode sense active electrode, which produces a monopolar (nondifferential) channel. Acquired data were processed using Brain Vision Analyzer (Version 2.2, GmbH; Munich, Germany). All data then were re-referenced offline to an average reference and filtered with a high pass cut-off of 0.1 Hz and a low pass cut-off frequency of 30 Hz. Following ocular correction procedure (Gratton et al., 1983), artifacts were identified using the following criteria and removed from analyses: any data with voltage steps greater than 50 μV, changes within a given segment greater than 300 μV, and activity lower than 0.5 μV per 100 ms. After data artifact identification, ERPs were quantified where each ERP component was at its maximum (see Denefrio et al., 2019 for results with ERP data).

Response-locked data were generated for each trial starting from 200 ms each pre-response onset to 1000 ms post stimulus onset. For baseline correction, the 200 ms window from − 200 ms to 0 ms pre-response onset was used. The ERN was calculated as the average amplitude between 0 and 100 ms at FCz. For each participant, an average ERN was calculated separately for error and correct incongruent trials and by stimulus (face) type (no face, non-aversive, aversive). ERN results are based on incongruent trials only, because almost all errors were made exclusively on incongruent trials (Denefrio et al., 2019; Santesso & Segalowitz, 2009; Segalowitz et al., 2010). Further, ERN difference scores were calculated (i.e., ERN-incongruent incorrect minus ERN in congruent correct; ΔERN) for each stimulus type. For all main analyses we used these difference scores to be consistent with previous research. More negative ΔERN difference scores indicate more exaggerated error monitoring of the stimulus. ΔERN for correct versus incorrect trials are presented in Fig. 1.

### Quantifying neural quenching

Neural quenching was quantified following procedures from Arazi et al. (2017), Arazi et al. (2017)) and Arazi et al. (2017). We used the formula function in Brain Vision Analyzer to generate variance across artifact-free trials, separately at each electrode and for each participant. This yielded variance at each millisecond across the entire segment (i.e., − 200 ms to 500 ms). Next, the average absolute variance was exported in pre-stimulus (baseline: − 200 to 0 ms) and post-stimulus (150 to 400 ms) intervals separately for each condition (aversive face, non-aversive face). Electrodes were selected based on those showing strongest visual P1 responses (PO7, PO8) and remaining consistent with the prior publication of data from this sample (Denefrio et al., 2019). Exported variance was averaged across electrodes for each participant, separately for each interval and condition. Finally, neural quenching was quantified for each condition and participant by computing difference scores (pre-stimulus variance minus post-stimulus variance), with greater scores indicating greater neural quenching to faces. Figure 2 shows NQ to aversive (angry) and non-aversive (neutral) faces for the sample as a whole.

### Data analytic strategy

We tested the hypothesis that higher GAD symptom severity would be associated with greater error monitoring (larger-magnitude ΔERN) when NQ to non-aversive stimuli is enhanced (greater processing inhibition) but would be associated with blunted error monitoring when NQ to non-aversive stimuli is blunted. We expected that this interaction effect would not reach significance for NQ to aversive stimuli (angry faces).

We conducted two linear regressions using SPSS PROCESS (version 3.5; Hayes & Scharkow, 2013) to test for the moderating effect of NQ on the association between GAD symptom severity (GAD-Q IV) and ΔERN (difference scores of incongruent incorrect minus ERN congruent correct), separately for non-aversive and aversive stimuli. To capture the full spectrum of anxiety severity, we used a continuous measure of GAD symptom severity (GAD-Q). Step 1 was the anxiety severity, step 2 was the moderator NQ, and step 3 was the interaction between the two.

Means of continuous variables were centered in order to reduce potential multicollinearity. In addition to using the pick-a-point approach (Rogosa, 1980), which allows examination of the effects of the predictor (GAD symptom severity) on the dependent variable (ΔERN) at − 1 SD, 0 SD, and + 1 SD of the moderator (NQ to either neutral or angry faces), we used the Johnson-Neyman approach for regions of significance analysis to plot and probe the interactions (Johnson & Neyman, 1936). The Johnson-Neyman approach identifies the discrete values at which the moderator significantly interacts to affect the association between predictor and outcome.

## Results

### Descriptive statistics

The GAD ($n = 16$; female = 14) and control ($n = 14$; female = 10) groups had statistically equal numbers of

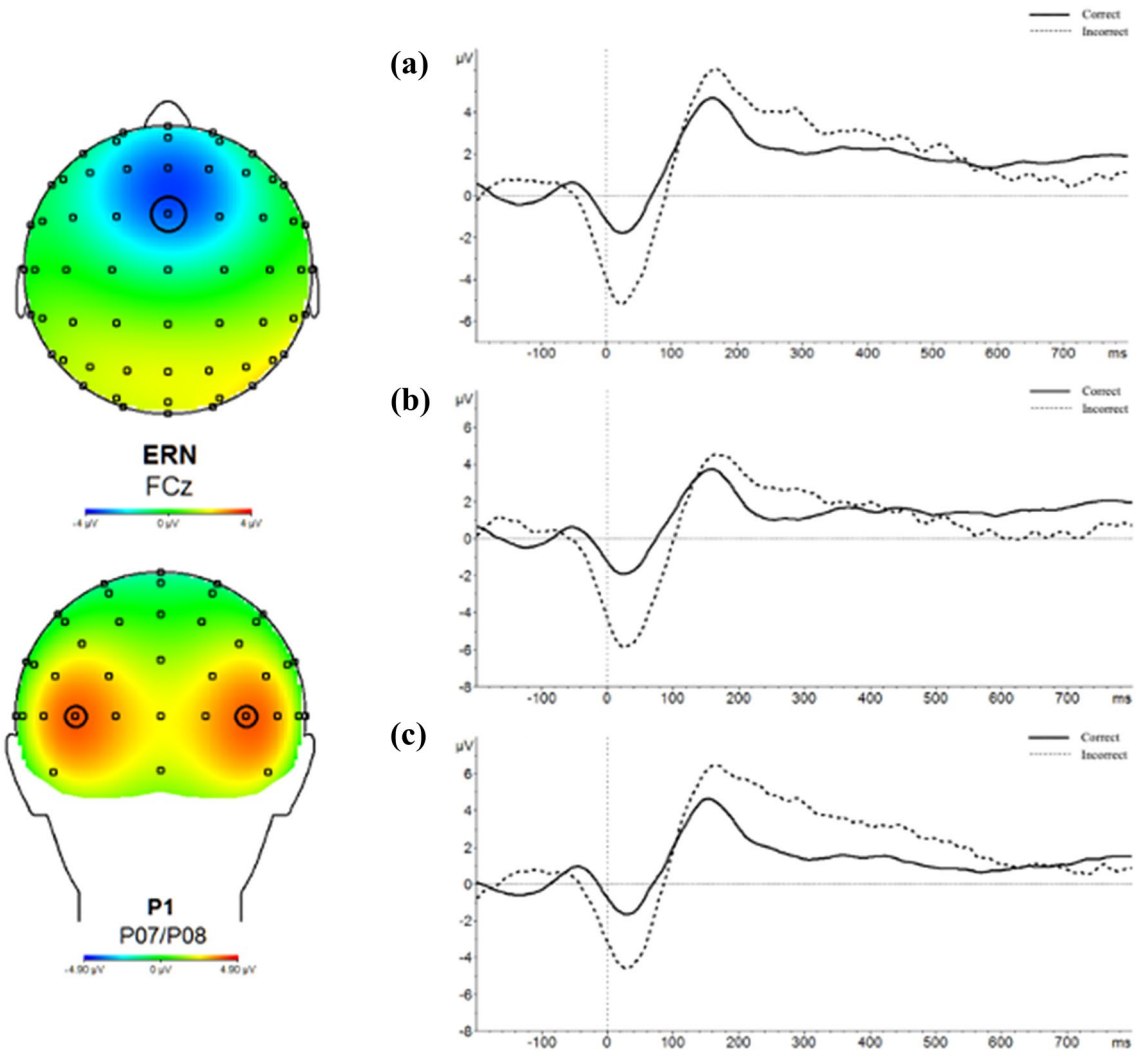


**Fig. 1** Headshots for ERN (FCz) and P1 (PO7 and PO8), and the ERN for correct versus incorrect trials **a** all participants, **b** high NQ, and **c** low NQ

males and females ($p > 0.05$). As expected, the GAD group scored significantly higher than the Control group on the GAD-Q [$t(17.4) = 5.30$, $p < 0.0001$; see Table 1 for descriptive statistics].

Correlations among study variables [GAD symptom severity (GAD-Q), neural quenching, and ΔERN] were assessed using Pearson's bivariate correlations. No correlations reached significance (all $p$'s $\geq 0.16$).

## Primary analyses

As shown in Table 2, there was a significant main effect of NQ to non-aversive stimuli on ΔERN magnitude, such that greater NQ to non-aversive neutral faces was associated with larger-magnitude ΔERNs ($b = -0.0096$, $p = 0.033$). Pick-a-Point analysis revealed a significant interaction ($R^2_{change} = 0.22$, $p = 0.009$), indicating that those reporting greater GAD symptom severity displayed a blunted ΔERN, but only if, as predicted, they also evidenced blunted NQ to non-aversive neutral faces [$b = 0.50$, $t(26) = 2.33$, $p = 0.028$; Fig. 3]. The Johnson-Neyman analysis further revealed that the association between GAD symptom severity and blunted ΔERN became significant when NQ was 0.74 standard deviations below the mean (− 99.66), and at low NQ [− 358.80; $b = 1.22$, $t(26) = 2.82$, $p = 0.009$].

In addition, there was a trend such that those reporting greater GAD symptom severity displayed a greater-magnitude ΔERN, but only if, as predicted, they also evidenced high NQ to non-aversive neutral faces [$b = -0.36$, $t(26) = -1.68$, $p = 0.11$; Fig. 1]. The Johnson-Neyman

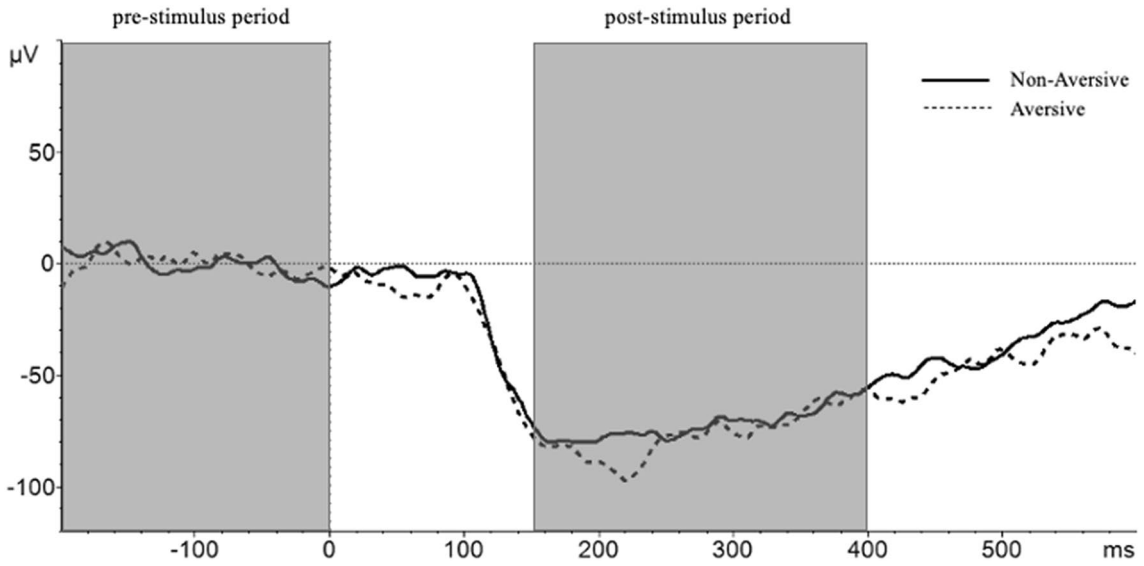


**Fig. 2** Time course of absolute trial-by-trial variance (neural quenching) for pre and post stimulus periods (− 200–0 ms and 150–400 ms, respectively) to aversive (angry) or non-aversive (neutral) faces derived from PO7 and PO8

**Table 1** Descriptive statistics on demographics and self-reported questionnaire

| | GAD ($n=16$) | Control ($n=14$) |
|---|---|---|
| Age | 22.19 (5.22) | 22.21 (4.99) |
| GAD-Q | 10.20 (1.42) | 5.30 (3.19) |
| NQ to non-aversive | 43.59 (123.73) | 98.22 (144.46) |
| NQ to aversive | 87.80 (168.90) | 75.77 (188.96) |
| ΔERN to no face | − 3.21 (3.69) | − 3.17 (3.47) |
| ΔERN to non-aversive | − 3.08 (4.35) | − 2.35 (3.28) |
| ΔERN to aversive | − 2.11(3.57) | − 2.64 (4.43) |
| ΔERN overall | − 2.80 (3.37) | − 2.72 (2.70) |

Mean (SD) for GAD and control groups. The ERN values are difference scores between incongruent incorrect trials minus incongruent correct trials

analysis further revealed the association between greater GAD symptom severity and exaggerated ΔERN became significant at about 1.43 standard deviations above the mean for NQ [193.21; $b=-0.55$, $t(26)=-2.06$, $p=0.05$], and also at the highest level of NQ [470.68; $b=-1.44$, $t(26)=$-2.58, $p=0.016$].

Taken together, results suggest that as GAD symptom severity increases, the ΔERN is blunted for those evidencing blunted NQ to non-aversive stimuli and that, conversely, the ΔERN is enhanced, at the level of a trend, for those evidencing enhanced NQ to non-aversive stimulus.

**Table 2** Regression model—effects of anxiety and NQ on ERN

| | *b* | SE *B* | *B* | $R^2$ | $\Delta R^2$ | *p* |
|---|---|---|---|---|---|---|
| Model | | | | | | |
| Step 1 | | | | 0.14 | 0.019 | 0.47 |
| GAD-Q | 0.12 | 0.17 | 0.14 | | | |
| Step 2 | | | | 0.24 | 0.057 | 0.31 |
| GAD-Q | 0.075 | 0.17 | 0.085 | | | |
| NQ to neutral | − 0.005 | 0.004 | − 0.202 | | | |
| Step 3 | | | | 0.53 | 0.22 | 0.009 |
| GAD-Q | 0.040 | 0.15 | 0.045 | | | |
| NQ to neutral | − 0.009 | 0.004 | − 0.39 | | | |
| GAD-Q × NQ to neutral | − 1.52 | 0.53 | − 0.51 | | | |

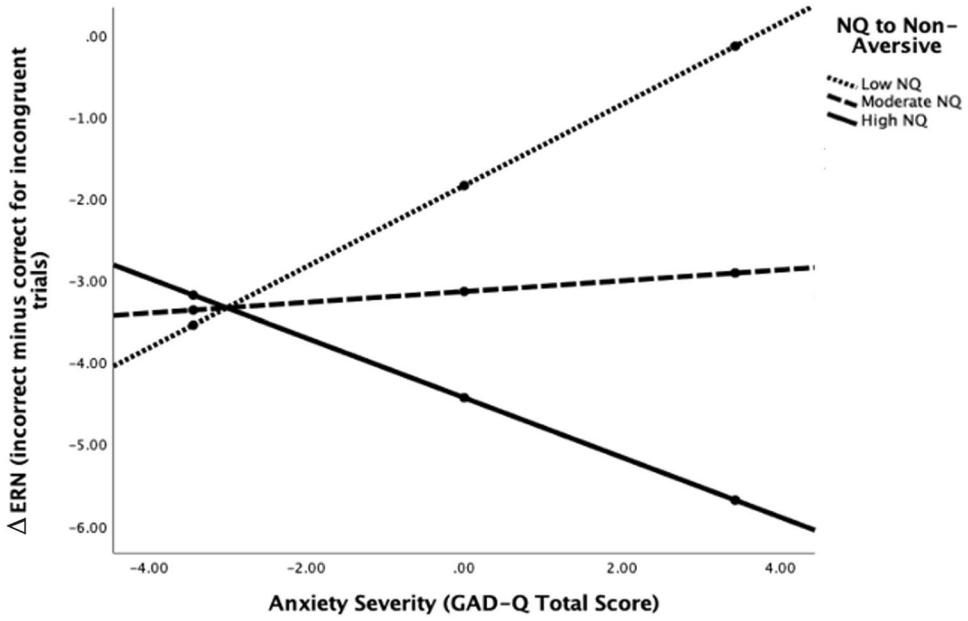


**Fig. 3** Higher anxiety severity was associated with blunted ΔERN, but only for those also showing blunted NQ to non-aversive neutral faces

As anticipated, regression analyses with NQ to aversive angry faces did not reach significance (models significant at $p = 0.79$).[1]

## Discussion

This study sought to clarify mixed findings on response monitoring disruptions in GAD measured via the ERN. Reanalyzing data from a previously published study (Denefrio et al., 2019) and adding a novel measure of processing inhibition, NQ, we found that the pattern of association between GAD symptom severity and the ERN depended on the magnitude of processing inhibition measured via NQ to non-aversive trials only. Specifically, individuals reporting greater GAD symptom severity evidenced a blunted ERN—dampened response monitoring of aversive errors—but only when they also showed blunted NQ to non-aversive stimuli, suggesting disrupted processing inhibition of non-aversive information. Conversely, at the level of a trend, those reporting greater GAD symptom severity also evidenced a greater-magnitude ERN—intact or exaggerated response monitoring of errors—when they instead showed enhanced NQ to non-aversive stimuli, suggesting intact processing inhibition of non-aversive information. Importantly, these patterns did not emerge for NQ to aversive stimuli, demonstrating the specificity of effects.

Although a significant literature posits that response monitoring is exaggerated in anxiety, and in GAD (Greenberg et al., 2013; Hajcak, 2012; Lissek et al., 2014; Weinberg et al., 2012), results of the present study suggest a more nuanced story. First, correlations did not show a simple, significant association between GAD symptom severity and ERN or NQ. This implies meaningful heterogeneity in these two anxiety-related cognitive disruptions that may work synergistically. Moreover, by examining the link between the two, and in the context of inconsistent patterns of links between GAD and the ERN, findings illustrate the importance of considering the processing of aversive and non-aversive information when examining cognitive disruptions in anxiety.

For example, a cognitive mechanism that is associated with anxiety, such as overgeneralized fear, can arise from both exaggerated fear learning (i.e., amplified fear acquisition and delayed extinction) and impaired safety learning (e.g., Lissek et al., 2005). These disruptions have been associated with the indiscriminate and excessive fear responses—overgeneralized fear—that characterize a broad range of anxiety symptoms (Craske et al., 2009; Dymond et al., 2015; Lissek, 2012; Pittig et al., 2018). In this study, we leveraged the temporal sensitivity of a novel neural index of processing inhibition, NQ, documenting that individual differences in overgeneralized threat processing influenced the association between GAD symptom severity and ERN. This suggests clinically relevant heterogeneity in the processing of aversive and non-aversive information that could

[1] As the previously published data (Denefrio et al., 2019) used diagnostic groups (GAD vs. Control), rather than symptom severity, to characterize anxiety, we ran the same moderation analyses entering the diagnostic grouping as the predictor variable instead of the continuous score of symptom severity. In the high GAD group, relative to health controls, higher NQ to neutral was associated with a more exaggerated ERN ($p = 0.044$).

inform the development of more personalized treatment and intervention approaches.

Although we predicted that the association between GAD symptom severity and ERN would be driven by individual differences in NQ to non-aversive stimuli, it is not clear why NQ to angry faces did not show significant direct associations with anxiety severity. One might posit, for example, that anxious individuals showing exaggerated response monitoring of errors would also tend to show disrupted neural quenching to aversive angry faces, suggesting blunted inhibition of threat processing, which is hypothesized to contribute to exaggerated error monitoring. However, in the original published study (Denefrio et al., 2019), anxiety-related ERP differences in both the ERN and other ERPs examined, including the later-emerging error positivity and the N170, appeared to be driven by neural responses to non-aversive correct responses and neutral faces. This is consistent with the idea that cognitive disruptions in GAD should be examined in relation to both threat and non-threat processing, rather than uniformly focusing on disruptions in response to aversive experiences and stimuli (Dunsmoor & Paz, 2015; Lissek et al., 2014).

By using neurophysiological measures of anxiety-related cognitive processes, limitations of self-report, like response biases, were minimized. However, because NQ is a relatively novel measure, it will be important to conduct further experimental examinations to better understand underlying mechanisms, and the degree to which it shows either trait-like continuity or contextual plasticity. In addition, because we were interested in capturing the full range of GAD symptoms, we used continuous ratings of GAD symptom severity rather than the diagnostic group as a between-subjects variable. Although patterns of results remained the same when we grouped participants as GAD-diagnosed or healthy controls, the continuous ratings of GAD symptom severity allowed us to consider the link between anxiety and ERN even in a non-diagnostic group, who did show non-zero levels of GAD symptoms. This is particularly relevant to disambiguating findings from the prior study, where the GAD sample had a primary diagnosis of GAD, but also evidenced comorbidities. GAD is comorbid with other disorders such as depression and other anxiety disorder 60% of the time (e.g., Goldstein-Piekarski et al., 2016), so capturing heterogeneity of symptoms rather than identifying only a binary distinction between diagnosed and non-diagnosed may provide a more generalizable set of findings given the heterogeneous clinical population at large. For example, comorbidity of depressive symptoms in our current sample may have influenced NQ (e.g., been associated with blunted NQ). However, given sample size limitations, it is difficult to parse out such effects. Future studies should examine the role of tertiary psychological variables such as depressive symptoms that could clarify the role of NQ in anxiety.

The present study targeted broader individual differences in the processing of aversive and non-aversive stimuli that might be relevant to response monitoring by quantifying NQ to task-irrelevant facial stimuli. Faces have the benefit of being rapidly and automatically processed, likely to elicit robust EEG-based metrics (e.g., ERN, N170; Denefrio et al., 2019), and have been used to assess dysfunctional emotional processing in GAD in previous studies (Mogg et al., 2000; Palm et al., 2011). Future studies should also examine individual differences in processing inhibition of complex or more emotionally arousing non-facial affective stimuli in relation to GAD-specific disruptions in response monitoring.

Taken together, results from the present study clarify inconsistent findings in the literature on GAD-related disruptions in response monitoring measured via the ERN, highlight the importance of employing complementary neurophysiological metrics to examine disrupted cognitive processes in anxiety, and have the potential to clarify etiological and treatment mechanisms that can inform the personalization of future therapeutic approaches.

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**Data availability** None of the data or materials for the experiments reported here is available, and none of the experiments was preregistered.

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