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Electrophysiological Indices of Performance Monitoring Across the Anxiety- and Obsessive-Compulsive Spectrum

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In Memory of My Late Mother

List of Publications

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Riesel, A., **Härpfer, K.,** Kathmann, N., & Klawohn, J. (2021). In the face of potential harm: The predictive validity of neural correlates of performance monitoring for perceived risk, stress, and internalizing psychopathology during the COVID-19 pandemic. *Biological Psychiatry: Global Open Science, 1*(4), 300-309. https://doi.org/10.1016/j.bpsgos. 2021.08.004

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Abstract

Errors are a ubiquitous part of human experiences, associated with potential risks but also serving as resources for improving performance. The neural monitoring of performance can be measured through event-related brain potentials (ERPs), such as the error-related negativity (ERN) and the correct-response negativity (CRN). Meta-analyses have identified an altered ERN as a trait-like risk marker for both internalizing and externalizing disorders, with increased amplitudes observed in anxiety and obsessive-compulsive disorders. However, this association is not consistent across all related diagnostic categories, often attributed to differing underlying transdiagnostic factors (e.g., trait worry). Challenging the assumed trait-like nature of the ERN, symptom fluctuations (e.g., state worry) may also influence the ERN. Furthermore, there is limited understanding of how an increased ERN contributes to psychopathology, along with only few studies evaluating multiple disorder categories and testing for effect specificity. This dissertation aims to refine the understanding of electrophysiological indices of performance monitoring across the anxiety and obsessive-compulsive spectrum by employing a transdiagnostic and dimensional approach that includes various diagnostic groups and ERPs.

Disentangling the associations of the ERN/CRN with trait and state worry, the preregistered randomized-controlled longitudinal study 1 (n = 90) did not indicate intraindividual variability of the ERN/CRN due to the experimental manipulation of state worry. Instead, higher levels of trait worry were linked to an enhanced ERN/CRN, particularly in women. Study 2 investigated the role of the ERN/CRN as risk markers using a preregistered cross-sectional design (n = 156). This study found no significant ERN/CRN differences between participants with obsessive-compulsive disorder, social anxiety disorder, or specific phobia, as well as control participants. However, after reclassifying the sample, study 2 found trend-level significant larger ERN/CRN amplitudes in clinical compared to nonclinical participants, as well as significant larger amplitudes for those with a family risk for internalizing symptoms. Additionally, joint analyses of study 1 and 2 (n = 246) confirmed the ERN/CRN link with trait worry for women. Finally, the prospective study 3 addressed the pathways from an enhanced ERN/CRN to the development of internalizing psychopathology by employing mediation analyses during the COVID-19 pandemic (n = 113). Results indicated that an increased pre-pandemic ERN/CRN were indirectly linked to anxiety, obsessive-compulsive, and depressive symptoms during the pandemic, mediated by a heightened COVID-19 risk perception and increased stress.

As a synthesis of the available literature and the current results, this dissertation proposes an integrative model of error-related brain activity and psychopathology. This model identifies an altered ERN as a neural vulnerability marker linked to both the internalizing and externalizing spectrum through latent dimensions underlying multiple clinical phenotypes, such as trait worry for anxiety- and obsessive-compulsive symptoms or impulsivity for substance use and hyperactivity. Moreover, it considers potential moderators and mediators of these associations, such as biological sex, temperament, adverse life events, and interpersonal stress. However, given the heterogeneous CRN literature, it seems premature to incorporate an altered CRN as an additional model component. Nevertheless, the model offers a neuroscientifically informed framework with the potential to not only guide future research but also to facilitate the development of targeted interventions based on knowledge from brain potentials, ultimately aimed at preventing mental health impairments and reducing the risk of psychopathological conditions.

German Abstract

Fehler sind ein allgegenwärtiger Bestandteil menschlicher Erfahrungen, die sowohl mit potenziellen Risiken als auch Möglichkeiten zur Verbesserung zukünftiger Handlungen verbunden sind. Die neuronale Handlungsüberwachung kann durch ereigniskorrelierte Hirnpotenziale (EKPs) gemessen werden, insbesondere durch die *error-related negativity* (ERN) und *correct-response negativity* (CRN). Meta-Analysen deuten darauf hin, dass eine veränderte ERN ein stabiler Risikomarker für internalisierenden und externalisierenden Störungen widerspiegelt, wobei erhöhte Amplituden bei Angst- und Zwangsstörungen gemessen werden konnten. Diese Assoziation ist jedoch nicht bei allen Störungsgruppen innerhalb dieses Spektrums beobachtbar, was auf zugrundeliegende transdiagnostische Faktoren (z.B. Sorgeneigung) zurückgeführt wurde. Da allerdings auch intraindividuelle Veränderungen der ERN in Abhängigkeit von Symptomausprägungen (z.B. situative Sorgen) gefunden wurden, stellt dies die vorrangig angenommene Stabilität der Zusammenhänge mit der ERN in Frage. Darüber hinaus ist das Verständnis darüber, wie eine erhöhte ERN zu psychopathologischen Symptomen führt, noch begrenzt, und es mangelt an Studien, die gleichzeitig mehrere Störungsgruppen und EKPs untersuchen. Aus diesem Grund möchte diese Dissertation die Zusammenhänge von elektrophysiologischen Korrelaten der Handlungsüberwachung im Angst- und Zwangsspektrum besser verstehen, indem ein transdiagnostischer und dimensionaler Ansatz gewählt wird, der verschiedene diagnostische Gruppen und EKPs einbezieht.

Die Zusammenhänge zwischen der ERN/CRN mit Sorgeneigung und situativen Sorgen untersuchend, zeigte Studie 1 (n = 90) unter Verwendung eines präregistrierten, randomisiert-kontrollierten Längsschnittdesigns, dass keine intraindividuellen Veränderungen der ERN/CRN aufgrund der experimentellen Manipulation von situativen Sorgen messbar waren. Stattdessen waren eine erhöhte Sorgeneigung mit einer größeren ERN/CRN verbunden, insbesondere bei Frauen. Studie 2 untersuchte die ERN/CRN als Risikomarker für Angst- und Zwangsstörungen unter Verwendung eines präregistrierten Querschnittdesigns (n = 156). Diese Studie fand keine signifikanten Unterschiede in der ERN/CRN zwischen Teilnehmenden mit Zwangsstörung, sozialer Angststörung, spezifischer Phobie sowie der Kontrollgruppe. Die nochmalige Aufteilung in klinische und nicht klinische Teilnehmende hinsichtlich einer internalisierenden Störung, jeweils mit oder ohne familiäres Risiko für eine internalisierende Störung, zeigte trend-signifikante größere ERN-/CRN-Amplituden bei klinischen Teilnehmenden sowie signifikant größere

Amplituden bei Teilnehmende mit familiärem Risiko. Darüber hinaus bestätigten zusätzliche Analysen in den kombinierten Stichproben von Studie 1 und 2 (n = 246) den Zusammenhang von erhöhter Sorgeneigung bei Frauen mit einer jeweils erhöhten ERN/CRN. Schließlich beschäftigte sich Studie 3 mit der Rolle einer erhöhten ERN/CRN bei der Entwicklung von internalisierender Symptomen unter Verwendung von Mediationsnalysen in einem prospektiven Design während der COVID-19-Pandemie (n = 113). Die Ergebnisse zeigten, dass eine erhöhte ERN/CRN vor der Pandemie indirekt mit stärkeren Angst-, Zwangs- und Depressionssymptomen während der Pandemie verbunden waren. Dieser Effekt wurde durch ein erhöhtes Risikoempfinden im Zusammenhang mit einer Corona-Infektion und einem höheren Stresslevel vermittelt.

Als Zusammenfassung der verfügbaren Literatur und der aktuellen Ergebnisse schlägt diese Dissertation ein integratives Modell der fehlerbezogenen Gehirnaktivität und Psychopathologie vor. Dieses Modell beschreibt eine veränderte ERN als einen neuralen Vulnerabilitätsmarker, der sowohl mit internalisierenden als auch externalisierenden Störungen über latente Dimensionen in Verbindung steht, die jeweils mehreren klinische Phänotypen zugrunde liegen, wie beispielsweise Sorgeneigung bei Angst- und Zwangssymptomen oder Impulsivität bei Substanzkonsum und Hyperaktivität. Darüber hinaus berücksichtigt das Modell potenzielle Moderatoren und Mediatoren dieser Zusammenhänge, wie beispielsweise Geschlecht, Temperament, kritische Lebensereignisse und zwischenmenschlichen Stress. Angesichts der heterogenen CRN-Literatur scheint es jedoch noch verfrüht, eine veränderte CRN als zusätzliche Modellkomponente einzubeziehen. Nichtsdestotrotz bietet das Modell einen neurowissenschaftlich informierten Rahmen, der nicht nur zukünftige Forschungsarbeiten leiten, sondern auch basierend auf dem bisherigen Wissen über Hirnpotentiale die Entwicklung gezielter Interventionen unterstützen kann, die letztlich darauf abzielen, Beeinträchtigungen der psychischen Gesundheit zu verhindern sowie das Risiko für eine psychopathologische Symptomatik zu reduzieren.

1. Theoretical Background

1.1 On the Role of Errors in Human Cognition and Emotion

1.1.1 A Common Human Experience

Making a mistake is a common and ubiquitous human experience. However, despite its commonality, most people find it neither pleasant nor desirable. Thus, it is unsurprising that many of us may not readily embrace or express enthusiasm for enjoying the act of committing errors, even though human learning is firmly rooted in trial-and-error approaches. An obvious reason for this hesitation is the wide range of potential consequences associated with errors. For instance, crossing an intersection as a pedestrian without thoroughly checking for vehicles beforehand poses significant danger and potential harm, not only to oneself but also to others. However, it is crucial to recognize that errors, despite their potential for adverse outcomes, do not universally result in fatal consequences, as exemplified in the aforementioned scenario. In fact, errors serve as important resources for acquiring new information. For example, during childhood, misspelling words is inevitable before mastering the art of writing. Hence, errors are not inherently harmful or dangerous events; instead, they also provide opportunities for humans to constantly enhance their knowledge, skills, and abilities through cognitive, motivational, and behavioral adjustments (Botvinick et al., 2001; Cavanagh & Shackman, 2015; Simons, 2010; Weinberg et al., 2012).

1.1.2 Electrophysiological Indices of Performance Monitoring

A testament to the pivotal role of errors for human performance and its improvement is the extensive focus errors have received from neuroscientific research over the past three decades (Gehring et al., 2018). A large body of literature contributing to our understanding of what role errors play in human cognition and emotion has emerged from studies investigating error monitoring through electroencephalography (EEG). Specifically, an event-related potential (ERP) associated with error commission, initially termed error negativity (N_E; Falkenstein et al., 1991) and later coined error-related negativity (ERN; Gehring et al., 1993) has garnered substantial attention, amassing approximately 5000 citations for the original studies (Meyer, 2022). The ERN manifests as a sharp response-locked ERP reaching its maximum peak between 0 – 100 ms after erroneous responses at fronto-central sites and is most commonly elicited in experiments utilizing response-conflict tasks (e.g., Flanker, Go/No-Go, Stroop, or an antisaccade task), or probabilistic reward learning tasks (Pasion & Barbosa, 2019). An example of the flanker task, which was

primarily used in this dissertation and is one of the most commonly employed tasks to study the ERN (Weinberg, Dieterich, et al., 2015), is illustrated in Figure 1. The ERN is measurable across the lifespan encompassing individuals from as young as five years to as advanced as 80 years (Davies et al., 2004a, 2004b; Nieuwenhuis et al., 2002; Torpey et al., 2009) and shows high internal consistency across tasks (Larson et al., 2010; Olvet & Hajcak, 2009b; Riesel et al., 2013) with good test-retest reliability over an interval of two to six weeks (Larson et al., 2010; Olvet & Hajcak, 2009a; Segalowitz et al., 2010) and even up to two years (Weinberg & Hajcak, 2011), demonstrating its utility as a trait-like marker.

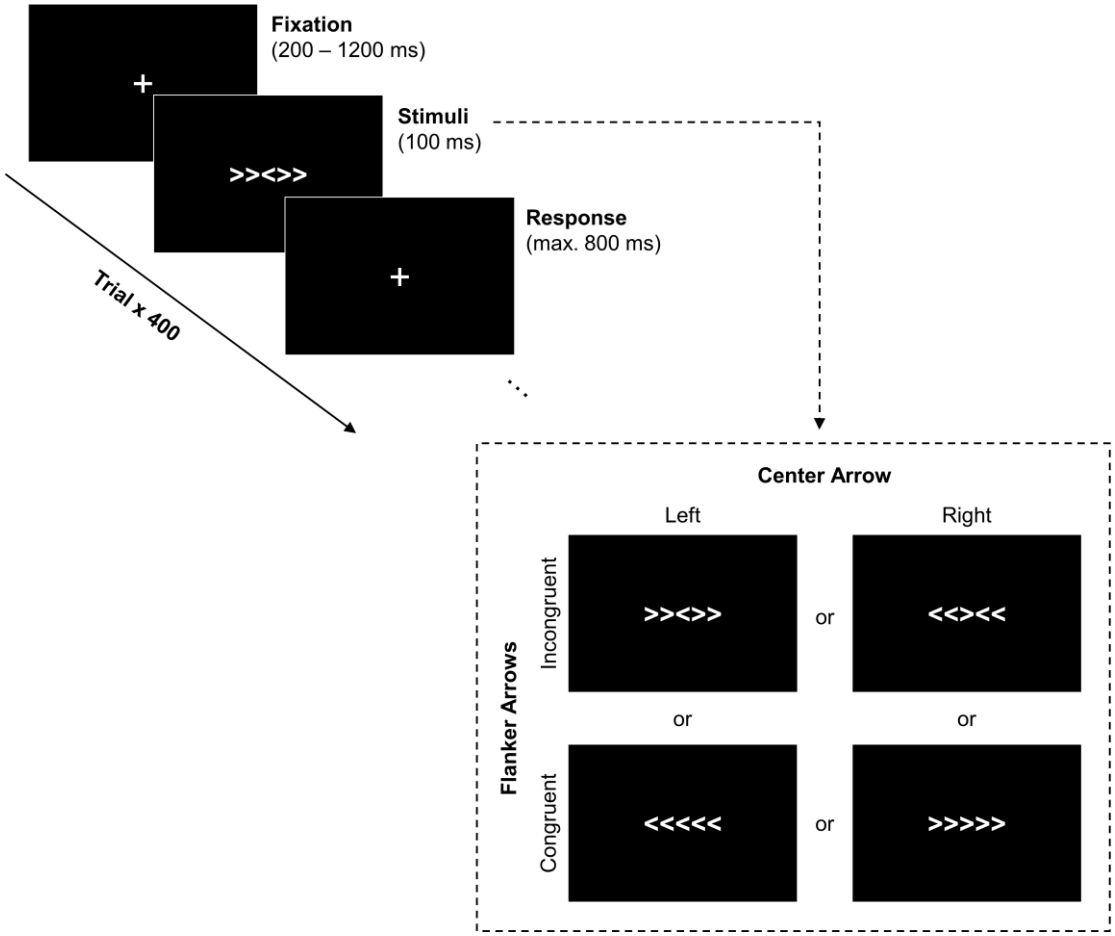

Figure 1. Modified flanker task consisting of an inter-trial interval with a fixation cross, the presentation of stimuli including index and flanker arrowheads, and a response window. Participants were instructed to indicate the direction of the center arrow by pressing a key with their respective left or right index finger as quickly and accurately as possible.

Within a similar time window and with a comparable topographical distribution of the ERN, another ERP is also elicited after correct responses, but with a relatively smaller amplitude. This ERP is commonly referred to as the correct-response negativity (CRN; Vidal et al., 2003; Vidal et al., 2000) and has received significantly less attention in previous research compared with the ERN (Michael et al., 2021). This is likely, at least in part, a result of different quantification approaches employed in past studies where an ΔERN was assessed as the difference or residualized score of the ERPs elicited by erroneous and correct responses. Together, the ERN and CRN represent two of the most commonly investigated neural indices of the cognitive system, monitoring human performance through the assessment of erroneous and correct responses (Figure 2).

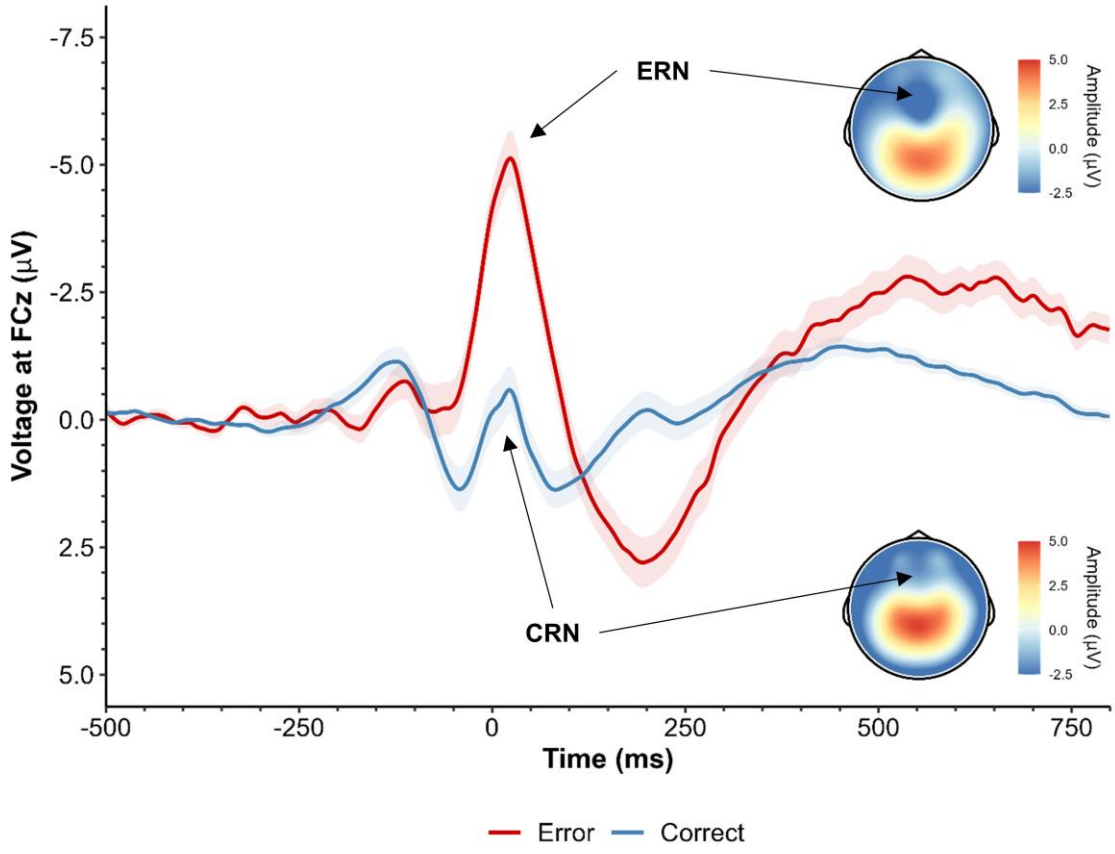

Figure 2. Exemplary illustration of response-locked grand-averaged waveforms at electrode FCz during erroneous and correct trials of a modified flanker task. Response was given at 0 ms by pressing one of two predefined keys referring to the direction of the center arrowhead. Corresponding topographic head maps of the ERN and CRN (mean activity 0 – 100 ms). ERN = error-related negativity; CRN = correct-response negativity.

A third ERP associated with error processing is the error positivity (Pe, Falkenstein et al., 2000). The Pe is characterized by a positive deflection subsequent to the ERN and can be divided into two potentially distinct subcomponents reflecting partially dissociable features (Endrass et al., 2007; Overbeek et al., 2005): an early, more fronto-central component (200 – 400 ms) and a late, more centro-parietal component (400 – 600 ms, Figure 3). Although the precise role of the Pe is generally less understood and remains subject of ongoing debate compared with the converging theoretical ground of ERN literature, the early Pe seems to be more closely linked to the ERN, while the late Pe has often been described to reflect error awareness or conscious error processing instead of the detection of errors (see Wessel, 2012, for a review).

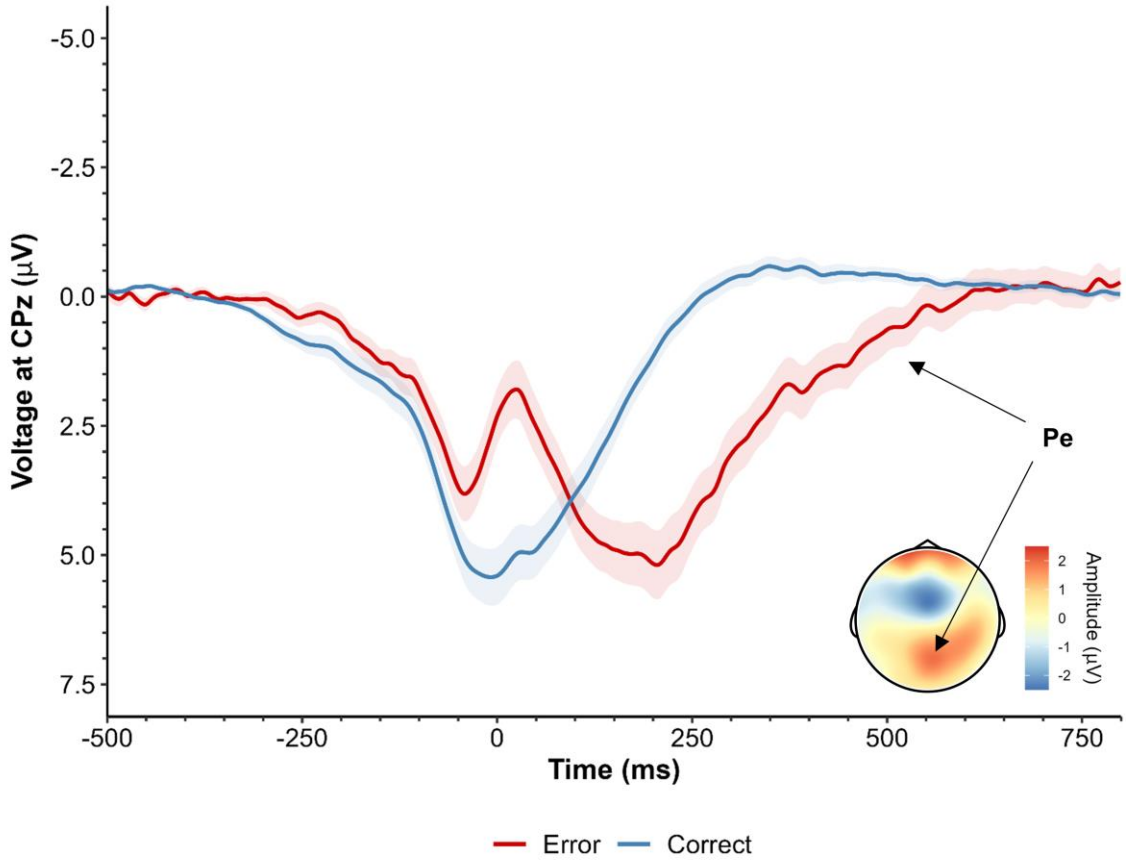

Figure 3. Exemplary illustration of response-locked grand-averaged waveforms at electrode CPz during erroneous and correct trials of a modified flanker task. Response was given at 0 ms by pressing one of two predefined keys referring to the direction of the center arrowhead. Corresponding topographic head map of the Pe (mean activity 400 – 600 ms). Pe = error positivity.

1.1.3 Functional Significance of Error Monitoring

The prolonged and ongoing neuroscientific research has given rise to an extensive body of theoretically and empirically grounded frameworks, all aimed at elucidating the functional role of the ERN within the cognitive system. The initial conceptualization, termed mismatch theory, described the ERN as a result of the divergence between the mental representations of the required and the actual response (Falkenstein et al., 1991; Gehring et al., 1993). Subsequently, conflict theory refined this perspective by proposing that the ERN arises from the conflict between two simultaneously activated response tendencies (Botvinick et al., 2001; Yeung et al., 2004). A third approach, the reinforcement learning theory, builds on the assumption that the ERN reflects alterations in dopamine activity whenever an outcome deviates from what was initially predicted (Holroyd & Coles, 2002). Finally, another approach underscores the role of motivation and emotion, suggesting that the magnitude of the ERN is linked to the individual significance and the potential consequences of committing an error (Hajcak, 2012; Hajcak & Foti, 2008; Proudfit et al., 2013). This approach is based on the observation that the ERN, for instance, is typically larger when errors are punished, making them more aversive or even threatening (Meyer & Gawlowska, 2017; Riesel, Kathmann, Wüllhorst, et al., 2019; Riesel et al., 2012). Despite their different underlying assumptions, all of these approaches acknowledge the crucial role of the ERN in cognitive control functions that are relevant in the processing of motivationally salient events. They propose that the ERN signals the need for flexible adjustments to meet the demands of a changing environment, serving as the starting point for a potential cascade of regulating processes that ultimately culminate in improved future behavior and performance (Weinberg, Dieterich, et al., 2015).

1.1.4 Neural Generators of the Error-Related Brain Activity

The frameworks discussing the functional role of the ERN have been informed and enriched by neurobiological findings that examine its neural foundations. Accumulating evidence consistently identifies the anterior cingulate cortex (ACC) as a principal neural generator of the ERN (Carter et al., 1998; Dehaene et al., 1994) with some more finely scaled studies inconsistently emphasizing the particular involvement of either the dorsal (Gilbertson et al., 2021; Hochman et al., 2014; Pourtois et al., 2010; Taylor et al., 2007), caudal (Brázdil et al., 2005; O'Connell et al., 2007; Ursu et al., 2009; van Boxtel et al., 2005), or rostral subdivision of the ACC (Brázdil et al., 2005; Hopfinger et al., 2000; Maier et al., 2015). These findings stem from a variety of methodological approaches, including

intracerebral single-unit recordings in primates (Godlove et al., 2011) and humans (Brázdil et al., 2005), measurements of local field potentials (LFP; Emeric et al., 2008; Pourtois et al., 2010), functional magnetic resonance imaging (fMRI; Carter et al., 1998; Ridderinkhof et al., 2004; Ursu et al., 2009), source localization in both electro- (EEG; Dehaene et al., 1994; Hochman et al., 2014; O'Connell et al., 2007; van Boxtel et al., 2005) and magnetoencephalographic studies (MEG; Miltner et al., 2003), as well as from the combination of neuroscientific methods (i.e., fMRI and EEG; Debener et al., 2005; Edwards et al., 2012; Gilbertson et al., 2021; Iannaccone et al., 2015; van Veen & Carter, 2002).

The ACC harbors a substantial density of dopaminergic neurons (Lumme et al., 2007; Olver et al., 2010), with both tonic and phasic dopamine levels modulating the magnitude of the ERN (de Bruijn et al., 2004; Meyer, Klein, et al., 2012; Zirnheld et al., 2004). Additionally, the ACC is widely connected to both “cognitive” prefrontal and “emotion processing” limbic regions (Bush et al., 2000; Tang et al., 2019). Consequently, error processing is not limited to ACC activity but is rather a product of a whole network of brain structures (Iannaccone et al., 2015). For instance, lesion studies found that patients with an impairment of the medial or lateral prefrontal cortex (Gehring & Knight, 2000; Maier et al., 2015; Stemmer et al., 2004; Swick et al., 2002; Ullsperger & von Cramon, 2006; Ullsperger et al., 2002), orbitofrontal cortex (Turken & Swick, 2008), or the basal ganglia (Ullsperger & von Cramon, 2006) show attenuated ERN amplitudes compared with controls. Moreover, experimental studies with functional (i.e., reversible) lesions of the medial frontal cortex after the application of transcranial magnetic stimulation (TMS) revealed corresponding results (Rollnik et al., 2004; but see Bellaïche et al., 2013). Similarly, transcranial direct current stimulation (tDCS) of the medial frontal cortex causes an in- or decrease in ERN amplitude depending on an anodal or cathodal stimulation (Reinhart & Woodman, 2014). Additionally, in line with the assumption of errors representing the risk for potential threat, activity of the cingulate cortex and the amygdala activity seem to be closely coupled during the processing of errors (Pourtois et al., 2010), suggesting the magnitude of the ERN as an indicator of an individual’s threat sensitivity (Hajcak & Foti, 2008; Heffer & Willoughby, 2021; Proudfit et al., 2013; Weinberg et al., 2016; Weinberg et al., 2012). Lastly, the ACC is not only involved in the processing of errors, but also activated during experiences of fear (Mechias et al., 2010) and pain (Bushnell et al., 2013; Peyron et al., 2000). Based on the aforementioned neurobiological

findings, the ACC, as the main generator of the ERN, is seen as an integrative hub within a network of brain structures that process cognitive and emotional information (Bush et al., 2000; Cavanagh & Shackman, 2015; Shackman et al., 2011; Shenhav et al., 2013), aiming at regulating human behavior in a volatile environment (Weinberg, Dieterich, et al., 2015).

1.1.5 Development and Interindividual Differences of Error-Related Negativity

Following the principles of structural and functional brain maturation, error-related brain activity undergoes normative developmental changes throughout the lifespan (Tamnes et al., 2013). While meta-analytical evidence indicates an increase of ERN magnitude during childhood and adolescence, eventually plateauing in adulthood (Boen et al., 2022), there are inconsistent findings regarding whether the ERN progressively decreases in older adults (Colino et al., 2017; Falkenstein et al., 2001; Hoffmann & Falkenstein, 2011; Nieuwenhuis et al., 2002; Schreiber et al., 2011) or remains stable (Eppinger et al., 2008; Larson et al., 2016; Niessen et al., 2017). In addition to a normative intraindividual development across the lifespan, various shaping factors during childhood, adolescence, and early adulthood contribute to stable trait-like interindividual differences of the ERN, which can be ordered on a continuum from features of an individual (micro level) to the surrounding environment of an individual (macro level).

On a micro level, genetics emerge as significant factors, as evidenced by an influential study comparing monozygotic and dizygotic twins in a large sample of 584 participants, which indicated heritability estimates ranging from 21% to 67% of ERN variance (Anokhin et al., 2008). Moreover, a medium-sized correlation (r = .28) between maternal and offspring ERN has been observed in 117 biological mother-child dyads (Suor et al., 2022) suggesting that a substantial portion of an individual's ERN may be determined by genetic predisposition, an observation just recently replicated (Trayvick et al., 2024). Additionally, biological sex or gender[1] seem to shape interindividual ERN differences, with males exhibiting larger ERN amplitudes compared with females (Fischer et al., 2016; Hill et al., 2018; Imburgio et al., 2020; Larson et al., 2011). The precise mechanisms underlying these sex differences have not yet been fully elucidated. Nonetheless, preliminary evidence suggests that sex hormones may play a mediating role (Gorday & Meyer, 2018).

[1] It is important to note that previous research did not accurately differentiate between an individual's biological sex and their gender identity.

Furthermore, on a meso level, cognitive capacity has been identified as being linked to error-related brain activity in both pediatric and adult populations (Meyer & Hajcak, 2019). Specifically, greater performance in domains such as working memory (Coleman et al., 2018; Miller et al., 2012; Weaver et al., 2017), attention/executive functions (Larson & Clayson, 2011), as well as attentional focusing/shifting and inhibitory control (Meyer & Klein, 2018; but see Brooker et al., 2011) has been associated with an increased ERN. Another influential factor shaping the ERN is temperament. For instance, both cross-sectional and prospective studies have demonstrated associations between shyness (Meyer & Klein, 2018; but see Brooker et al., 2011), behavioral inhibition (Buzzell et al., 2017; Lahat et al., 2014; McDermott et al., 2009), and fearfulness (Brooker & Buss, 2014a, 2014b; Meyer, Hajcak, et al., 2018) with increased ERN amplitudes, respectively. In contrast, impulsivity (Taylor et al., 2018), risk-taking behavior, and sensation-seeking tendencies (Santesso & Segalowitz, 2009) have been associated with smaller ERN amplitudes.

Lastly, on a macro level, the psychosocial environment has a significant impact on the development of the ERN in children and adolescents. On the one hand, children faced with early deprivation, neglect, and institutionalization are seemingly characterized by a blunted ERN (Debnath et al., 2023; Loman et al., 2013; Troller-Renfree et al., 2016; but see McDermott et al., 2013). On the other hand, an authoritarian and punitive parenting style characterized by high control and sanctioning (Chong et al., 2020; Meyer et al., 2019; Meyer, Proudfit, et al., 2015; Meyer & Wissemann, 2020; Suor et al., 2022), as well as adverse life events during childhood and adolescence, such as interpersonal conflicts, the loss of a parent, physical violence, or sexual abuse (Compton et al., 2024; Mehra et al., 2022), likely contribute to higher levels of ERN amplitudes.

Based on these findings, interindividual differences of the ERN are thought to be indicative of the individual's trait-like responsiveness to the demands of their environment along the lines of threat sensitivity and impulsivity (Heffer & Willoughby, 2021). A larger trait-like ERN is seemingly associated with an individual's heightened sensitivity to potential threats (Hajcak & Foti, 2008; Heffer & Willoughby, 2021; Proudfit et al., 2013; Weinberg et al., 2016; Weinberg et al., 2012), while a consistently diminished ERN is rather linked to a tendency towards impulsivity (Heffer & Willoughby, 2021; Hill et al., 2016;

Overmeyer et al., 2021; Ruchsow et al., 2005; Taylor et al., 2018) and a greater tolerance towards risk-taking behaviors (Santesso & Segalowitz, 2009; Zheng et al., 2014).

1.1.6 Interim Summary of Neural Indices of Error Monitoring

Committing an error is a universal aspect of the human experience, often accompanied by discomfort. Although errors can be potentially dangerous and harmful, they are important sources for human learning and future performance enhancement. Instances of errors are registered by the brain, as signaled by the error-related negativity (ERN) – a response-locked event-related potential (ERP) likely originating from the anterior cingulate cortex (ACC). The ERN appears to exhibit trait-like characteristics, with stable interindividual differences believed to be formed during childhood, adolescence, and early adulthood by a multiverse of early shaping factors encompassing genetics, biological sex or gender, cognition, temperament, and the psychosocial environment. An elevated trait-like ERN has been proposed to signify an individual's heightened sensitivity to potential threats, whereas a consistently diminished ERN is seemingly associated with an individual's tendency towards impulsivity and risk-taking behaviors.

1.2 Linking Altered Error Monitoring to Psychopathology

1.2.1 The Anxiety- and Obsessive-Compulsive Spectrum

Among multiple shared features, anxiety disorders and obsessive-compulsive disorder (OCD) are notably characterized by an overestimation of danger and threat, accompanied by a strong emotional reactivity and avoidance motivation, persisting beyond developmentally appropriate periods (American Psychiatric Association, 2013). As a result, the fourth version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 2000) listed anxiety disorders and OCD within a singular chapter, reflecting their relatedness. However, in the current fifth version (DSM-5; American Psychiatric Association, 2013), OCD has been relocated to its own dedicated chapter alongside obsessive-compulsive related disorders, while the relatedness of anxiety disorders and OCD is still explicitly emphasized, as exemplified by the order of OCD directly following the anxiety disorders chapter. This transition has been the subject of intense debates regarding the nosological justification. Some authors argued in favor (Bartz & Hollander, 2006; Phillips et al., 2010) while others contended against the separation of these disorders into distinct chapters (Bienvenu et al., 2012; Stein et al., 2010; Storch et al., 2008). Concurrently, some authors concluded that further research is imperative for a final

decision (Hollander et al., 2008; Stein, 2008a, 2008b), or proposed to implement a broader category of anxiety and obsessive-compulsive spectrum disorders (Bienvenu et al., 2012; Phillips et al., 2010; Stein et al., 2010).

While individuals with anxiety disorders or OCD may encounter a diverse array of potentially triggering objects and situations, there exists a significant overlap in phenomenology and occurrence. Transdiagnostic symptom overlap of these disorders, as described by the DSM-5, include intrusive thoughts revolving around danger and threat, sensations of fear and anxiety coupled with physiological arousal, heightened muscle tension, and hypervigilance, as well as behavioral response patterns characterized by avoidance, reassuring, and repetition. Furthermore, high comorbidity rates among anxiety disorders and OCD might be a result of the etiological interrelatedness of these disorders and their corresponding symptoms (Kaufman & Charney, 2000; Kessler et al., 2005; Ruscio et al., 2010; Sharma et al., 2021), challenging the assumption of a categorical classification system, wherein psychopathology is conceptualized using strictly defined and distinct disorder boundaries, although there is substantial symptom overlap within and between chapters of the DSM-5 (Forbes et al., 2024). Additionally, disorders of the anxiety and obsessive-compulsive spectrum share multiple alterations with regard to genetics, brain structure, functional connectivity, as well as neurotransmitters (see Maron et al., 2018, for a review), further questioning the justification for the validity of distinct disorder categories from a neurobiological perspective.

Addressing the phenomenon of heterogeneity within and comorbidity among distinct disorders categories, the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017) suggests a dimensional framework. Leveraging a quantitative nosology that clusters clinical variables using factor analytical methods, this alternative classification conceptualizes psychopathology as transdiagnostic syndromes, grouping together related traits, components, and symptoms. HiTOP proposes overarching spectra (i.e., internalizing, thought disorder, detachment, externalizing), each comprising finely delineated subfactors, syndromes, symptoms, traits, and associated disorders. Considering the historical sequencing of anxiety disorders and OCD in past and present categorical classification systems, such as the DSM, alongside the observed overlap in symptoms and traits across disorder boundaries, as delineated in dimensional approaches like HiTOP, this dissertation will adopt a transdiagnostic perspective of related disorders on a continuum termed anxiety

and obsessive-compulsive spectrum. By adopting a transdiagnostic and dimensional perspective, it is possible to address the shortcomings of conventional categorical approaches, which have difficulties with accounting for shared neurobiological structures and processes, high rates of comorbidity, arbitrary diagnostic thresholds, overlapping criteria, and the inability to capture the heterogeneity within individual diagnoses (Eaton et al., 2023).

1.2.2 Cross-Sectional Associations of Error-Related Brain Activity

As an index along the lines of threat sensitivity and impulsivity, the ERN has demonstrated considerable clinical utility in unveiling the etiopathogenesis of psychopathology (Hajcak et al., 2019). Consequently, the ERN has been incorporated into the Research Domain Criteria Initiative (RDoC), which provides a framework focusing on the neurobiological mechanisms of mental health and disorders (Cuthbert, 2014; Insel et al., 2010; Kozak & Cuthbert, 2016). Given its function as a central hub intertwining cognition and emotion, the ERN is currently categorized as a physiological measure within two domains in the RDoC matrix: cognitive systems (cognitive control: performance monitoring) and negative valence systems (sustained threat). Positioned at the midpoint of the spectrum of units of analysis, spanning from cellular mechanisms to behavioral paradigms, the ERN embodies a neurobiological marker with significant promise for elucidating the pathways underlying psychopathological conditions.

Previous meta-analyses have indicated that alterations in error monitoring correspond to the continuum from internalizing to externalizing mental disorders (Lutz et al., 2021; Pasion & Barbosa, 2019), whereby internalizing disorders are characterized by inwardly directed symptoms while externalizing disorders by outwardly directed behaviors (Achenbach et al., 2016; Schoemaker et al., 2013; Yang et al., 2022). Internalizing disorders, such as anxiety disorders (Michael et al., 2021; Moser et al., 2013; Saunders & Inzlicht, 2020) as well as OCD (Bellato et al., 2021; Mathews et al., 2012; Riesel, 2019) have been consistently reported to be linked to enhanced ERN amplitudes. This has also been proposed for depression (Moran et al., 2017), although this relationship is characterized by considerable between-study heterogeneity and is likely confounded by publication bias. Additionally, a more recent meta-analysis has failed to replicate a link between depression and altered error monitoring (Pasion & Barbosa, 2019). Conversely, diminished ERN amplitudes have been observed in externalizing disorders, such as substance use disorder

(SUD; Fairbairn et al., 2021; Webber et al., 2024), attention deficit hyperactivity disorder (ADHD; Bellato et al., 2021; Geburek et al., 2013; but see Kaiser et al., 2020), psychopathy (Vallet et al., 2021), and psychosis (Martin et al., 2018). As the primary focus lies on the anxiety and obsessive-compulsive spectrum and to maintain clarity and rigor of this dissertation, readers interested in the literature concerning the externalizing spectrum are recommended to consult recent reviews in this field (Lutz et al., 2021; Pasion & Barbosa, 2019).

While there is convincing meta-analytical evidence for increased error monitoring across the anxiety and obsessive-compulsive spectrum, not all corresponding disorder categories show this characteristic. Generalized anxiety disorder (GAD; Meyer, Weinberg, et al., 2012; Weinberg et al., 2010), social anxiety disorder (SAD; Endrass et al., 2014; Kujawa et al., 2016), health anxiety (Riesel et al., 2017), and OCD (Bellato et al., 2021; Mathews et al., 2012; Riesel, 2019) have been associated with enhanced ERN amplitudes. However, specific phobia (Hajcak et al., 2003; Moser et al., 2005) and post-traumatic stress disorder (PTSD; Gorka et al., 2016; Rabinak et al., 2013) have not been linked to altered error monitoring in previous research. Regarding panic disorder, limited literature exists, with only one study, indicating an enhanced ERN in individuals primarily diagnosed with panic disorder (Valt et al., 2018), albeit the relatively small sample size warrants replication in future studies to confirm this relationship. Additionally, to the best of my knowledge, there is a lack of available research investigating potential alterations of error monitoring in agoraphobia. These heterogeneous findings underscore the necessity for further investigation into the specific associations between heightened neural error monitoring and psychopathological variables associated with the anxiety and obsessive-compulsive spectrum. It is also noteworthy that symptom severity and clinical status seem to play an important role for the link between increased error monitoring and anxiety and obsessive-compulsive symptomatology (Saunders & Inzlicht, 2020). In fact, investigations focusing on subclinical symptom levels have yielded less consistent findings regarding enhanced ERN amplitudes (Chen & Itier, 2024; Clayson et al., 2023; Härpfer et al., 2020; Seow et al., 2020).

1.2.3 Latent Underlying Transdiagnostic Dimensions

The heterogeneity of findings regarding altered error monitoring across various disorder categories within the anxiety and obsessive-compulsive spectrum have been

hypothesized to result from different latent symptom dimension profiles underlying these disorders (Cox et al., 2010; Krueger, 1999). Previous studies have suggested several potential candidates for these dimensions, including intolerance of uncertainty (Jackson et al., 2016; Jackson et al., 2015), anxious misery (Riesel et al., 2023), harm avoidance (Meyer et al., 2021; Riesel, Klawohn, et al., 2019), and checking behavior (Weinberg, Kotov, et al., 2015; Weinberg et al., 2016), among others. However, aligning with the transdiagnostic and dimensional perspectives provided by the RDoC and HiTOP frameworks, two anxiety dimensions have garnered particular attention in neuroscientific research: anxious apprehension and anxious arousal (see Sharp et al., 2015, for a review).

Anxious apprehension describes a cognitive facet of anxiety, characterized by an individual's tendency towards future-oriented worries, repetitive negative thoughts, and verbal rumination, often accompanied by negative affect (Andrews & Borkovec, 1988; Barlow, 1991). In contrast, anxious arousal represents a more physiological component of anxiety, wherein individuals respond to even mild stressors with heightened arousal and somatic tension (Watson et al., 1995). Since anxious apprehension reflects the propensity to experience an enduring pattern of state worry (Sharp et al., 2015), it is often used interchangeably with the term trait worry (Carsten et al., 2023; Rutherford et al., 2020; Saunders & Inzlicht, 2020). Therefore, this dissertation will consistently use ‘trait worry’ as a synonym for ‘anxious apprehension’, allowing for a clearer differentiation in the context of trait and state worry. Similarly, anxious arousal describes the trait-like tendency to experience an enduring pattern of state arousal. Thus, this dissertation will differentiate between trait and state arousal in a consistent manner. In other words, while the literature frequently employs the terms anxious apprehension and anxious arousal (Sharp et al., 2015), this dissertation will instead refer to trait and state worry, as well as trait and state arousal, to ensure clarity and precision regarding the respective constructs.

Beyond investigations of the ERN, research adopted a similar approach to explore the associations of these transdiagnostic dimensions (i.e., trait worry and trait arousal) with brain structures (e.g., Castagna et al., 2018), hemispheric differences in brain activity (e.g., Nitschke et al., 1999), functional connectivity (e.g., Burdwood et al., 2016), coupling of oscillations (e.g., Knyazev et al., 2006), and startle responses (e.g., Rutherford et al., 2020). Within the ERN literature, the association between heightened neural error monitoring and the dimension of trait worry seems to be more pronounced than that with trait arousal. This

notion finds support in both primary studies (Hajcak et al., 2003; Lin et al., 2015; Moran et al., 2012; Moser et al., 2005; Moser et al., 2012; Weinberg et al., 2010; but see Härpfer et al., 2020; Macedo et al., 2021) and meta-analyses (Moser et al., 2013; Saunders & Inzlicht, 2020). Consequently, trait worry has been used as a post-hoc rationale to explain the observation that only some disorders of the anxiety and obsessive-compulsive spectrum are characterized by elevated ERN amplitudes. For instance, GAD (Meyer, Weinberg, et al., 2012; Weinberg et al., 2010), SAD (Endrass et al., 2014; Kujawa et al., 2016), and OCD (Bellato et al., 2021; Mathews et al., 2012; Riesel, 2019), which are presumably more strongly characterized by trait worry, seem to exhibit stronger associations with ERN enhancements compared to specific phobia (Hajcak et al., 2003; Moser et al., 2005) and PTSD (Gorka et al., 2016; Rabinak et al., 2013), where trait arousal might be more prominent. However, these findings are largely based on simple case-control studies that did not recruit transdiagnostic samples. Only a few studies have compared multiple disorder categories and/or a control group (Carrasco, Hong, et al., 2013; Endrass et al., 2014; Kujawa et al., 2016; Riesel et al., 2017; Weinberg, Kotov, et al., 2015). Therefore, comparing multiple groups is essential to test the transdiagnostic influence of factors such as trait worry.

Furthermore, some research suggests that the link between trait worry and enhanced error monitoring is more dominant in women (Lin et al., 2015; Moran et al., 2012; Moser et al., 2012), which was attributed to the regulatory impact of ovarian hormones on frontal brain regions associated with cognitive control (Moser et al., 2016). However, the specific underlying mechanisms of the more pronounced link in women compared with men remain to be thoroughly investigated (Russman Block et al., 2024). Nevertheless, a dimensional and transdiagnostic conceptualization of the anxiety and obsessive-compulsive spectrum by employing trait worry as a driving underlying dimensions, may provide a more comprehensive understanding of how heightened error monitoring is linked to anxiety and obsessive-compulsive symptoms, while broad and ostensibly distinct disorder categories may overshadow specific associations.

1.2.4 Translating Neural Vulnerability into Internalizing Psychopathology

As pointed out by the cross-sectional and transdiagnostic associations, altered error monitoring likely signifies an increased risk for psychopathology. This is further underscored by evidence indicating that not only individuals with clinically relevant

disorders show alterations in error monitoring, but also their unaffected (first-degree) relatives. For instance, individuals with a relative suffering from an anxiety disorder (Riesel, Klawohn, et al., 2019) or OCD (Carrasco, Harbin, et al., 2013; Riesel et al., 2011; Riesel, Klawohn, et al., 2019) are characterized by increased ERN amplitudes, while those with a relative affected by SUD (Euser et al., 2013; Riesel, Klawohn, et al., 2019), ADHD (McLoughlin et al., 2009), or psychosis (Simmonite et al., 2012) show a decreased ERN; despite not being diagnosed with the respective disorder themselves. These findings suggest that individuals with internalizing or externalizing disorders, along with their unaffected relatives, share a susceptibility to psychopathology as indicated by altered error-related brain activity. However, it remains to be shown by twin and adoption studies whether this susceptibility is due to genetic predisposition or attributable to shared environmental factors. Additionally, it remains an open question whether the alteration in the error monitoring system is even more pronounced in individuals who suffer from a clinical condition and have a familial predisposition, or if either factor alone is sufficient to alter the error monitoring system of this person.

Results from prospective studies further highlight the potential of altered error monitoring as a risk marker for psychopathology. For instance, healthy children and adolescents exhibiting larger error-related brain activity are more likely to develop an anxiety disorder within a period of three years (Meyer, Hajcak, et al., 2015; Meyer, Nelson, et al., 2018), although this prediction does not necessarily differentiate among specific types of anxiety disorders (Meyer, Hajcak, et al., 2015). However, the ERN can serve as a more fine-graded prognostic tool when evaluating anxious children and adolescents who have crossed the clinical threshold. These studies show that an increased ERN was prospectively linked to generalized and social anxiety symptoms (Lahat et al., 2014; Meyer et al., 2021), but found no association with other dimensions including panic or physical symptoms (Meyer et al., 2021). Consequently, the ERN might be less specific in identifying the particular type of disorder before its first manifestation. However, after disorder onset, the predictive utility becomes more nuanced, offering insights into the diverse symptom dimensions characterizing the disorder.

Only recently, studies have delved into the mechanisms translating neural vulnerability markers into clinical anxiety and its various facets, shedding light on relevant factors along these pathways. Past research, (e.g., Meyer, 2017; Weinberg et al., 2022)

frequently employed the diathesis-stress model, which depicts psychopathology (e.g., anxiety and obsessive-compulsive symptoms) as arising from the interplay between dispositional vulnerability (e.g., heightened ERN) and stressors (e.g., adverse life events). The diathesis-stress model might explain the multifinal observation, that on the one hand, enhanced error monitoring clearly signals risk for internalizing psychopathology (e.g., Riesel, Klawohn, et al., 2019; Saunders & Inzlicht, 2020), but on the other hand, not all individuals with an increased ERN inevitably develop a corresponding disorder (Carrasco, Harbin, et al., 2013; Riesel et al., 2011; Riesel, Klawohn, et al., 2019). To date, instances of adverse life events, such as interpersonal stress among first year university students (Banica et al., 2020) or a natural disaster like hurricane "Sandy" (Meyer et al., 2017) have been identified as moderators of the relationship between heightened error monitoring and internalizing symptoms. A mediating function on offspring internalizing psychopathology has been demonstrated for negative parenting style as a result of a larger maternal ERN (Suor et al., 2022). Moreover, besides stressful life events (i.e., environmental factors), other features of a person (i.e., individual factors), such as a irritability (Filippi et al., 2020; Kessel et al., 2016), behavioral inhibition (Filippi et al., 2020; Lahat et al., 2014; McDermott et al., 2009), sleep quality (Mehra et al., 2024), and biological sex (Moser et al., 2016), seemingly influence the link between enhanced error monitoring and internalizing psychopathology. Taken together, extending the diathesis-stress model by further moderating and mediating factors could provide an interesting framework for a more detailed understanding of the trajectories leading from neural vulnerability to internalizing psychopathology.

1.2.5 Interventions Targeting the Error-Related Negativity

In addition to cross-sectional studies employing correlational or case-control designs, as well as prospective observational studies, there are a number of randomized-controlled trials aimed at targeting the ERN through various interventions, ranging from less to more tailored approaches. Within rather less tailored but long-termed interventions, cognitive-behavioral therapy (CBT) has not been found to influence the ERN, despite the successful reduction of anxiety (Gorka et al., 2018; Kujawa et al., 2016; Ladouceur et al., 2018) and obsessive-compulsive symptoms (Hajcak et al., 2008; Riesel et al., 2015). This finding, consistent with results from family risk studies (Carrasco, Harbin, et al., 2013; Euser et al., 2013; McLoughlin et al., 2009; Riesel et al., 2011; Riesel, Klawohn, et al., 2019), suggests that the ERN might be independent of symptomatic changes. Nevertheless,

it remains uncertain whether these less tailored interventions are per se capable of decreasing heightened error monitoring and ultimately mitigating the risk of internalizing symptoms, as they were originally not designed to target the ERN.

Regarding more tailored but short-termed interventions, results appear more promising. A reduced ERN was measured after the employment of a one-hour computerized program reducing error sensitivity in undergraduate students (Meyer et al., 2020; but see Meyer et al., 2023), single or multiple short training sessions to shift attentional bias from negative to neutral stimuli in undergraduate students and OCD patients (Klawohn, Hajcak, et al., 2020; Nelson et al., 2015; Tan et al., 2021; but see Carlson et al., 2021), or an eight-minute emotional expressive writing paradigm aimed at reducing state worry in high worriers (Schroder et al., 2018). Notably, the latter investigation raises intriguing questions about a fundamental assumption concerning the stable trait-like nature of the ERN, suggesting its sensitivity to symptom fluctuations such as state worry. However, despite the utilization of a randomized controlled design, the fact that only post-measurements of the ERN were assessed, introduces the possibility of preexisting unmeasured baseline differences, potentially confounding the attributed reducing effects of the emotional expressive writing intervention. Nevertheless, given that the majority of existing literature reports cross-sectional associations between heightened ERN and trait worry, this study lays the foundation for future investigations aiming at causal inferences through the employment of an experimental research paradigm.

1.2.6 Error-Related Brain Activity as an Endophenotype

The previously presented extensive literature in the field of error-related brain activity and its links to psychopathology has accumulated in the proposal that an altered ERN may serve as a transdiagnostic endophenotype for various mental disorders (Manoach & Agam, 2013; Olvet & Hajcak, 2008; Riesel, Klawohn, et al., 2019). An endophenotype denotes a measurable component aimed at elucidating the complex processes spanning from genetic predisposition to the phenotypic manifestation of psychopathology (Gottesman & Gould, 2003). However, a neurobiological marker must meet specific criteria to qualify as an endophenotype (Gottesman & Gould, 2003).

A fundamental requirement is that the marker is linked to psychopathology at all. As described earlier, meta-analytical evidence clearly shows a link between an increased ERN and internalizing psychopathology, while a decreased ERN is associated with the

externalizing spectrum (Lutz et al., 2021; Pasion & Barbosa, 2019). Regarding the internalizing dimension, particularly disorders characterized by trait worry within the anxiety and obsessive-compulsive spectrum, such as GAD, SAD, and OCD, seem related to an enhanced ERN (Moser et al., 2013; Saunders & Inzlicht, 2020). Another criterion is that psychopathological conditions and the marker need to co-segregate within families and demonstrate increased prevalence among unaffected family members compared with the general population. This has been shown by family risk studies observing similar alterations of ERN amplitudes between individuals with anxiety disorders, OCD, SUD, and ADHD and their unaffected (first-degree) relatives compared with unrelated healthy control participants (Carrasco, Harbin, et al., 2013; Euser et al., 2013; McLoughlin et al., 2009; Riesel et al., 2011; Riesel, Klawohn, et al., 2019). The third criterion for an endophenotype is also fulfilled, such that the ERN is unaffected by symptomatic status, as evidenced by intervention studies wherein a successful reduction of anxiety (Gorka et al., 2018; Kujawa et al., 2016; Ladouceur et al., 2018) and obsessive-compulsive symptoms (Hajcak et al., 2008; Riesel et al., 2015) was not accompanied by alterations in ERN amplitudes. However, when symptomatic state is construed not as clinical status but as symptomatic fluctuations, preliminary findings indicate short-term alterations of the ERN following an experimental reduction in state worry (Schroder et al., 2018). Finally, a marker needs to be heritable, as evidenced in transgenerational studies (Anokhin et al., 2008; Suor et al., 2022; Trayvick et al., 2024). Taken together, there is compelling evidence for the assumption that altered error-related brain activity serves as endophenotype for disorders of the anxiety- and obsessive-compulsive spectrum, particularly for those characterized by trait worry.

1.2.7 Interim Summary of Error-Related Brain Activity in Clinical Research

Anxiety disorders and OCD share multiple clinical characteristics, likely forming a broad anxiety and obsessive-compulsive spectrum. Meta-analyses have consistently demonstrated cross-sectional associations of increased error-related brain activity in anxiety disorders and OCD. However, not all disorders within that spectrum, nor subclinical levels of symptoms, show a consistent association with increased ERN amplitudes. This was attributed to underlying transdiagnostic dimensions, wherein trait worry, particularly in women, correlates with the ERN. Serving as an endophenotype, heightened error-related brain activity predicts the onset of anxiety disorders in children and adolescence, indicating risk for internalizing psychopathology. While only a subset of individuals with an increased ERN progresses to a clinical disorder, environmental factors

(e.g., adverse life events) and individual features (e.g., biological sex or gender) appear to work as catalysts in translating latent risk into the development of psychopathology. Challenging the traditional view on the ERN as a stable trait-like characteristic, preliminary evidence suggests that fluctuations in state worry may influence variations in error-related brain activity.

Overall, the research field of altered error monitoring in disorders of the anxiety and obsessive-compulsive spectrum is primarily characterized by cross-sectional studies with case-control designs comparing a single clinical with one healthy control group. Following these delineations, several gaps in the literature can be identified which provide a vantage point for this dissertation. To mitigate the risk of overestimating effect sizes, studies should incorporate comparisons of multiple clinical groups and utilize a naturalistic, instead of a strictly defined healthy control group. This approach is also particularly important for examining the transdiagnostic influences on the ERN, which are likely shared across various disorder categories. In other words, study designs that jointly investigate multiple clinical groups are essential for accurately exploring the potentially transdiagnostic processes reflected in ERN variability. Another research gap is that most (cross-sectional) studies have focused on the link of the ERN and trait-like dimensions, such as trait worry, while its link to mental states associated with trait worry, such as state worry, has received comparatively less attention. Moreover, there is a scarcity of experimental or prospective study designs, which could better elucidate the mechanisms underlying the translation of neural risk markers into psychopathological conditions, as not all individuals with a risk for internalizing psychopathology, indexed by an increased ERN, develop corresponding clinical symptoms. Finally, while much of the literature centers on the ERN, the specificity of these findings warrants further investigation, as other related ERPs, such as the CRN and Pe, have been relatively understudied.

2. Research Objectives

2.1 Aim

The current dissertation aims to explore error-related brain activity within the framework of internalizing psychopathology, particularly focusing on its role as a pathophysiological risk marker of the anxiety and obsessive-compulsive spectrum. Specifically, the impact of trait worry as a potentially underlying transdiagnostic trait dimension, as well as its related states, is of primary interest. Additionally, the influence of clinical status and family risk is thoroughly examined. Finally, trajectories from enhanced neural error monitoring to the development of anxiety and obsessive-compulsive symptoms are investigated, while considering stress as a potential catalyst. To this end, this dissertation utilizes cross-sectional, experimental, and prospective studies that include large transdiagnostic and dimensional samples, encompassing participants with a wide range of psychopathological symptoms and severity across the anxiety and obsessive-compulsive spectrum, as well as including various brain potentials beyond error-related activity.

2.2 Research Questions and Hypotheses

The primary research questions of this dissertation, along with the corresponding hypotheses, are grounded in the existing literature on the role of error-related brain activity across the anxiety and obsessive-compulsive spectrum. For a better overview, these inquiries are categorized into three main topics: a) the susceptibility of error-related brain activity to intraindividual state influences, b) the investigation of factors associated with interindividual trait differences in error-related brain activity, and c) the identification of potential moderators and mediators of the link between heightened error-related brain activity and the development of anxiety and obsessive-compulsive symptoms. In order to assess the specificity of these effects, this dissertation includes exploratory analyses of additional brain potentials (i.e., CRN and Pe) without predefined hypotheses.

Question 1 (Study 1):
Is intraindividual variability in error-related brain activity influenced by mental states that are associated with trait worry? And if so, which role plays state worry?

Hypothesis 1: The experimental induction of state worry increases, while its reduction decreases error-related brain activity.

Question 2 (Study 2):

Which factors are linked to interindividual differences in error-related brain activity? What influence do transdiagnostic trait dimensions, clinical status, and family risk have?

Hypothesis 2a: Trait worry, but not trait arousal, is associated with enhanced error monitoring. This association is stronger for women compared with men.

Hypothesis 2b: Disorders such as OCD and SAD, which are presumably characterized by trait worry, exhibit greater error-related brain activity compared to specific phobia and control participants.

Hypothesis 2c: On a broader scale, enhanced error-related brain activity is linked not only to a clinical status of an internalizing disorder but also to the family risk for internalizing psychopathology.

Question 3 (Study 3):

How do error-related brain activity, stress, and internalizing symptoms interact with each other? Are adverse life events, such as the COVID-19 pandemic, catalysts for the development of internalizing symptoms in individuals with heightened error-related brain activity?

Hypothesis 3: Pandemic-related stress enhances the link between increased error-related brain activity and internalizing symptoms.

3. Summary of Studies

3.1 Study 1: Disentangling Trait and State Worry

Citation. Härpfer, K., Carsten, H. P., Löwisch, K., Westermann, N., & Riesel, A. (2022). Disentangling the effects of trait and state worry on error-related brain activity: Results from a randomized controlled trial using worry manipulations. *Psychophysiology, 59*(9), e14055. https://doi.org/10.1111/psyp.14055

Objective. As pointed out in the introduction, the ERN is commonly understood as trait-like component reflecting an individual's heightened sensitivity to potential threats (Hajcak & Foti, 2008; Heffer & Willoughby, 2021; Proudfit et al., 2013; Weinberg et al., 2016; Weinberg et al., 2012). However, some studies suggest intraindividual variability of the ERN; for instance, when accuracy is emphasized over speed (Falkenstein et al., 2000; Gehring et al., 1993; Riesel, Kathmann, & Klawohn, 2019), when errors are punished (Meyer & Gawlowska, 2017; Riesel, Kathmann, Wüllhorst, et al., 2019; Riesel et al., 2012) or socially evaluated (Buzzell et al., 2017; Voegler et al., 2018), during dual task demands (Klawohn et al., 2016), or with changing positive affect (Nigbur & Ullsperger, 2020) and negative affect (Wiswede, Münte, Goschke, et al., 2009; Wiswede, Münte, & Rüsseler, 2009). On the one hand, these primary studies suggest state-dependent variability of the ERN. On the other hand, meta-analytical evidence (Moser et al., 2013; Saunders & Inzlicht, 2020) indicates a link between a heigthened ERN and the anxiety dimension trait worry. Consequently, these observations raise the critical question of whether the ERN is also susceptible to experimental manipulations of state worry, thereby further supporting the role of trait worry and its related states in both inter- and intraindividual variability of the ERN.

In fact, preliminary experimental evidence suggests that an emotional expressive writing intervention, aimed at reducing state worry, can decrease the ERN of dispositionally worrying individuals (Schroder et al., 2018). However, this first study has some methodological limitations, as the authors measured the ERN only post-intervention, leaving the possibility open that unmeasured baseline differences may have influenced the observed effects. The intervention was also not accompanied by a significant decrease in self-reported anxiety, further questioning the assumed mechanism leading to a smaller ERN in the intervention group. Additionally, it would have been conceptually and epistemologically valuable to investigate the impact of experimentally increasing state

worry. Consequently, the first study of this dissertation examined both the cross-sectional associations between interindividually differing levels of trait worry and the ERN, as well as the causal impact of experimental manipulations of state worry on intraindividual ERN variability.

Method. The design and analyses of this randomized-controlled longitudinal study were preregistered prior to data collection (https://osf.io/j8k6z). The sample size was determined based on an a prior power analysis. Ninety unselected participants (n = 66 female) aged 18-30 years were randomly assigned and gender matched to either a worry induction, reduction, or passive control group (each n = 30) receiving a group-specific intervention for eight minutes. In order to assess and control for clinical data, all participants underwent a structured clinical interview for clinical diagnoses (SCID-5-CV; Beesdo-Baum et al., 2019; First et al., 2016) and completed a battery of questionnaires including the Penn State Worry Questionnaire (PSWQ; Glöckner-Rist & Rist, 2014; Meyer et al., 1990) as a measure for trait worry, which was of special interest to the current study.

At first, participants identified personally relevant topics that they currently worry about (Arch & Craske, 2006; Boehnke et al., 1998). Subsequently the induction group, following a catastrophizing approach (e.g., Borkovec & Inz, 1990; Verkuil et al., 2009), was instructed to intensively worry by contemplating worst-case scenarios related to their topics. Conversely, the reduction group, in line with the emotional expressive writing technique (e.g., Pennebaker, 2017; Ramirez & Beilock, 2011; Schroder et al., 2018), was encouraged to write down their topic-related thoughts, concerns, and feelings without self-judgment. The passive control group did not undergo any intervention. Manipulation checks throughout the study were implemented to track intraindividual fluctuations of state worry, state arousal, and state affect (assessed via a forced-choice visual analogue scale ranging from 0 to 100). ERPs were measured before and after the respective intervention using an arrowhead version of the flanker task with speed feedback. Statistical analyses involved χ2-tests, t-tests, one-way as well as mixed-measures analyses of variance (ANOVA), and multiple linear regression models, alongside corresponding Bayesian analyses.

Results. Randomization was successful, as indicated by the absence of baseline differences in demographic or clinical data. Manipulation checks revealed that both the induction and reduction groups reported significantly higher levels of state worry, state

arousal, and more negative state affect after the intervention compared to the control group (Figure 4), constituting large effect sizes of the experimental manipulations on state measures, particularly state worry. While the induction paradigm resulted in the anticipated effects, the emotional expressive writing intervention, did not decrease state worry as expected, but resulted in an increase. However, these changes in symptom states were not accompanied by group-specific alterations in ERPs (Figure 5), a finding further supported by Bayesian analyses. Instead, the CRN decreased over time across all groups. Consequently, neither the ERN nor CRN showed intraindividual variability as a result of the experimental induction of state worry.

In the cross-sectional analyses examining interindividual differences (trait worry) rather than intraindividual variability (state worry), findings revealed that elevated levels of trait worry were linked to a larger ERN in females (n = 66), while the effects for males were in the opposite direction (n = 24). Participants with higher levels of trait worry also showed a larger CRN, but this effect was not moderated by gender. Since a subset of participants fulfilled the criteria for a current or lifetime internalizing diagnosis, we compared ERPs of them (n = 14) with all other non-clinical participants (n = 69), finding enhanced amplitudes of large effect size for the ERN but not the CRN in the clinical group.

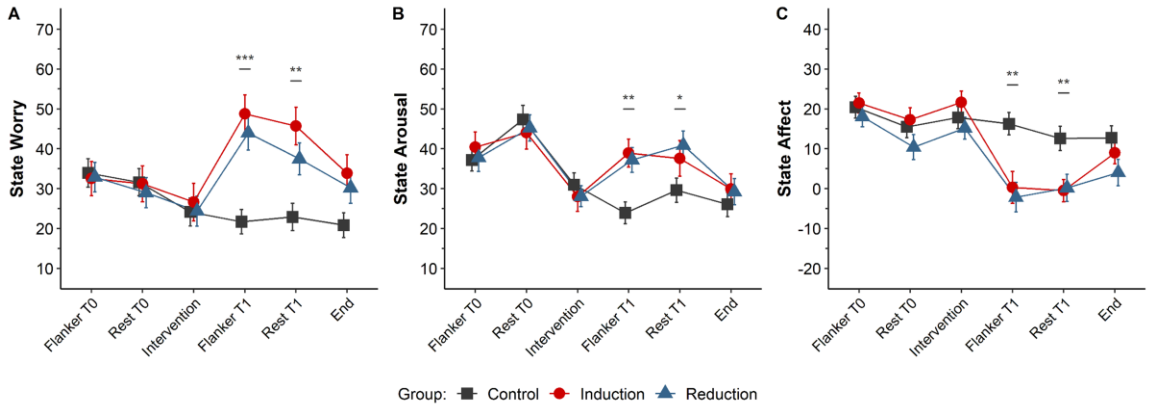

Figure 4. State measurements tracking fluctuations of state worry (A), state arousal (B), and state affect (C) throughout the study. Each scale consisted of two items rated on a visual analogue scale. For state worry and state arousal, the potential range was 0 to 100, for state affect it was -50 to 50. Error bars represent one standard error. Asterisks indicate significant differences between groups at each time point: *** $p < .001$. ** $p < .01$. * $p < .05$. $N = 90$. Figure from Härpfer et al. (2022).

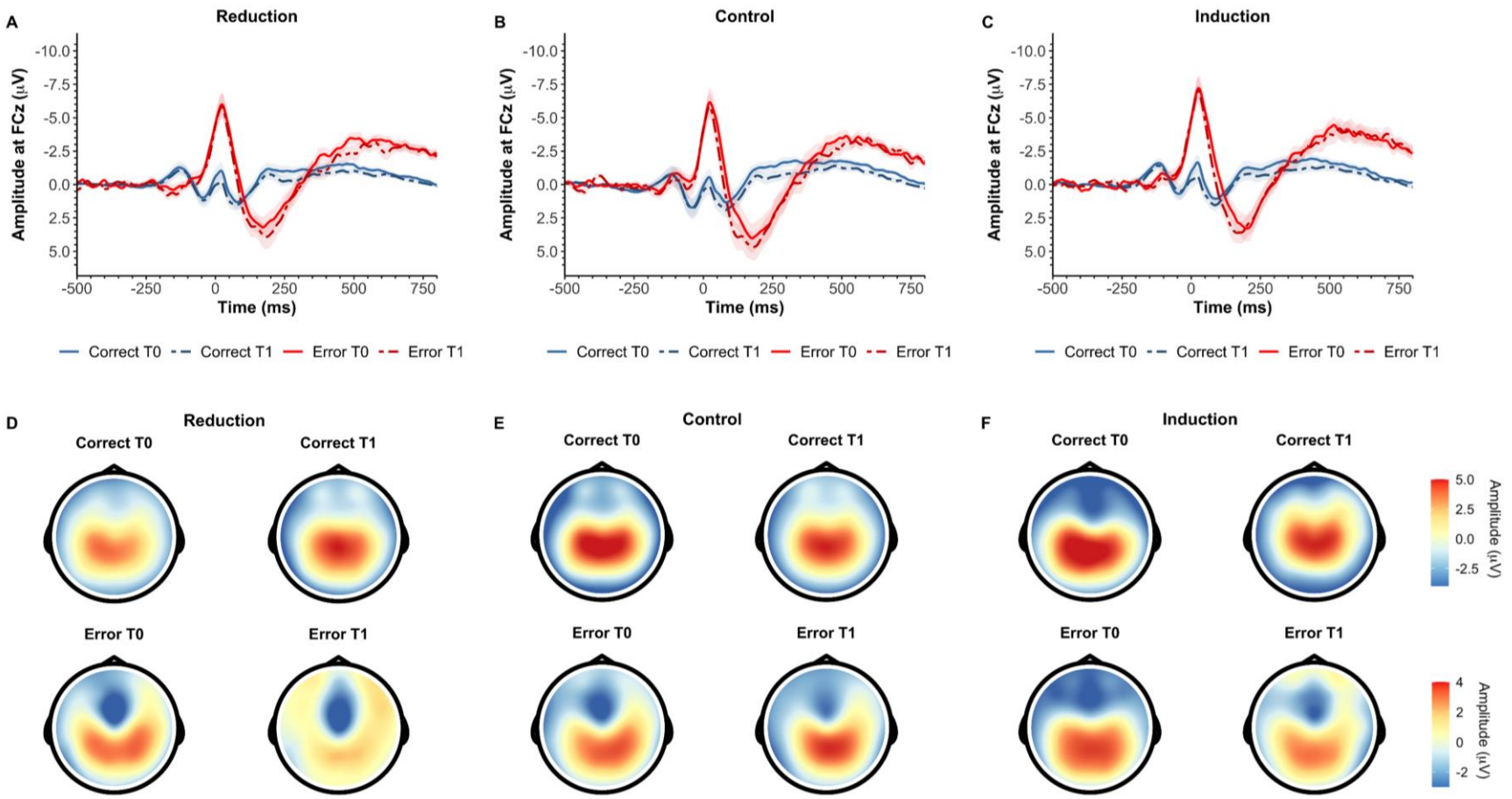

Figure 5. Response-locked grand-averaged waveforms of correct and erroneous trials at baseline (T0) and after the intervention (T1) for the worry reduction group (A), passive control group (B), and worry induction group (C). Corresponding topographic head maps of the ERN and CRN (mean activity 0 – 100 ms) for the worry reduction group (D), passive control group (E), and worry induction group (F). ERN = error-related negativity; CRN = correct-response negativity. $N = 90$. Figure from Härpfer et al. (2022).

Additional Analyses. Results of additional analyses, not included in the main publication, are detailed in Appendix A1. The first analysis (Table A1.1) aimed to explore the potential interaction between trait and state worry on ERN/CRN alterations before the first flanker task (T0) and after the interventions (T1). Separate multiple linear regression models were performed for the difference in ERN and CRN between T1 and T0. Predictors included the PSWQ (trait worry), the difference in state worry between T1 and T0 (state worry), and their interaction term. However, none of the predictors reached statistical significance. To test for the specificity of the main investigations, the second analysis (Table A1.2) explored potential effects of the interventions on the Pe. The results of the mixed-measures ANOVA with the between-subject factor group (induction, reduction, control) and the within-subject factor time (T0, T1) did not reveal significant effects on the Pe. The third analysis (Table A1.3) investigated the influence of gender and trait worry on the Pe, finding larger Pe amplitudes in men compared with women. However, trait worry was not a significant predictor. Finally, clinical and non-clinical participants did no differ regarding the Pe, $t(81) = -0.27$, $p = .788$, $d = -0.08$.

Discussion. The results of the first study of this dissertation suggest that neither the ERN nor CRN appear to be influenced by experimentally induced intraindividual changes in state worry. It is essential to note that a final conclusion regarding the impact of the state worry reduction intervention cannot be made, as the reduction group unexpectedly reported higher levels of state worry following the emotional expressive writing intervention. This is in contrast to the initial evidence of the first study, which showed a reduced ERN in the emotional expressive writing group (Schroder et al., 2018). Nevertheless, prior treatment studies also indicated that the ERN is seemingly insensitive to intraindividual symptomatic reductions in anxiety (Gorka et al., 2018; Kujawa et al., 2016; Ladouceur et al., 2018) and obsessive-compulsive symptoms (Hajcak et al., 2008; Riesel et al., 2015). Regarding the CRN results of the current study, amplitudes decreased over time across all three groups, without differential effects between the groups. This decrease may reflect either training effects or task disengagement. Additionally, no effects were found for the Pe.

The divergent results between the original study (Schroder et al., 2018) and this study, which sought to replicate and extend the previous findings, may stem from crucial differences in research designs. The original study employed a state worry reduction paradigm with chronically worrying individuals, while this study used an unselected

sample to examine the effects of both a state worry induction and reduction intervention. It is conceivable that a state worry reduction paradigm effectively reduces error-related brain activity only in individuals with high levels of trait worry. However, this assumption about the underlying mechanism in the initial study must be questioned for two main reasons: First, the intervention did not reduce self-reported state anxiety, the measure used as a manipulation check, from pre- to post-intervention, as one would expect. Second, the ERN was assessed only once after the intervention, questioning the assumed reducing effect as this result might just only originate from potentially preexisting but not assessed baseline differences between the experimental groups. Addressing these methodological considerations, the current study meticulously tracked the influence of both interventions on intraindividual changes in symptom states while also measuring ERPs before and after the intervention, providing a more detailed and nuanced understanding. Furthermore, supplementary analysis did not reveal any significant interaction between state and trait worry on ERP magnitudes. This finding speaks against the possibility that the state worry interventions differentially affected participants with varying levels of trait worry.

In contrast to intraindividual variations in state worry, interindividual differences in the propensity to worry (i.e., trait worry) demonstrated greater explanatory power for variability in ERN and CRN. Specifically, heightened levels of trait worry were found to be associated with larger ERN amplitudes in females, which is in line with meta-analytical evidence suggesting that women show a stronger associations between anxiety and error-related brain activity (Moser et al., 2016). Conversely, this association was the opposite direction among males, although the limited number of male participants necessitates further replication to confirm this finding. Regardless of gender, the results also suggest an association between trait worry and CRN magnitude, thereby extending the current understanding (Härpfer et al., 2020; Lin et al., 2015; Moran et al., 2012; Moser et al., 2012) to encompass not only error monitoring but also performance monitoring in general to be linked to trait worry. Moreover, this study strengthens the existing body of evidence indicating that mental disorders within the internalizing spectrum are characterized by heightened ERN amplitudes (Pasion & Barbosa, 2019; Riesel, 2019; Saunders & Inzlicht, 2020), and that this finding is specific to the ERN, as the CRN and Pe analyses did not reveal similar results.

Conclusion. Taken together, the first study of this dissertation underscores the notion that variations in ERN and CRN predominantly reflect interindividual (i.e., trait-like) differences in the tendency to worry rather than intraindividual (i.e., state-like) changes in situational worrying. Additionally, gender appears to act as a moderator for the link between trait worry and the ERN, but not the CRN. Consistent with prior investigations, this study also found further evidence for the assumption that increased ERN and CRN amplitudes reflect the risk for a clinical disorder of the internalizing spectrum. A critical limitation, however, is that this study examined a mostly nonclinical sample and the emotional expressive writing paradigm did not result in a reduction of state worry.

3.2 Study 2: Enhanced Performance Monitoring as a Transdiagnostic Risk Marker

Citation. Härpfer, K., Carsten, H. P., Kausche, F. M., & Riesel, A. (in revision). Enhanced performance monitoring as a transdiagnostic risk marker of the anxiety and obsessive-compulsive spectrum: The role of disorder category, clinical status, family risk, and anxiety dimensions. *Depression and Anxiety*.

Objective. While meta-analytical evidence compellingly supports the notion that an increased ERN serves a risk marker for disorders of the anxiety and obsessive-compulsive spectrum (e.g., OCD, SAD; Moser et al., 2013; Riesel, 2019; Saunders & Inzlicht, 2020), this pattern may not extend uniformly across all disorder categories within this spectrum (e.g., specific phobia; Hajcak et al., 2003; Moser et al., 2005). Inconsistencies in findings between disorder categories may originate from variations in latent underlying symptom dimension profiles (Cox et al., 2010; Krueger, 1999 Sharp et al., 2015), such as transdiagnostic anxiety dimensions (i.e., trait worry and trait arousal). This underscores the necessity for a refined conceptualization of how ERN alterations relate to specific clinical phenotypes within the broader spectrum of internalizing disorders (Riesel et al., 2023; Weinberg et al., 2016; Weinberg et al., 2012). Furthermore, studies investigating subclinical symptom levels have yielded less consistent results regarding heightened ERN amplitudes (Clayson et al., 2023; Härpfer et al., 2020; Seow et al., 2020), suggesting that clinical status may serve as a potential moderator (Saunders & Inzlicht, 2020). Lastly, the simultaneous examination of clinical status and family risk for internalizing psychopathology (Carrasco, Harbin, et al., 2013; Riesel et al., 2011; Riesel, Klawohn, et al., 2019), on an elevated ERN has not yet been explored. Consequently, this second study consisting of a transdiagnostic clinical sample aimed at investigating the interindividual differences of the ERN by

focusing on the role of disorder category, clinical status, family risk, gender, and transdiagnostic anxiety dimensions.

Method. Similar to the first study, the sample size of this cross-sectional study was determined based on an a prior power analysis. The study design and analyses were preregistered prior to data collection (https://osf.io/kxv5h). In line with open science practices, data and code are accessible in an online repository which is linked to the preregistration, ensuring transparency and reproducibility. EEG data of 156 participants (n = 115 female) aged 18 to 63 years and diagnosed with obsessive-compulsive disorder (OCD; n = 39), social anxiety disorder (SAD; n = 39), or specific phobia (PHOB; n = 39), as well as naturalistic control participants (CON; n = 39) were measured while performing an arrowhead version of a flanker task with speed feedback. Ensuring a higher external validity, participants of the naturalistic control group were allowed to fulfill the criteria for a mental disorder with the exception of the index diagnoses.

The four groups were matched regarding gender, age, and education years. All participants underwent structured clinical interviews to assess their diagnoses (SCID-5-CV; Beesdo-Baum et al., 2019; First et al., 2016) and diagnoses of their first-degree relatives (Family History Screen; Weissman et al., 2000). Most frequent diagnoses of the participants encompassed internalizing disorders including depression (41.0%), specific phobia (31.4%), SAD (29.5%), OCD (25.6%), and sleep disorders (12.8%). Similarly, participants with a family history of psychopathology most frequently reported internalizing disorders affecting at least one first-degree relative. The most common internalizing conditions included depression (45.5%), specific phobia (21.8%), SAD (13.5%), OCD spectrum disorders such as tic disorder, skin picking, and hoarding behavior (11.5%), panic disorder (11.5%), and GAD (10.9%). Among externalizing disorders, a family history of substance use disorder was the most frequently reported (22.4%). Based on the internalizing clinical diagnoses of the participants and their family risk for an internalizing psychopathology, these data allowed to reclassify them into four clinical and family risk groups: clinical participants with family risk (n = 88) and without family risk (n = 34), as well as nonclinical participants with family risk (n = 15) and without family risk (n = 19).

Additionally, symptom severity of each participant was measured using a battery of clinical questionnaires, including disorder-specific and transdiagnostic questionnaires. Disorder-specific measures encompassed obsessive-compulsive symptoms, measured by

the Obsessive-Compulsive Inventory Revised (OCI-R; Foa et al., 2002; Gönner et al., 2007), social phobia symptoms by the self-report version of the Liebowitz Social Anxiety Scale (LSAS-SR; Liebowitz, 1987; Stangier & Heidenreich, 2003), specific phobia symptoms by the Severity Measure for Specific Phobia (SMSP; Craske et al., 2013), and depression symptoms by the Beck Depression Inventory (BDI-II; Beck et al., 1996; Hautzinger et al., 2006). Transdiagnostic measures encompassed trait worry, measured by the Penn State Worry Questionnaire (PSWQ; Glöckner-Rist & Rist, 2014; Meyer et al., 1990), trait arousal by the anxious arousal subscale of the Mood and Anxiety Symptom Questionnaire (MASQ-AA; Watson & Clark, 1991; Watson et al., 1995), and trait anxiety by the respective subscale of the State-Trait-Anxiety Inventory (STAI-T; Laux et al., 1981; Spielberger et al., 1970).

The utilization of overlapping questionnaire batteries and the identical task from the first study facilitated even more powered analyses within a transdiagnostic sample comprising 246 participants (n = 181 female) spanning a broad range across the symptom severity spectrum (n = 136 clinical, n = 110 nonclinical). The most frequent disorders were depression (42.9%), specific phobia (32.7%), SAD (29.5%), OCD (26.1%), and sleep disorders (12.8%). Statistical analyses involved χ^2-tests, t-tests, one-way as well as mixed-measures analyses of variance (ANOVA) and analyses of covariance (ANCOVA), alongside multiple linear regression models.

Results. Both the diagnostic groups and the reclassified clinical and family risk groups showed no differences in demographic data. However, as expected, differences emerged regarding clinical symptoms and their severity. The diagnostic groups were each most pronounced in the disorder-specific symptoms. Regarding the transdiagnostic anxiety dimensions, trait worry and trait arousal were highest in the OCD and SAD group, on intermediate levels in the PHOB group, and lowest in the CON group. Trait anxiety, as well as depressive symptoms, were most pronounced in the OCD and SAD groups, with the CON group displaying the smallest manifestations, and the PHOB group having intermediate symptom severity. The reclassified clinical and family risk groups showed a consistent pattern in both disorder-specific symptoms and transdiagnostic anxiety dimensions, such that reported levels increased from nonclinical participants without family risk to nonclinical participants with family risk, clinical participants without family risk, and clinical participants with family risk.

Regarding the ERPs, comparing the diagnostic groups revealed no significant differences in ERN or CRN (Figure 6) among the clinical groups (OCD, SAD, PHOB) or when compared with the naturalistic control group (CON). However, after creating a more rigorously defined healthy control group (HC), there were small to medium sized trend-level significant differences in ERN amplitudes, with PHOB participants exhibiting larger amplitudes compared to HC participants. Moreover, clinical participants with a lifetime history of internalizing disorders displayed small to medium sized trend-level significant larger ERN and CRN amplitudes compared with those without any past or present diagnoses. Similarly, ERN and CRN were larger in the family risk group compared with the no risk group with a small to medium effect size (Figure 7). However, please note that these results of the clinical and family risk groups differed across ERP scoring strategies, with some of them not reaching statistical significance. Dimensional analyses did not reveal a general association between trait worry and ERN or CRN, respectively. Instead, gender emerged as a moderating factor in this relationship, with a small correlation of trait worry with larger ERN and CRN amplitudes in women but not in men (Figure 8).

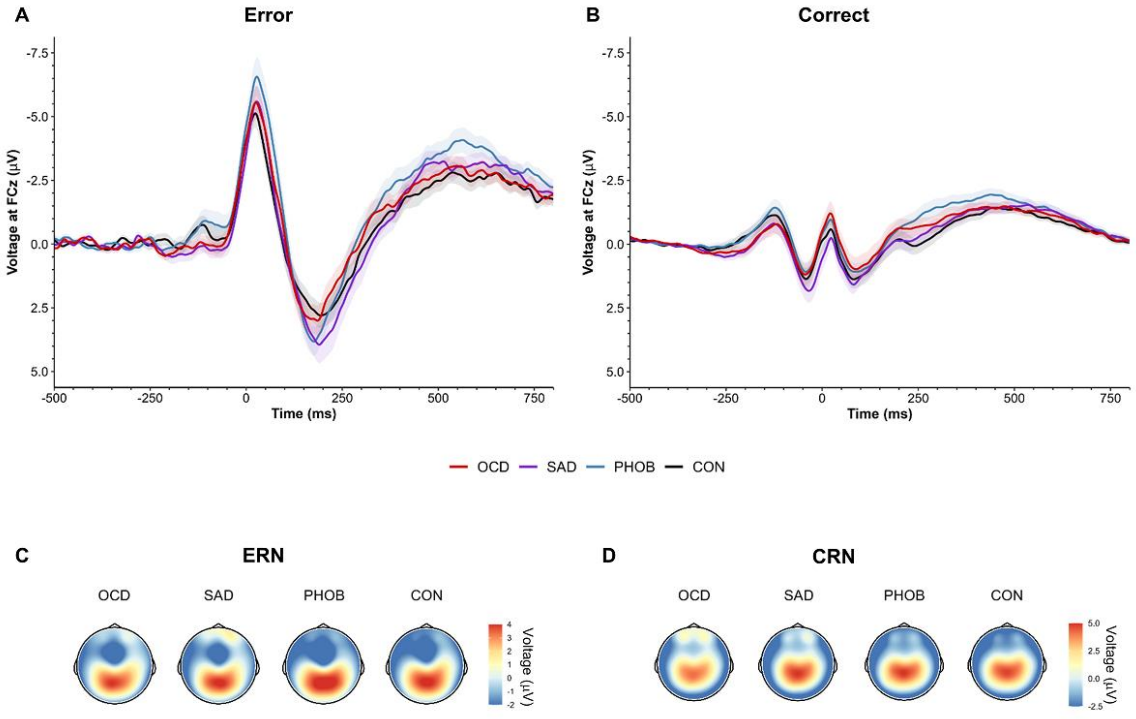

Figure 6. Response-locked grand-averaged waveforms of erroneous (A) and correct trials (B) of each group. OCD = Obsessive-Compulsive Disorder; SAD = Social Anxiety Disorder; PHOB = Specific Phobia; CON = Control Group. Corresponding topographic head maps (mean activity 0 – 100 ms) of the ERN (C) and CRN (D). ERN = error-related negativity; CRN = correct-response negativity. *N* = 156. Figure from Härpfer et al. (in revision).

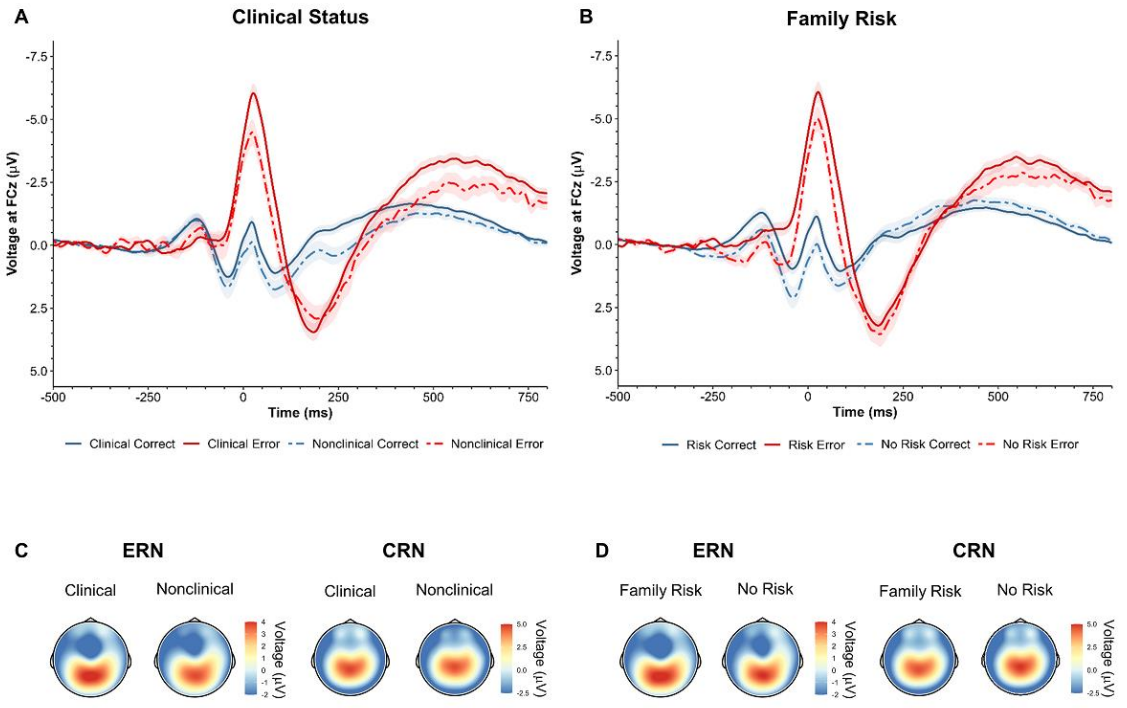

Figure 7. Response-locked grand-averaged waveforms of correct and erroneous trials for participants with or without a clinical status of an internalizing disorder (A) as well as for participants with or without a family risk for internalizing psychopathology (B). Corresponding topographic head maps (mean activity 0 – 100 ms) of the ERN (C) and CRN (D). ERN = error-related negativity; CRN = correct-response negativity. *N* = 156. Figure from Härpfer et al. (in revision).

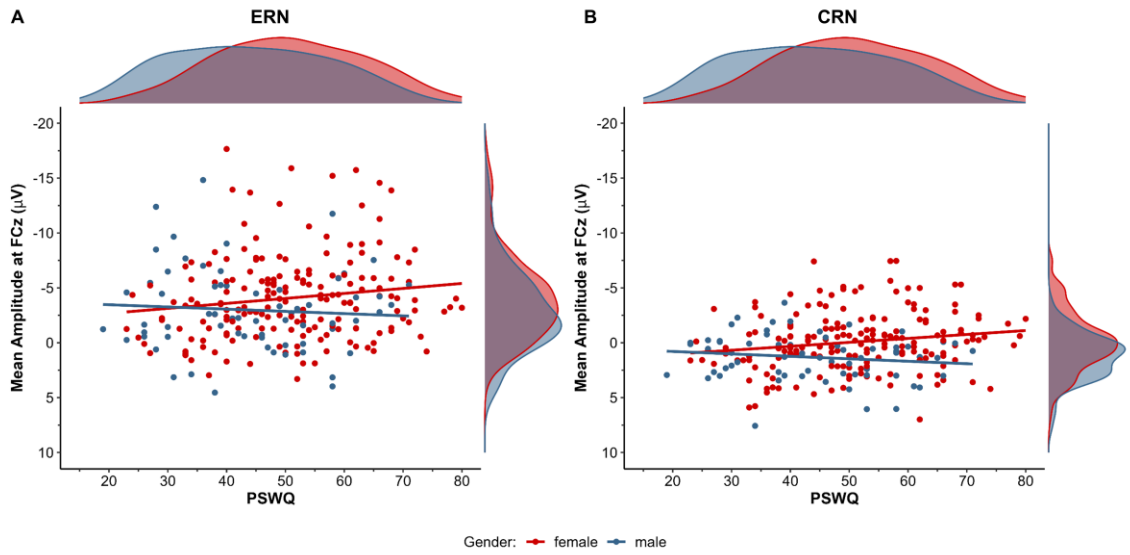

Figure 8. Gender moderating the link between trait worry and (A) ERN as well as (B) CRN. ERN = error-related negativity; CRN = correct-response negativity. PSWQ = Penn State Worry Questionnaire. *N* = 246. Figure from Härpfer et al. (in revision).

Additional Analyses. Results of additional analyses, not included in the main publication, are detailed in Appendix A2. To test for the specificity of the main investigations, potential differences between the diagnostic groups regarding the Pe were explored in the first analyses. None of the analyses revealed significant differences between the diagnostic groups (Table A2.1), nor when utilizing a strictly defined healthy control group (Table A2.2). The second analyses focused on the role of clinical status and family risk for internalizing psychopathology (Table A2.3). There was a significant interaction effect of clinical status and family risk on the early Pe with follow-up analyses indicating smaller amplitudes for nonclinical participants with family risk compared with those without family risk. Regarding the associations of the anxiety dimensions and the Pe, neither trait worry nor trait arousal were significant predictors when entered simultaneously (Table A2.4). However, when solely focusing on trait worry, both early and late Pe were smaller with increasing levels of trait worry (Table A2.5).

Discussion. Results revealed trend-level significant differences pointing to larger ERN amplitudes in der PHOB compared with the more strictly defined HC group, which suggests possible disorder-specific associations. This is particularly interesting, given that specific phobia is traditionally seen as being mostly associated with fear and physical symptoms, and thus, has not been linked to an enhanced ERN (Hajcak et al., 2003; Moser et al., 2005). Challenging the notion of disorder-specific associations of the ERN, comparisons of the OCD, SAD, and PHOB group against the more conservative naturalistic CON group did not yield significant differences. Similarly, when omitting disorder-specific groups and integrating them into a broader spectrum of internalizing disorders (e.g., anxiety disorders, obsessive-compulsive disorders, depression, eating disorders etc.), participants with a lifetime internalizing disorder showed a trend-level increased ERN and CRN compared to those without any current or past diagnoses. This supports the perspective that both an enhanced ERN and CRN may serve as broader neural risk markers across the internalizing spectrum (Pasion & Barbosa, 2019), rather than being specific to certain disorder categories within this spectrum. Regarding the Pe, no significant associations were found.

In addition to clinical status of an internalizing disorder, family risk for internalizing psychopathology was identified as being associated with heightened ERN and CRN amplitudes (Carrasco, Harbin, et al., 2013; Riesel et al., 2011; Riesel, Klawohn, et al., 2019).

Interestingly, these factors did not exhibit an additive effect, but rather independently accounted for variance in ERN and CRN. This is a new finding since previous studies did not simultaneously examine the potential influence of an individual's clinical status as well as their family risk. Consequently, while some individuals (i.e., those with familial risk) may have inherited an enhanced ERN or CRN, predisposing them to clinical anxiety and obsessive-compulsive symptoms, others (i.e., those without familial risk) may have developed increased amplitudes due to other influences (e.g., temperament, parenting style), carrying a comparable risk for clinical symptom development. Another new finding is that the early Pe was reduced in individuals with family risk but did not differ between those with or without an internalizing disorder. However, since the functional role of the Pe is less clear, with some arguing it represents an indicator of error awareness (Endrass et al., 2007; Overbeek et al., 2005; Wessel, 2012), future research is needed to firstly, replicate this finding, and secondly, examine the potential underlying mechanism of this potential transgenerational transmission.

Regarding the role of transdiagnostic anxiety dimensions, trait worry but not trait arousal (Lin et al., 2015; Moran et al., 2012; Moser et al., 2012; Weinberg et al., 2010), was found to be associated with enhanced ERN and CRN amplitudes (Moser et al., 2013; Saunders & Inzlicht, 2020), particularly in women (Lin et al., 2015; Moran et al., 2012; Moser et al., 2012; Moser et al., 2016). This finding highlights the potential of a transdiagnostic perspective on brain activity to uncover more precise associations between psychological and physiological measures, that are likely overshadowed when utilizing heterogeneous and possibly artificial disorder categories (Cuthbert, 2014; Insel et al., 2010; Kozak & Cuthbert, 2016; Sharp et al., 2015). For the early Pe, the opposite direction of effects of trait worry were found, suggesting decreasing conscious processing of errors in individuals with a tendency to persistent worries. This might be an indicator of the distracting effects of worries on working memory capacity (Moser et al., 2013; Schroder et al., 2018) or avoidance motivation.

Conclusion. Overall, the second study supports the role of an enhanced ERN as well as CRN as dispositional markers of vulnerability for internalizing psychopathology, potentially influenced by multiple moderators such as family risk and gender in the etiopathogenesis of clinical symptoms and their potential underlying dimensions, such as trait worry. However, this was a cross-sectional research design, most of the effects were

of small to medium size, and some of them were only trend-level significant, warranting further replication in future and preferably prospective studies. Furthermore, the internalizing spectrum was not fully represented, limited to the index diagnoses including depression, specific phobia, SAD, OCD, and sleep disorders.

3.3 Study 3: Translating Risk into Internalizing Psychopathology

Citation. Riesel, A., Härpfer, K., Kathmann, N., & Klawohn, J. (2021). In the face of potential harm: The predictive validity of neural correlates of performance monitoring for perceived risk, stress, and internalizing psychopathology during the COVID-19 pandemic. *Biological Psychiatry: Global Open Science, 1*(4), 300-309. https://doi.org/10.1016/j.bpsgos. 2021.08.004

Objective. A substantial body of literature has identified an increased ERN as a neural risk marker predisposing individuals to internalizing psychopathology (Pasion & Barbosa, 2019). However, the mechanisms underlying how an elevated ERN, indicative of vulnerability, translates into clinical conditions remain less elucidated, with only few prospective investigations delving into these pathways (Lahat et al., 2014; Meyer, Hajcak, et al., 2015; Meyer et al., 2021; Meyer, Nelson, et al., 2018). The diathesis-stress model underscores the pivotal role of stress as a catalyst in the transition from vulnerability to the onset of clinical symptoms (Banica et al., 2020; Meyer, 2017; Weinberg et al., 2022). The historic event of the COVID-19 pandemic provided a unique research environment to test the hypothesis that a major real-life stressor, with potentially unpredictable negative consequences for one's personal health, might facilitate the development of internalizing psychopathology in individuals at risk, particularly those who exhibit an enhanced ERN reflecting heightened threat sensitivity. Thus, this third study sought to explore the predictive value of the pre-pandemic ERN on the subsequent risk perception, self-reported stress, and internalizing symptoms during the initial phase after the outbreak of the pandemic.

Method. This third prospective longitudinal study was rather explorative in nature and strongly influenced by the outbreak of the COVID-19 pandemic. A cohort comprising 317 control participants from three previous EEG studies (Härpfer et al., 2020; Klawohn, Hajcak, et al., 2020; Riesel, Klawohn, et al., 2019) were invited to participate in a subsequent follow-up study during the initial phase of the pandemic from February until May 2020. Ultimately, the final sample consisted of 113 individuals (n = 71 female), aged

20 to 63 years, whose EEG data were recorded during a flanker task between 0.83 and 5.38 years before the pandemic, participated in the present follow-up study. In addition to EEG data, anxiety, depression, and obsessive-compulsive symptoms were measured at the baseline assessment preceding the pandemic. Obsessive-compulsive symptoms were measured by the Obsessive-Compulsive Inventory Revised (OCI-R; Foa et al., 2002; Gönner et al., 2007), depression symptoms by the Beck Depression Inventory (BDI-II; Beck et al., 1996; Hautzinger et al., 2006). and trait anxiety by the respective subscale of the State-Trait-Anxiety Inventory (STAI-T; Laux et al., 1981; Spielberger et al., 1970). Subsequently, these measures alongside measures of stress perception and the perceived risk for a severe COVID-19 infection were readministered during the initial phase of the COVID-19 pandemic in the follow-up. A structured clinical interview for DSM-IV (Wittchen et al., 1997) was also conducted to assess the potential onset of mental disorders. Participants that fully completed the follow-up phase did not differ regarding demographic or clinical characteristics compared to those that did not, nor did they differ regarding ERN and CRN amplitudes. Additionally, no significant differences regarding ERPs were observed between the three original studies. Statistical analyses involved χ^2-tests, *t*-tests, one-way analyses of variance (ANOVA), alongside simple and serial mediation models.

Results. Clinical interviews revealed that only a minority of participants showed a first onset of a disorder after the baseline assessment, including depression (n = 9), agoraphobia (n = 3), posttraumatic stress disorder (n = 2), and panic disorder (n = 1). Due to the limited frequency of these first-onset disorders, categorical analyses regarding clinical diagnoses were not conducted. Furthermore, there was no overall increase in anxiety, obsessive-compulsive, or depressive symptoms from baseline to follow-up assessments. However, the conducted mediation models uncovered differential pathways from ERPs to internalizing symptoms between individuals.

The simple mediation model revealed that neither pre-pandemic ERN nor CRN were directly linked to stress perception during the pandemic. Instead, perceived (but not objective) COVID-19 risk mediated the indirect effect. Specifically, an increased ERN and CRN were associated with greater stress perception through higher perceived risk. Individuals with heightened neural performance monitoring reported higher levels of perceived risk for a COVID-19 infection and a severe course, which in turn, was associated with elevated stress levels. Additionally, the serial mediation model demonstrated an

indirect effect of an elevated ERN and CRN on internalizing symptoms, mediated by heightened perceived risk and increased stress perception (Figure 9). These mediations were also evident for anxiety, obsessive-compulsive, and depressive symptoms, when controlling for the respective baseline symptom severities, and were specific to ERN and CRN, as no significant mediating effects were observed for the response-locked Pe (neural index of error awareness) and stimulus-locked N2 (neural index of response inhibition).

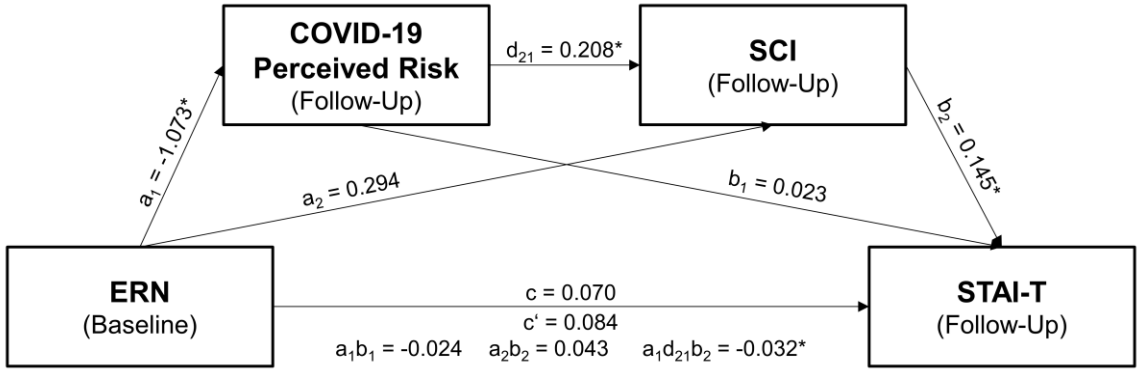

Figure 9. Serial mediation model examining the pre-pandemic ERN (baseline) as a predictor of anxiety symptoms during the pandemic (follow-up), with perceived COVID-19 risk and stress as mediators. The mediation model is controlled for STAI-T at baseline and time difference between baseline and follow-up. ERN = error-related negativity; SCI = Stress and Coping Inventory; STAI-T = Spielberger State-Trait Anxiety Inventory (trait anxiety). Asterisks indicate significance level: * $p < .05$. $N = 113$. Figure adapted from Riesel et al. (2021).

Additional Analyses. Results of additional analyses, not included in the main publication, are detailed in Appendix A3. To investigate the predictive value of ERPs regarding the onset of psychopathology, we compared those participants with a first onset of an internalizing disorder after the baseline assessment with those without a lifetime history of psychopathology (Table A3.1). Neither ERN, CRN, Pe, nor N2 differed between these groups. However, please note, diagnoses were assessed retrospectively, only at the follow-up assessement.

Discussion. The COVID-19 pandemic provided a unique opportunity to investigate how neural markers of vulnerability translate into internalizing symptoms under real-life conditions. Findings from this third study unequivocally demonstrate that a real-life stressor (Banica et al., 2020; Meyer, 2017), such as a pandemic, does not uniformly impact all individuals, but rather selectively affects those most vulnerable and at risk (Weinberg et

al., 2022). The study also underscores the conceptualization of performance monitoring related ERPs as neural indices of threat sensitivity (Hajcak & Foti, 2008; Heffer & Willoughby, 2021; Proudfit et al., 2013; Weinberg et al., 2016; Weinberg et al., 2012): Interindividual variations in ERN and CRN, but not Pe or N2, appear closely linked to risk perception. Risk perception, in turn, emerges as a potential mediating factor explaining why individuals with heightened ERN are more prone to stress, a new finding warranting replication to further test this hypothesis.

Moreover, this study demonstrates the predictive utility of the ERN and CRN for internalizing psychopathology above and beyond preexisting symptoms (Lahat et al., 2014; Meyer, Hajcak, et al., 2015; Meyer et al., 2021; Meyer, Nelson, et al., 2018). Importantly, these indirect effects mediated by risk and stress perception extend beyond error-specific processes to encompass broader facets of general performance monitoring. However, direct links between ERN/CRN and internalizing symptoms within this mainly nonclinical sample were not discerned, pointing to the pivotal role of clinical status as a likely moderator (Saunders & Inzlicht, 2020). Critically, it is noted that the research design lacked prospective assessment in all paths of the mediation models, as risk and stress perception, along with internalizing symptoms, were measured concurrently during the pandemic and clinical diagnoses were assessed retrospectively.

Conclusion. The findings of this study not only detail trajectories from increased neural performance monitoring to internalizing symptoms, they also highlight possible underlying mechanisms including risk perception and stress. As this study was guided by an explorative nature, this mechanism can be a valuable hypothesis to be tested in future research. Additionally, this study also unveiled compelling new targets for tailored interventions: Addressing dispositional vulnerability by modulating neural performance monitoring, along with strategies to mitigate risk perception and enhance stress management, might be a promising approach in preventing the exacerbation of internalizing symptoms during stressful periods for individuals at risk.

4. General Discussion

4.1 Summary of Findings

Employing cross-sectional, randomized-controlled, and prospective study designs, this dissertation sought to elucidate the role of error-related brain activity in the pathophysiology of the anxiety and obsessive-compulsive spectrum. More precisely, this research endeavor aimed at disentangling trait and state influences on the ERN, exploring its associations with transdiagnostic symptom dimensions across boundaries of traditional diagnostic categories, and investigating potential moderators and mediators that might translate neural vulnerability into psychopathology, altogether while demonstrating the potential specificity of the corresponding brain potentials.

In study 1 (Härpfer et al., 2022), experimental manipulations of state worry did not influence the magnitude of the ERN and CRN, suggesting that intraindividual variability in the ERN and CRN are not a correlate of situational worries. Instead, study 1 and 2 (Härpfer et al., in revision; Härpfer et al., 2022) identified interindividual differences of trait worry – a trait characterized by a higher propensity to worrisome rumination – as an underlying transdiagnostic trait dimension cross-sectionally linked to both ERN and CRN variations, while this link was not observed for trait arousal – an individual's tendency to respond with heightened arousal and somatic tension to even mild stressors. Moreover, results imply that an increased ERN and CRN are not specific to particular disorder types but rather to broader categories such as clinical status of an internalizing disorder (trend-level significant effect) and family risk for internalizing psychopathology. Together, these findings underscore the trait-like nature of the association (i.e., trait worry) with electrophysiological indices of performance monitoring, while no evidence was found for a link with state-associated variability (i.e., state worry). Regarding differential associations with internalizing psychopathology, gender emerged as a moderator of the cross-sectional link between ERN/CRN and trait worry, such that larger amplitudes associated with increased levels of trait worry were observed specifically in women.

Additionally, the prospective research design of study 3 (Riesel et al., 2021) demonstrated that both an increased pre-pandemic ERN and CRN were indirectly linked to stronger anxiety, obsessive-compulsive, and depressive symptoms during the COVID-19 pandemic. This effect was mediated by risk perception and the reported levels of stress. Specifically, participants with an enhanced ERN and CRN exhibited higher levels of risk

perception regarding an infection and a severe course of COVID-19. This heightened risk perception was associated with increased stress levels, which, in turn, were linked to greater internalizing symptoms. Although there was no overall increase in symptom severity in comparison to pre-pandemic levels when comparing the distribution of the sample, these findings indicate, on a finer resolution level, that individuals with heightened performance monitoring are particularly at risk of experiencing more severe internalizing symptoms under stressful conditions, due to their increased risk or threat perception.

Furthermore, across all three studies, the majority of associations within the anxiety and obsessive-compulsive spectrum were not error-specific. Instead, they extended to general performance monitoring processes including the monitoring of correct and erroneous responses, as indexed by both altered ERN and CRN amplitudes. In addition, later components such as the Pe and N2 did not exhibit comparable associations with psychopathology, suggesting a potential specificity for ERPs associated with performance monitoring rather than those involved in the conscious processing of actions (Pe) or response inhibition (N2).

4.2 Investigating Trait and State Variability of the Error-Related Negativity

Previous research has investigated various potential underlying latent dimensions linking enhanced ERN amplitudes and anxiety. Among these dimensions are intolerance of uncertainty (Jackson et al., 2016; Jackson et al., 2015), anxious misery (Riesel et al., 2023), harm avoidance (Meyer et al., 2021; Riesel, Klawohn, et al., 2019), and checking behavior (Weinberg, Kotov, et al., 2015; Weinberg et al., 2016), all of which have been supported by primary studies. However, the focus of this dissertation has been inspired by meta-analytical evidence suggesting that the association between enhanced ERN amplitudes and anxiety might be attributed to the latent dimension of trait worry (Moser et al., 2013; Saunders & Inzlicht, 2020). Predominantly, research in this area conceptualized trait worry as a stable trait-like characteristic, while ignoring the potential varying associated states such as state worry, and focused mostly on interindividual differences of the ERN without testing for the specificity regarding other brain potentials.

Nonetheless, the ERN has been shown to be dynamic in its magnitude depending on the task demands. For instance, the ERN shows state variability within a person when accuracy is emphasized over speed (Falkenstein et al., 2000; Gehring et al., 1993; Riesel, Kathmann, & Klawohn, 2019), when errors are punished (Meyer & Gawlowska, 2017;

Riesel, Kathmann, Wüllhorst, et al., 2019; Riesel et al., 2012) or socially evaluated (Buzzell et al., 2017; Voegler et al., 2018), during dual task demands (Klawohn et al., 2016), or with changing positive affect (Nigbur & Ullsperger, 2020) and negative affect (Wiswede, Münte, Goschke, et al., 2009; Wiswede, Münte, & Rüsseler, 2009). In the context of trait worry and its link to altered ERN amplitudes, it is conceivable to hypothesize that this link may stem from a situational increase of state worry that trait-worrisome individuals might more frequently experience during task performance, which could lead to an overestimation of the true association of trait worry and the ERN due to increased state worry. In fact, a first study found that an emotional expressive writing paradigm, aiming at reducing state worry in chronic worriers, was associated with a reduced ERN, suggesting a link between variability in state worry and the ERN (Schroder et al., 2018).

Building on this initial evidence, study 1 focused on disentangling the specific effects of trait and state worry on the ERN through experimental manipulations designed to either induce or reduce state worry within a randomized controlled repeated-measures design. However, fluctuations in state worry did not lead to intraindividual changes in the amplitude of the ERN, CRN, or Pe. Instead, interindividual differences in trait worry were found to be associated with neural indices of performance monitoring; increased trait worry correlated with heightened ERN and CRN amplitudes, but not with Pe. These findings support the assertion of the literature of a trait-like nature of the relationship between error-related brain activity and anxious psychopathology (e.g., Michael et al., 2021; Moser et al., 2013; Proudfit et al., 2013; Weinberg et al., 2012) and align with other research demonstrating the independence of the ERN from state fluctuations of anxiety symptoms (Gorka et al., 2018; Kujawa et al., 2016; Ladouceur et al., 2018) and obsessive-compulsive symptoms (Hajcak et al., 2008; Riesel et al., 2015). Considering the results of study 1 together with previous findings, no evidence could be identified qualifying the ERN, CRN, or Pe as neural correlates of state worry. Instead, ERN and CRN appear to reflect a more general trait-like predisposition to experiencing worry and anxiety (i.e., trait worry), independent of state conditions (i.e., state worry). Consequently, both ERN and CRN manifest as promising biomarkers for identifying individuals predisposed to mental states of habitual worry (Hajcak et al., 2019; Meyer, 2016; Michael et al., 2021; Moser et al., 2013), potentially serving as valuable tools in the risk assessment for anxiety-related conditions in the future (Lahat et al., 2014; Meyer, 2017; Meyer, Hajcak, et al., 2015; Meyer et al., 2021; Meyer, Nelson, et al., 2018).

4.3 Adopting a Transdiagnostic and Dimensional Perspective

As previously noted, disorders within the anxiety and obsessive-compulsive spectrum exhibit a high degree of comorbidity (Kessler et al., 2005; Ruscio et al., 2010; Sharma et al., 2021) and share overlapping diagnostic criteria (Forbes et al., 2024); however, they can be phenotypically heterogeneous with various triggering objects and situations provoking corresponding symptoms. This heterogeneity is also mirrored in the association with increased error-related brain activity, as only GAD (Meyer, Weinberg, et al., 2012; Weinberg et al., 2010), SAD (Endrass et al., 2014; Kujawa et al., 2016), health anxiety (Riesel et al., 2017), and OCD (Bellato et al., 2021; Mathews et al., 2012; Riesel, 2019) show an increased ERN, while links have not been found in specific phobia (Hajcak et al., 2003; Moser et al., 2005) and PTSD (Gorka et al., 2016; Rabinak et al., 2013). A hypothesis regarding these differential associations suggests that shared latent dimensions underlie these interrelated disorders. Previous research posits that disorders associated with an increased ERN may be characterized by elevated levels of trait worry (Moser et al., 2013; Saunders & Inzlicht, 2020).

Study 1 and 2 support this transdiagnostic perspective, finding that categorical comparisons of specific diagnostic groups (OCD, SAD, specific phobia, naturalistic control group) did not yield significant differences in neither ERN, CRN, nor Pe. However, broader comparisons encompassing all internalizing disorders against control participants, as well as assessments focusing on transdiagnostic dimensions such as trait worry, identified enhanced amplitudes of both ERN and CRN. Regarding the Pe, study 2 found that trait worry was associated with a smaller Pe, indicating less conscious processing of errors. Furthermore, the serial mediation models employed in study 3 did not uncover specific associations with either anxiety, obsessive-compulsive, or depressive symptoms. Together, these observations strengthen the utility of a transdiagnostic perspective by indicating a broader association between enhanced neural indices of performance monitoring and internalizing psychopathology, cutting across the boundaries of distinct diagnostic categories and their specific symptom dimensions. Additionally, these findings imply that an increased ERN and CRN may primarily function as risk markers specifically for those individuals with internalizing psychopathology that are characterized by the latent dimension of trait worry The lack of specificity to a particular diagnostic category may also support a multifinal perspective, suggesting that enhanced neural performance monitoring is linked to a broader at-risk state for internalizing psychopathology, preceding the potential

diverse developmental pathways of internalizing disorders. Overall, the methodological disparity between a cross-sectional, etiologically-blind, symptom-based categorical classification of complex mental disorders and the typically continuous distribution of brain mechanisms ranging from normal to extreme values, potentially obstructs the investigation of interactions between these domains (Abi-Dargham et al., 2023; Clayson, 2024; Moriarity et al., 2022; Teixeira et al., 2023). Alternatively, adopting a transdiagnostic and dimensional framework for psychopathology could mitigated this methodological disparity, thereby facilitating the identification of specific associations between psychological constructs and physiological measures (Hajcak et al., 2019; Insel et al., 2010). The presented results of this dissertation support this assertion, showing no notable differences between particular diagnostic groups while significant effects were found when examining broader internalizing categories, as well as continuous transdiagnostic dimensions, which more closely capture the continuous nature of psychopathological phenomena. A transdiagnostic and dimensional approach may offer a more nuanced understanding of the underlying mechanisms contributing to mental disorders, thereby holding significant potential to illuminate the neural underpinnings of psychopathology.

4.4 The Role of Clinical Status and Family Risk

Research on the potential neural foundations of internalizing psychopathology has demonstrated that heightened error-related neural activity is present not only in patients diagnosed with disorders of the anxiety and obsessive-compulsive spectrum but also in their unaffected relatives (Carrasco, Harbin, et al., 2013; Riesel et al., 2011; Riesel, Klawohn, et al., 2019). Study 2 was designed to elucidate contributions of the clinical status of an individual on the one hand and the risk transmitted within families on the other. Findings indicated that both clinical status of an internalizing disorder and family history of internalizing psychopathology independently contributed to elevated levels in both ERN and CRN amplitudes. This suggests that heightened neural performance monitoring may be transmitted within families only in a subset of patients, whereas in those without family risk, alternative factors likely influenced the development of increased performance monitoring. Among others, the equifinality of factors discussed as candidates shaping an individual's performance monitoring system encompasses for instance, biological sex (Larson et al., 2011), gender (Fischer et al., 2016; Hill et al., 2018; Imburgio et al., 2020), cognitive capacity (Meyer & Hajcak, 2019), and temperamental factors such as shyness (Meyer & Klein, 2018; but see Brooker et al., 2011), behavioral inhibition (Buzzell et al.,

2017; Lahat et al., 2014; McDermott et al., 2009), fearfulness (Brooker & Buss, 2014a, 2014b; Meyer, Hajcak, et al., 2018), impulsivity (Taylor et al., 2018), and risk-tolerance (Santesso & Segalowitz, 2009).

However, study 2 does not permit an in-depth exploration of the mechanisms responsible for the transmission of family risk. Both genetic (Anokhin et al., 2008) and environmental factors (Chong et al., 2020; Meyer et al., 2019; Meyer, Proudfit, et al., 2015; Meyer & Wissemann, 2020; Suor et al., 2022) would be plausible mediators, yet such causal relationships need to be investigated through longitudinal research designs. Consequently, only twin or adoption studies (Anokhin et al., 2008) allow to effectively disentangle the contributions of genetic and environmental factors to family risk (Suor et al., 2022; Trayvick et al., 2024); however, such studies are relatively scarce. Nonetheless, considering not only the clinical status but also the family history of psychopathology of an individual might help identify those prone to neural vulnerability more precisely.

4.5 Moderating and Mediating Factors

In addition the influence of family risk transmission, this dissertation aimed at elucidating the specific associations more generally along the pathway from neural vulnerability to psychopathology. A prominent factor moderating the association between increased ERN amplitudes and anxiety is biological sex (or gender[2]), with stronger associations observed in women compared with men (Moser et al., 2016). Study 1 and 2 confirmed this finding, demonstrating that higher levels of trait worry were associated with increased ERN and CRN amplitudes in women, but not in men. This was specific for the ERN and CRN, since gender did not appear to modulate the association between trait worry and the Pe. However, the cause of the differential findings between women and men remains open. As many previous studies, the assessment of the studies included in this dissertation was based on a single demographic item not differentiating between biological sex and gender. This does not allow to disentangle the influences of biological factors and an individual's gender identity. Nonetheless, two hypotheses, each emphasizing different aspects, have been proposed to explain the gender-specific link between trait worry and increased error monitoring (Moser et al., 2016).

[2] As described earlier, this depends on how previous research operationalized and assessed the biological sex or gender of an individual.

The first hypothesis assumes that the impact of verbal worries on cognitive performance is stronger in women. Extensive research indicates that anxiety is both cross-sectionally and prospectively linked to cognitive impairments (Gulpers et al., 2022; Lindert et al., 2021; Tetzner & Schuth, 2016), with some studies pointing to more substantial impairments in women (de Visser et al., 2010; Gulpers et al., 2019; Zainal & Newman, 2023). This finding could be attributed to the tendency of women, relative to men, to engage more in verbal processing during problem-solving activities, thereby more frequently employing subvocal articulation as a response to threatening or uncertain situations (Moser et al., 2016). Alternatively, the second perspective highlights the organizational and regulatory impact of ovarian hormones on frontal brain regions associated with cognitive control in women (Beltz & Moser, 2020; Russman Block et al., 2024; Shansky & Lipps, 2013). It has been proposed that the heightened concentration of estradiol in brain regions associated with error monitoring, combined with the influence of estradiol on dopamine—a key neurotransmitter involved in ERN generation—provides a promising neurobiological rationale for the observed link between trait worry and ERN in women (Moser et al., 2016). In fact, recent findings suggest that estradiol and progesterone may act as protective factors, with women exhibiting relatively higher levels of these hormones showing a weakened correlation between worry and error-related brain activity in the ACC (Russman Block et al., 2024). Nevertheless, the study designs employed in this dissertation do not permit an endorsement of one hypothesis over the other regarding these gender-specific links. However, given these insights, error-related brain activity might represent a more accurate risk marker for trait worry in women compared with men, while the exact mechanisms underlying this gender-specific link are yet to be explored. Consequently, this finding highlights the critical importance of incorporating biological sex or gender as a variable when exploring the neural mechanisms underlying disorders within the anxiety and obsessive-compulsive spectrum. Such a focus can augment our understanding of these disorders and potentially guide more tailored and personalized therapeutic approaches.

Another important modulating impact on the relationship between enhanced error-related brain activity and internalizing psychopathology has been observed for stress related to adverse life events (Meyer et al., 2017) or persistent interpersonal conflicts (Banica et al., 2020). The COVID-19 pandemic provided a unique context to further explore how adverse life events impact individuals at risk for mental health issues, as indicated by enhanced ERN amplitudes. Study 3 found that not stress itself influenced the link between

the ERN and internalizing symptoms including anxiety, obsessions, compulsions, and depression. Instead, individuals with an increased ERN reported higher levels of stress associated with a stronger subjective perception of the risk for their personal health during the pandemic. Furthermore, the increased levels of stress observed in these individuals were nonspecifically associated with a rise in internalizing symptoms above pre-pandemic levels. This is a new finding pointing to a more complex interaction in which interindividual differences in error-related brain activity contribute to the subjective experience of stress during potentially threatening periods like a pandemic, further strengthening the perspective of the ERN as an indicator of threat sensitivity (Hajcak & Foti, 2008; Heffer & Willoughby, 2021; Proudfit et al., 2013; Weinberg et al., 2016; Weinberg et al., 2012). Additionally, a similar pattern of indirect effects was observed for the CRN, but not for the Pe or the N2. This suggests a more specific linkage between the processing of errors and correct responses, and heightened internalizing symptoms under stress, highlighting the relevance of ERN and CRN as potential neural markers for vulnerability in the face of significant stressors. Taken together, study 3 indicates a complex interaction where heightened sensitivity to threats and risks, as indexed by a larger ERN and CRN, can longitudinally amplify the psychological impact of stressful events, which in turn exacerbates various internalizing symptoms. Furthermore, internalizing symptoms may themselves contribute to heightened stress, for instance, through interpersonal conflicts resulting from reassurance-seeking behavior in patients with anxiety or obsessive-compulsive disorders. This dynamic could perpetuate internalizing symptoms, particularly in individuals who exhibit increased baseline ERN or CRN. These potential cycles highlight the need for targeted interventions that address both the heightened risk or threat sensitivity and the resulting stress responses in individuals at risk for internalizing psychopathology in the context of significant external stressors.

4.6 An Integrative Model of Error-Related Brain Activity and Psychopathology

The extensive body of literature on the ERN and its association with anxiety and obsessive-compulsive psychopathology has provided valuable insights into the neural processes underlying these clinical conditions. However, previous studies have employed diverse frameworks, leading to partially heterogeneous findings regarding the precise pathways through which neural markers of vulnerability translate into clinical manifestations of anxiety, obsessions, and compulsions. From a neurobiological perspective, the RDoC framework (Cuthbert, 2014; Insel et al., 2010; Kozak & Cuthbert,

2016) prepares the foundation for psychophysiological research by delineating domains of basic cognitive, emotional, motoric, regulatory, and social functions, along with their corresponding neurobiological substrates and levels of analysis. Regarding the clinical viewpoint, the HiTOP framework (Kotov et al., 2017) provides a hierarchical and dimensional taxonomy of psychopathology cutting across the boundaries of diagnostic groups, describing phenotypical expressions of mental disorders in subdivisions of transdiagnostic spectra, components, and symptoms. Finally, bridging neurobiological and psychopathological outcomes, the diathesis-stress model elucidates the development of psychopathology, such as anxiety and obsessive-compulsive symptoms (Meyer, 2017; Weinberg et al., 2022), as a result of the interplay between predispositional vulnerabilities (e.g., heightened ERN) and environmental stressors (e.g., adverse life events). Integrating these three frameworks provides great potential for researchers to better understand the complex interplay between individual neural vulnerability, psychopathological conditions, and environmental influences, thus advancing our comprehension of anxiety and obsessive-compulsive disorders and guiding future research.

To date, the research field has not established an overarching and integrative model elucidating the role of error monitoring in the development of psychopathological conditions, despite numerous studies exploring various facets of this relationship. Consequently, this dissertation seeks to fill this gap by proposing an integrative model of error-related brain activity and psychopathology (Figure 10), which not only outlines the pathways leading to psychopathology, but also specifies testable predictions for future research. This model pursues three primary objectives: first, to integrate knowledge on the equifinal trajectories to neural vulnerability, as indexed by trait-like alterations of the ERN; second, to incorporate current frameworks including the RDoC matrix, the HiTOP taxonomy, and the diathesis-stress model, and third, to expand the diathesis-stress model by delineating individual and environmental moderators and mediators, representing multifinal pathways from altered error-related brain activity to psychopathology. Through this integrative approach, the present dissertation aims to offer a comprehensive understanding of the interplay between altered error monitoring processes of an individual and the development of psychopathological conditions, thereby advancing theoretical and empirical research in the field. The integrative model consists of six pivotal components including early shaping factors, vulnerability, moderators and mediators, as well as psychopathological spectra, dimensions, and symptoms.

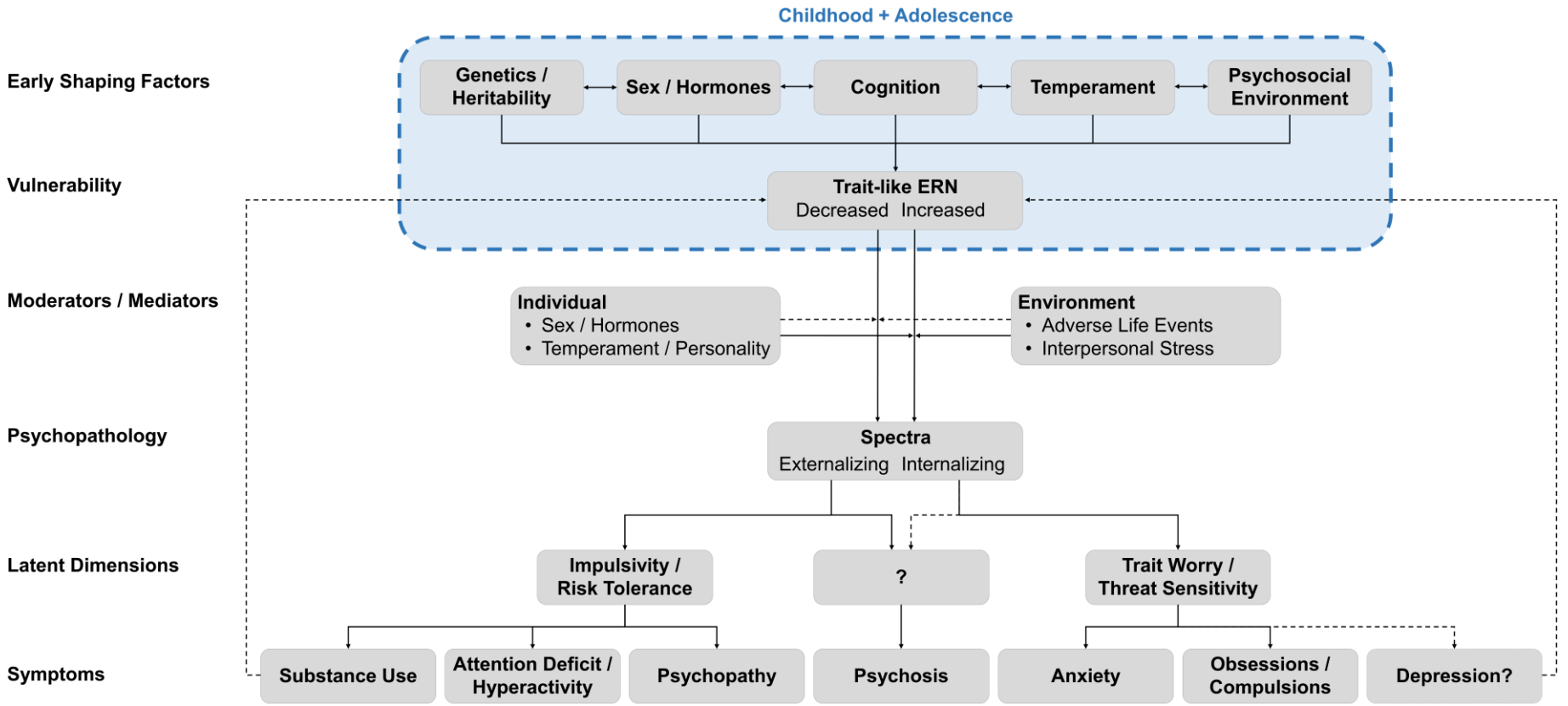

Figure 10. Integrative model of error-related brain activity and psychopathology. Error-related brain activity is indexed by the response-locked event-related potential termed error-related negativity (ERN). Dashed lines and question marks visualize hypothetical relationships or empirically inconsistent findings.

First, the model proposes a complex interplay of early shaping factors during childhood and adolescence influencing the formation of a trait-like hypo- or hyperactive error monitoring system. As pointed out in the introduction, previous research has investigated multiple potential factors resulting in a long but certainly not exhaustive list of equifinal factors including genetics (Anokhin et al., 2008; Suor et al., 2022; Trayvick et al., 2024), biological sex (Larson et al., 2011), gender (Fischer et al., 2016; Hill et al., 2018; Imburgio et al., 2020), sex hormones (Gorday & Meyer, 2018), cognitive capacity (Meyer & Hajcak, 2019), temperament (e.g., McDermott et al., 2009; Meyer & Klein, 2018; Santesso & Segalowitz, 2009; Taylor et al., 2018), and psychosocial environment (e.g., Debnath et al., 2023; Mehra et al., 2022; Meyer, Proudfit, et al., 2015). Moreover, the model posits that these stable interindividual alterations in the error monitoring network, as signaled by altered ERN amplitudes, constitute a neural vulnerability (Meyer, 2017; Weinberg et al., 2022) for either externalizing psychopathology in individuals with a decreased ERN (Lutz et al., 2021) or for internalizing psychopathology in individuals with an increased ERN (Pasion & Barbosa, 2019).

However, these trajectories are not inherently deterministic, as only a subset of individuals develop corresponding clinical symptoms (Carrasco, Harbin, et al., 2013; Euser et al., 2013; McLoughlin et al., 2009; Riesel et al., 2011; Riesel, Klawohn, et al., 2019; Simmonite et al., 2012). The relationship between altered neural error monitoring and psychopathology is moderated and mediated by additional individual and environmental factors. In accordance with predictions of the diathesis-stress model, adverse environmental conditions, such as a natural disaster (Meyer et al., 2017), a pandemic (Riesel et al., 2021), or prolonged interpersonal stress (Banica et al., 2020), exert a greater negative effect on those who are more vulnerable (i.e., those with heightened ERN), potentially leading to serious mental health impairments. Additionally, negative parenting styles have been found to mediate the relationship between an enhanced maternal ERN and offspring internalizing psychopathology (Suor et al., 2022). Alongside these environmental influences, individual characteristics also play a significant role. Biological sex or gender have been identified as a crucial moderating factor, with the association between increased ERN amplitudes and anxiety being stronger in females than in males, although this does not extend to obsessive-compulsive symptoms (Moser et al., 2016). Emerging research also indicates that variations in ovarian hormone levels, specifically estradiol and progesterone, affect the association between anxiety and heightened error monitoring in females (Russman Block et al., 2024).

Furthermore, temperament and personality traits such as irritability (Filippi et al., 2020; Kessel et al., 2016) and behavioral inhibition of a child (Filippi et al., 2020; Lahat et al., 2014; McDermott et al., 2009) have been found to interact with increased error monitoring, potentially leading to internalizing psychopathology. Lastly, there is preliminary evidence for sleep quality predicting anxiety symptoms in children and adolescents, but only for those with a larger ERN (Mehra et al., 2024).

However, it remains an open question whether the potential mediators and moderators of an individual and their environment also apply to the link between a trait-like decreased ERN and externalizing psychopathology. Most studies investigating mediators and moderators have focused predominantly on the internalizing spectrum (e.g., Banica et al., 2020; Meyer et al., 2017; Riesel et al., 2021). It is also important to note that some factors can play a double role, acting both as an early shaping factor forming an altered trait-like error monitoring system and as a mediator or moderator influencing the link between the ERN and psychopathology. For example, biological sex or gender can both shape the development of a trait-like altered error monitoring system early on (Fischer et al., 2016; Hill et al., 2018; Imburgio et al., 2020; Larson et al., 2011) and later act as a moderator influencing the connection between an increased ERN and anxiety (Moser et al., 2016). Furthermore, the respective associations of internalizing and externalizing psychopathology with altered error-related brain activity, may not be governed by a common underlying mechanism. Meta-analytical evidence indicates that individuals with externalizing symptoms exhibit more significant deficits in executive functioning—including impaired information processing and diminished behavioral performance—compared to unaffected individuals (Schoemaker et al., 2013; Yang et al., 2022). In contrast, this finding does not appear to hold for individuals with internalizing symptoms (Agnes Brunnekreef et al., 2007). Consequently, the cognitive aspect of the ERN may be more closely associated with externalizing symptoms, which often include behavioral deficits, while the affective aspect of the ERN may be more closely related to internalizing symptoms, which tend to be accompanied by intact behavioral performance.

Finally, both the internalizing and externalizing spectrums encompass a broad array of mental disorders and corresponding symptoms, prompting two principal inquiries. First, what underlying latent phenotypical dimensions connect these symptoms and disorders to one another? And second, which of these dimensions are specifically related to alterations

in the ERN? Within the internalizing spectrum, trait worry (Moser et al., 2013; Saunders & Inzlicht, 2020) and threat sensitivity (Hajcak & Foti, 2008; Heffer & Willoughby, 2021; Proudfit et al., 2013; Weinberg et al., 2016; Weinberg et al., 2012) have been discussed as latent dimensions underlying anxiety, obsession, and compulsions, and being linked to an increased ERN. However, due to the heterogeneous findings regarding ERN alterations in depression, it remains uncertain whether these dimensions also relate to depressive symptoms, or whether a different latent dimension might potentially link depression to altered ERN amplitudes (if there is a relationship between the ERN and depression at all).

Within the externalizing spectrum, impulsivity (Heffer & Willoughby, 2021; Hill et al., 2016; Overmeyer et al., 2021; Ruchsow et al., 2005; Taylor et al., 2018) and risk tolerance (Santesso & Segalowitz, 2009; Zheng et al., 2014) are considered potential latent dimensions that underlie substance use, deficits in attentional control, hyperactivity, as well as psychopathy, and are associated with a decrease in the ERN. However, at the intersection of internalizing (e.g., paranoia, anhedonia) and externalizing symptoms (e.g., hallucinations, disorganized behavior), the specific latent dimension that links a hypoactive error monitoring system with psychosis remains an open question.

Another area that warrants further investigation is the direction of effects. While altered error-related brain activity is traditionally viewed as a marker of neurobiological vulnerability, it is conceivable to hypothesize that clinical conditions could create a feedback loop affecting the error monitoring system. For instance, anxiety in return may facilitate the sensitivity of an individual to perceived threats and, consequently, to errors. In this case, even though altered error-related brain activity may represent vulnerability preceding the development of clinical symptoms, there could be mutually reinforcing effects between error-related brain activity and clinical symptoms, rather than a strictly unidirectional influence. Lastly, it is important to emphasize that the proposed integrative model reflects only one of several possible (neural) vulnerabilities—specifically, altered error-related brain activity—that contribute to the etiology of psychopathology.

Overall, the model serves as a descriptive framework that integrates current literature without delving into the specific mechanisms underlying the proposed associations. Nonetheless, it provides a significant foundation for future research in the area of altered error-related brain activity as a risk marker for both internalizing and externalizing psychopathology. The model facilitates the development and formulation of

testable hypotheses regarding neural vulnerabilities, their relationship with psychopathological phenotypes, and the potential interindividual variations in developmental trajectories. For example, one specific prediction of the model suggests that individuals exposed to a punitive parenting style during childhood and adolescence may develop a hyperactive error monitoring system, which increases the risk of internalizing psychopathological conditions (e.g., anxiety and obsessive-compulsive symptoms). This risk is particularly heightened in women and those facing adverse life conditions that induce significant stress. In summary, the integrative model can guide future research by informing study designs aimed at rigorously investigating each of the proposed predictions or even entire pathways. Additionally, it may inspire researchers to identify and examine the precise mechanisms behind these predictions, thereby progressively enhancing our understanding of the neurobiological etiology of psychopathological conditions.

4.7 Strengths and Limitations

Naturally, the research covered in this dissertation comes with certain strengths but also with considerable limitations. One of the main strengths of this dissertation is the overarching aim to explore specific associations linking neural indices of performance monitoring with psychopathology of the anxiety- and obsessive-compulsive spectrum. The recruitment of participants was guided by a transdiagnostic and dimensional approach, yielding large samples that encompass a wide spectrum of symptom severity and mental disorders (n = 151 clinical and n = 208 nonclinical participants across all three studies). Moreover, the research designs incorporated a diverse range of methodological strategies, including observational, experimental, and prospective elements. This included a randomized-controlled trial, cross-sectional group comparisons across various diagnostic categories, and long-term longitudinal follow-up assessments under real-life conditions, providing a comprehensive understanding of the topics studied. The research questions and hypotheses of all studies were theoretically and empirically derived, thereby justified based on the extensive body of available literature. Aligning with the principles of open science, the majority of research questions, hypotheses, and study designs were preregistered prior to data collection. Additionally, the employed explorative analyses allow for future confirmatory testing of new hypotheses on potential relationships and mechanisms. Furthermore, sample size estimations were based on a priori power analyses and a substantial amount of the presented data, alongside the corresponding analytical code, are publicly accessible in an online repository, enhancing transparency and reproducibility.

Ultimately, addressing the methodological heterogeneity in ERP scoring, all three studies focused on demonstrating the specificity of the observed effects across a multiverse of potential quantification strategies and employed measures with strong psychometric properties that reliably captured various clinical facets and neural processes among participants.

However, there are also several limitations to take into consideration. First and foremost, although the principle aim was to employ a transdiagnostic and dimensional approach in order to explore the link between neural indices of performance monitoring and the spectrum of anxiety and obsessive-compulsive symptoms, the presented research did not cover the full range of potentially relevant disorders. Incorporating generalized anxiety disorder, panic disorder, and agoraphobia in future investigations could significantly enrich our understanding of the entire transdiagnostic spectrum of anxiety and obsessive-compulsive disorders. Notably, regarding the participants with mental disorders included in the sample, disorders most frequently categorized within the internalizing spectrum in this dissertation were depression (42.9%), specific phobia (32.7%), SAD (29.5%), OCD (26.1%), and sleep disorders (12.8%). Furthermore, the study samples exclusively comprised adult participants, which precludes the generalization of these findings to younger populations. This is particularly significant as the initial onset of anxiety disorders typically occurs during adolescence and early adulthood (Lijster et al., 2017; Solmi et al., 2022).

Another significant limitation of this dissertation is its inability to delineate specific neurobiological mechanisms that modulate the differential trajectories from neural vulnerability to psychopathology, as this was not the primary aim of the included studies. While the data presented herein, in conjunction with existent literature, suggest that adverse life events, interpersonal stress, biological sex or gender, and temperament are important players in this dynamic, the precise neurobiological processes by which these factors influence an individual's performance monitoring system remain to be investigated by future research (e.g., the influence of sex hormones or cortisol). Moreover, similar to broader issues of psychophysiological research (Paul et al., 2022; Simmons et al., 2011), this dissertation is faced with the unresolved issue regarding the methodological multiverse of EEG preprocessing and ERP scoring. Although efforts were made to mitigate this by employing various commonly used scoring strategies (n = 4 forking paths in study 1, n = 5

in study 2), some of the results of this dissertation were partially sensitive to the chosen method. First studies have begun to address this fundamental methodological gap (Klawohn, Meyer, et al., 2020; Sandre et al., 2020); nonetheless, the field of anxiety research still lacks unequivocal, empirically supported guidelines for measuring and scoring electrophysiological indices of performance monitoring. Establishing such guidelines is imperative not only for reporting valid outcomes but also for improving the comparability of results across different studies.

4.8 Future Directions

Based on the research conducted in this dissertation and previous studies, several conclusions can be drawn for future research. Firstly, refining methodological recommendations for measuring the ERN would greatly enhance its potential as a valuable diagnostic and prognostic tool for individuals at risk of psychopathology. The identification of these individuals, particularly in clinic and school settings, could be highly useful in guiding decisions on how to prevent or mitigate the potential challenges of mental health in vulnerable populations (Lahat et al., 2014; Meyer, 2017; Meyer, Hajcak, et al., 2015; Meyer et al., 2021; Meyer, Nelson, et al., 2018). However, this requires standardized tasks alongside developmentally appropriate norms that are comparable to other established clinical outcomes, such as those obtained through clinical questionnaires. Fortunately, initial efforts toward establishing these standards have been made (Imburgio et al., 2020). Once these obstacles have been overcome, measuring an individual's EEG while performing a response conflict task could provide a time-efficient and cost-effective method to enhance existing clinical protocols. This approach would allow for more targeted preventive actions or treatments that consider the neural functioning of an individual, in addition to traditional factors used in developing treatment plans. Current exposure-based interventions used for the treatment of anxiety and obsessive-compulsive symptoms, such as CBT, have not demonstrated significant capacity to modify altered ERN amplitudes (Gorka et al., 2018; Hajcak et al., 2008; Kujawa et al., 2016; Ladouceur et al., 2018; Riesel et al., 2015). Thus, augmenting CBT with tailored interventions that directly target an enhanced ERN presents a promising approach to potentially increase the long-term efficacy of treatments for anxiety and obsessive-compulsive disorders.

Initial evidence suggests that an error sensitivity training can successfully reduce ERN amplitudes in nonclinical adults (Meyer et al., 2020); however, this training had no

significant impact on the ERN in a pediatric sample (Meyer et al., 2023). Similarly, the results concerning the efficacy of attentional bias modification are mixed, showing positive effects on the ERN in undergraduate students and OCD patients (Klawohn, Hajcak, et al., 2020; Nelson et al., 2015; Tan et al., 2021), but no significant changes in individuals with high trait anxiety (Carlson et al., 2021). Moreover, findings regarding the impact of emotional expressive writing interventions on the ERN (Schroder et al., 2018) have been inconsistent, with study 1 of this dissertation being unable to replicate previous results. These mixed findings highlight the urgent need for the development of robust, tailored interventions that can effectively modify neural indices associated with mental health risks. It is also vital to not only verify the principal efficacy of these interventions but also to examine their incremental effect and long-term sustainability of therapy outcomes, when used as augmentation strategies. Such efforts will ensure a more comprehensive approach of enhancing mental health treatment through neuroscientifically informed outcomes. Lastly, although the results of this dissertation lean towards viewing the CRN as a potential vulnerability marker similar to the ERN, suggesting that it could be a promising candidate for further interventions, it is premature to reach a definitive conclusion given the limited literature on the CRN. Further research is needed to explore the role of altered CRN amplitudes in internalizing and externalizing psychopathology.

Taken together, the research field emphasizes the need for future studies to investigate the specific neurobiological mechanisms underlying the associations between neural indices of performance monitoring across the anxiety and obsessive-compulsive spectrum and their role in the etiology of related disorders. In particular, exploring the influence of sex and stress hormones presents promising avenues for investigation, as this dissertation clearly highlights the interactions involving gender and stress response. Additionally, future research should encompass not only the recruitment of large samples that extend beyond traditional diagnostic boundaries but also the examination of a variety of neural indices and scoring methodologies. By demonstrating the specificity of potential effects, the validity of the findings concerning these relationships will be enhanced. This approach holds the potential to contribute to a deeper understanding of the neural mechanisms underlying these disorders and may lead to more effective, targeted interventions. Moreover, there is a compelling need to investigate the interplay of neurobiological markers with individual and environmental factors, utilizing more nuanced research approaches. These approaches should aim to precisely delineate the underlying

mechanisms leading to psychopathological conditions, considering different levels within a bio-psycho-social framework, as well as their chronological sequence during developmental stages across the lifespan. In exploring the etiology of vulnerability to these conditions, it is crucial to consider a multitude of contributing factors. This includes various neurobiological substrates (e.g., genetic predispositions and brain functioning), psychological outcomes (e.g., personality traits and coping mechanisms), and environmental influences (e.g., family context and parenting style). Investigating the interplay between these diverse factors holds promise for identifying individuals who are at heightened risk for developing psychopathological symptoms. It is also important to recognize that these vulnerability factors may evolve over time, shaped by the distinct developmental phases an individual experiences. Consequently, conducting longitudinal cohort studies will be vital for enhancing our understanding of these dynamics. Overall, achieving a comprehensive understanding of the intricate relationships among biological, psychological, and social factors in the development and progression of anxiety and obsessive-compulsive symptoms is essential. This understanding will inform the conception of targeted prevention and intervention strategies, ultimately leading to more effective support for individuals at risk.

4.9 Conclusion

In conclusion, this dissertation aimed at refining the understanding of neural performance monitoring within the anxiety and obsessive-compulsive spectrum by conducting observational, experimental, and prospective studies. Key findings indicate that traits dimensions like trait worry are significantly associated with larger ERN and CRN amplitudes, rather than state variations in symptoms such as state worry. This suggests that brain activity associated with performance monitoring reflects a more permanent, trait-like characteristic of an individual rather than fluctuating state conditions. Furthermore, enhanced neural performance monitoring was not indicative of a specific disorder type but was more broadly linked with the internalizing spectrum, predominantly encompassing anxiety and obsessive-compulsive, and depressive disorders. However, not only the clinical status of an individual, but also a family history of internalizing psychopathology is seemingly associated with heightened performance monitoring, supporting the notion of an increased ERN/CRN as electrophysiological risk markers. Additionally, the presented research highlights important moderators such as biological sex or gender, which influence the link between neural performance monitoring and anxiety, particularly noting stronger

associations of ERN/CRN amplitudes and trait worry in women. Moreover, adverse life events that pose risks to personal health, such as the COVID-19 pandemic, did not directly alter the relationship between neural indices of performance monitoring and internalizing symptoms. Instead, they mediated this relationship indirectly by increased perceived risk and stress levels, which in turn nonspecifically exacerbated anxiety, obsessive-compulsive, and depressive symptoms. This suggests a potential mechanism through which neural vulnerability—as indicated by altered electrophysiological indices of performance monitoring—translates into internalizing symptoms, thereby providing directions for future research.

Overall, the application of a transdiagnostic and dimensional perspective allows for a more nuanced understanding that extends beyond categorical diagnostic boundaries. This not only emphasizes the commonalities across different disorders but also points to the utility of the ERN and CRN as potential tools to identify individuals at risk, regardless of specific diagnostic labels. Consequently, this dissertation advocates a shift towards a more integrative model of understanding internalizing and externalizing psychopathology, incorporating a complex interplay of genetic, biological, cognitive, temperamental, and environmental factors shaping an individual's neural vulnerability. Additionally, the proposed model considers differential developmental trajectories of psychopathology by emphasizing the influence of various potential moderators and mediators such as biological sex, gender, or adverse and stressful life events, while also outlining latent dimensions underlying clinical phenotypes. However, while this work primarily addresses internalizing psychopathology, it remains open whether the identified modulating factors are also applicable to the externalizing or thought disorder spectrum. Future research should build upon these findings by refining the theoretical and empirical frameworks used to analyze neural indices of performance monitoring and their links to psychopathology. To this end, a developmental perspective that examines how biological, psychological, and environmental factors collectively influence neural performance monitoring in early life is essential, while also considering potential interactions with future developmental stages. Such a comprehensive understanding could facilitate the development of more targeted and personalized interventions that address both the neural and psychosocial factors contributing to development of anxiety and obsessive-compulsive symptoms.

References

Abi-Dargham, A., Moeller, S. J., Ali, F., DeLorenzo, C., Domschke, K., Horga, G., Jutla, A., Kotov, R., Paulus, M. P., Rubio, J. M., Sanacora, G., Veenstra-VanderWeele, J., & Krystal, J. H. (2023). Candidate biomarkers in psychiatric disorders: state of the field. *World Psychiatry, 22*(2), 236-262. https://doi.org/10.1002/wps.21078

Achenbach, T. M., Ivanova, M. Y., Rescorla, L. A., Turner, L. V., & Althoff, R. R. (2016). Internalizing/externalizing problems: review and recommendations for clinical and research applications. *Journal of the American Academy of Child & Adolescent Psychiatry, 55*(8), 647-656. https://doi.org/10.1016/j.jaac.2016.05.012

Agnes Brunnekreef, J., De Sonneville, L. M. J., Althaus, M., Minderaa, R. B., Oldehinkel, A. J., Verhulst, F. C., & Ormel, J. (2007). Information processing profiles of internalizing and externalizing behavior problems: evidence from a population-based sample of preadolescents. *Journal of Child Psychology and Psychiatry, 48*(2), 185-193. https://doi.org/10.1111/j.1469-7610.2006.01695.x

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR)*.

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.).

Andrews, V. H., & Borkovec, T. D. (1988). The differential effects of inductions of worry, somatic anxiety, and depression on emotional experience. *Journal of Behavior Therapy and Experimental Psychiatry, 19*(1), 21-26. https://doi.org/10.1016/0005-7916(88)90006-7

Anokhin, A. P., Golosheykin, S., & Heath, A. C. (2008). Heritability of frontal brain function related to action monitoring. *Psychophysiology, 45*(4), 524-534. https://doi.org/10.1111/j.1469-8986.2008.00664.x

Arch, J. J., & Craske, M. G. (2006). Mechanisms of mindfulness: Emotion regulation following a focused breathing induction. *Behaviour Research and Therapy, 44*(12), 1849-1858. https://doi.org/10.1016/j.brat.2005.12.007

Banica, I., Sandre, A., Shields, G. S., Slavich, G. M., & Weinberg, A. (2020). The error-related negativity (ERN) moderates the association between interpersonal stress and anxiety symptoms six months later. *International Journal of Psychophysiology, 153*, 27-36. https://doi.org/10.1016/j.ijpsycho.2020.03.006

Barlow, D. H. (1991). Disorders of emotion. *Psychological Inquiry, 2*(1), 58-71. https://doi.org/10.1207/s15327965pli0201_15

Bartz, J. A., & Hollander, E. (2006). Is obsessive–compulsive disorder an anxiety disorder? *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 30*(3), 338-352. https://doi.org/10.1016/j.pnpbp.2005.11.003

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory-II*. Psychological Corporation.

Beesdo-Baum, K., Zaudig, M., & Wittchen, H. (2019). *Strukturiertes Klinisches Interview für DSM-5-Störungen – Klinische Version* [The Structured Clinical Interview for DSM-5 Disorders – Clinical Version]. Hogrefe.

Bellaïche, L., Asthana, M., Ehlis, A.-C., Polak, T., & Herrmann, M. J. (2013). The modulation of error processing in the medial frontal cortex by transcranial direct current stimulation. *Neuroscience Journal, 2013*, 187692. https://doi.org/10.1155/2013/187692

Bellato, A., Norman, L., Idrees, I., Ogawa, C. Y., Waitt, A., Zuccolo, P. F., Tye, C., Radua, J., Groom, M. J., & Shephard, E. (2021). A systematic review and meta-analysis of altered electrophysiological markers of performance monitoring in Obsessive-Compulsive Disorder (OCD), Gilles de la Tourette Syndrome (GTS), Attention-Deficit/Hyperactivity disorder (ADHD) and Autism. *Neuroscience & Biobehavioral Reviews, 131*, 964-987. https://doi.org/10.1016/j.neubiorev.2021.10.018

Beltz, A. M., & Moser, J. S. (2020). Ovarian hormones: a long overlooked but critical contributor to cognitive brain structures and function. *Annals of the New York Academy of Sciences, 1464*(1), 156-180. https://doi.org/10.1111/nyas.14255

Bienvenu, O. J., Samuels, J. F., Wuyek, L. A., Liang, K. Y., Wang, Y., Grados, M. A., Cullen, B. A., Riddle, M. A., Greenberg, B. D., Rasmussen, S. A., Fyer, A. J., Pinto, A., Rauch, S. L., Pauls, D. L., McCracken, J. T., Piacentini, J., Murphy, D. L., Knowles, J. A., & Nestadt, G. (2012). Is obsessive–compulsive disorder an anxiety disorder, and what, if any, are spectrum conditions? A family study perspective. *Psychological Medicine, 42*(1), 1-13. https://doi.org/10.1017/S0033291711000742

Boehnke, K., Schwartz, S., Stromberg, C., & Sagiv, L. (1998). The structure and dynamics of worry: Theory, measurement, and cross-national replications. *Journal of Personality, 66*(5), 745-782. https://doi.org/10.1111/1467-6494.00031

Boen, R., Quintana, D. S., Ladouceur, C. D., & Tamnes, C. K. (2022). Age-related differences in the error-related negativity and error positivity in children and adolescents are moderated by sample and methodological characteristics: A meta-analysis. *Psychophysiology, 59*(6), e14003. https://doi.org/10.1111/psyp.14003

Borkovec, T. D., & Inz, J. (1990). The nature of worry in generalized anxiety disorder: A predominance of thought activity. *Behaviour Research and Therapy, 28*(2), 153-158. https://doi.org/10.1016/0005-7967(90)90027-G

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*(3), 624-652. https://doi.org/10.1037/0033-295X.108.3.624

Brázdil, M., Roman, R., Daniel, P., & Rektor, I. (2005). Intracerebral error-related negativity in a simple Go/NoGo task. *Journal of Psychophysiology, 19*(4), 244-255. https://doi.org/10.1027/0269-8803.19.4.244

Brooker, R. J., & Buss, K. A. (2014a). Harsh parenting and fearfulness in toddlerhood interact to predict amplitudes of preschool error-related negativity. *Developmental Cognitive Neuroscience, 9*, 148-159. https://doi.org/10.1016/j.dcn.2014.03.001

Brooker, R. J., & Buss, K. A. (2014b). Toddler fearfulness is linked to individual differences in error-related negativity during preschool. *Developmental Neuropsychology, 39*(1), 1-8. https://doi.org/10.1080/87565641.2013.826661

Brooker, R. J., Buss, K. A., & Dennis, T. A. (2011). Error-monitoring brain activity is associated with affective behaviors in young children. *Developmental Cognitive Neuroscience, 1*(2), 141-152. https://doi.org/10.1016/j.dcn.2010.12.002

Burdwood, E. N., Infantolino, Z. P., Crocker, L. D., Spielberg, J. M., Banich, M. T., Miller, G. A., & Heller, W. (2016). Resting-state functional connectivity differentiates anxious apprehension and anxious arousal. *Psychophysiology, 53*(10), 1451-1459. https://doi.org/10.1111/psyp.12696

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4*(6), 215-222. https://doi.org/10.1016/S1364-6613(00)01483-2

Bushnell, M. C., Čeko, M., & Low, L. A. (2013). Cognitive and emotional control of pain and its disruption in chronic pain. *Nature Reviews Neuroscience, 14*(7), 502-511. https://doi.org/10.1038/nrn3516

Buzzell, G. A., Troller-Renfree, S. V., Barker, T. V., Bowman, L. C., Chronis-Tuscano, A., Henderson, H. A., Kagan, J., Pine, D. S., & Fox, N. A. (2017). A neurobehavioral mechanism linking behaviorally inhibited temperament and later adolescent social anxiety. *Journal of the American Academy of Child & Adolescent Psychiatry, 56*(12), 1097-1105. https://doi.org/10.1016/j.jaac.2017.10.007

Carlson, J. M., Fang, L., & Andrzejewski, J. A. (2021). No change in electrocortical measures of performance monitoring in high trait anxious individuals following multi-session attention bias modification training. *Neuroimage: Reports, 1*(4), 100067. https://doi.org/10.1016/j.ynirp.2021.100067

Carrasco, M., Harbin, S. M., Nienhuis, J. K., Fitzgerald, K. D., Gehring, W. J., & Hanna, G. L. (2013). Increased error-related brain activity in youth with obsessive-compulsive disorder and unaffected siblings. *Depression and Anxiety, 30*(1), 39-46. https://doi.org/10.1002/da.22035

Carrasco, M., Hong, C., Nienhuis, J. K., Harbin, S. M., Fitzgerald, K. D., Gehring, W. J., & Hanna, G. L. (2013). Increased error-related brain activity in youth with obsessive-compulsive disorder and other anxiety disorders. *Neuroscience Letters, 541*, 214-218. https://doi.org/10.1016/j.neulet.2013.02.017

Carsten, H. P., Härpfer, K., Nelson, B. D., Kathmann, N., & Riesel, A. (2023). Don't worry, it won't be fine. Contributions of worry and anxious arousal to startle responses and event-related potentials in threat anticipation. *Cognitive, Affective, & Behavioral Neuroscience, 23*(4), 1141-1159. https://doi.org/10.3758/s13415-023-01094-4

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science, 280*(5364), 747-749. https://doi.org/10.1126/science.280.5364.747

Castagna, P. J., Roye, S., Calamia, M., Owens-French, J., Davis, T. E., & Greening, S. G. (2018). Parsing the neural correlates of anxious apprehension and anxious arousal in the grey-matter of healthy youth. *Brain Imaging and Behavior, 12*(4), 1084-1098. https://doi.org/10.1007/s11682-017-9772-1

Cavanagh, J. F., & Shackman, A. J. (2015). Frontal midline theta reflects anxiety and cognitive control: Meta-analytic evidence. *Journal of Physiology-Paris, 109*(1), 3-15. https://doi.org/10.1016/j.jphysparis.2014.04.003

Chen, Z., & Itier, R. J. (2024). No association between error-related ERPs and trait anxiety in a nonclinical sample: Convergence across analytical methods including mass-univariate statistics. *Psychophysiology*, e14645. https://doi.org/10.1111/psyp.14645

Chong, L. J., Mirzadegan, I. A., & Meyer, A. (2020). The association between parenting and the error-related negativity across childhood and adolescence. *Developmental Cognitive Neuroscience, 45*, 100852. https://doi.org/10.1016/j.dcn.2020.100852

Clayson, P. E. (2024). The psychometric upgrade psychophysiology needs. *Psychophysiology, 61*(3), e14522. https://doi.org/10.1111/psyp.14522

Clayson, P. E., McDonald, J. B., Park, B., Holbrook, A., Baldwin, S. A., Riesel, A., & Larson, M. J. (2023). Registered replication report of the construct validity of the error-related negativity (ERN): A multi-site study of task-specific ERN correlations with internalizing and externalizing symptoms. *Psychophysiology*, e14496. https://doi.org/10.1111/psyp.14496

Coleman, J. R., Watson, J. M., & Strayer, D. L. (2018). Working memory capacity and task goals modulate error-related ERPs. *Psychophysiology, 55*(3), e12805. https://doi.org/10.1111/psyp.12805

Colino, F. L., Howse, H., Norton, A., Trska, R., Pluta, A., Luehr, S. J. C., Handy, T. C., & Krigolson, O. E. (2017). Older adults display diminished error processing and response in a continuous tracking task. *Psychophysiology, 54*(11), 1706-1713. https://doi.org/10.1111/psyp.12907

Compton, R. J., Shudrenko, D., Ng, E., Mann, K., & Turdukulov, E. (2024). Adversity and error-monitoring: Effects of emotional context. *Psychophysiology*, e14644. https://doi.org/10.1111/psyp.14644

Cox, B. J., Clara, I. P., Hills, A. L., & Sareen, J. (2010). Obsessive-compulsive disorder and the underlying structure of anxiety disorders in a nationally representative sample: Confirmatory factor analytic findings from the German health survey. *Journal of Anxiety Disorders, 24*(1), 30-33. https://doi.org/10.1016/j.janxdis.2009.08.003

Craske, M., Wittchen, H. U., Bogels, S., Stein, M., Adrews, G., & Lebeu, R. (2013). *Severity Measure for Specific Phobia—Adult*. American Psychiatric Association.

Cuthbert, B. N. (2014). Translating intermediate phenotypes to psychopathology: The NIMH Research Domain Criteria. *Psychophysiology, 51*(12), 1205-1206. https://doi.org/10.1111/psyp.12342

Davies, P. L., Segalowitz, S. J., & Gavin, W. J. (2004a). Development of error-monitoring event-related potentials in adolescents. *Annals of the New York Academy of Sciences, 1021*(1), 324-328. https://doi.org/10.1196/annals.1308.039

Davies, P. L., Segalowitz, S. J., & Gavin, W. J. (2004b). Development of response-monitoring ERPs in 7- to 25-year-olds. *Developmental Neuropsychology, 25*(3), 355-376. https://doi.org/10.1207/s15326942dn2503_6

de Bruijn, E. R. A., Hulstijn, W., Verkes, R. J., Ruigt, G. S. F., & Sabbe, B. G. C. (2004). Drug-induced stimulation and suppression of action monitoring in healthy volunteers. *Psychopharmacology, 177*(1-2), 151-160. https://doi.org/10.1007/s00213-004-1915-6

de Visser, L., van der Knaap, L. J., van de Loo, A. J. A. E., van der Weerd, C. M. M., Ohl, F., & van den Bos, R. (2010). Trait anxiety affects decision-making differently in healthy men and women: Towards gender-specific endophenotypes of anxiety. *Neuropsychologia, 48*(6), 1598-1606. https://doi.org/10.1016/j.neuropsychologia.2010.01.027

Debener, S., Ullsperger, M., Siegel, M., Fiehler, K., von Cramon, D. Y., & Engel, A. K. (2005). Trial-by-trial coupling of concurrent electroencephalogram and functional magnetic resonance imaging identifies the dynamics of performance monitoring. *The Journal of Neuroscience, 25*(50), 11730-11737. https://doi.org/10.1523/JNEUROSCI.3286-05.2005

Debnath, R., Troller-Renfree, S. V., Zeanah, C. H., Nelson, C. A., & Fox, N. A. (2023). Effects of institutional rearing and foster care intervention on error monitoring and externalizing behaviors in adolescence. *Developmental Cognitive Neuroscience, 63*, 101295. https://doi.org/10.1016/j.dcn.2023.101295

Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error detection and compensation. *Psychological Science, 5*(5), 303-305. https://doi.org/10.1111/j.1467-9280.1994.tb00630.x

Eaton, N. R., Bringmann, L. F., Elmer, T., Fried, E. I., Forbes, M. K., Greene, A. L., Krueger, R. F., Kotov, R., McGorry, P. D., Mei, C., & Waszczuk, M. A. (2023). A review of approaches and models in psychopathology conceptualization research. *Nature Reviews Psychology, 2*(10), 622-636. https://doi.org/10.1038/s44159-023-00218-4

Edwards, B. G., Calhoun, V. D., & Kiehl, K. A. (2012). Joint ICA of ERP and fMRI during error-monitoring. *NeuroImage, 59*(2), 1896-1903. https://doi.org/10.1016/j.neuroimage.2011.08.088

Emeric, E. E., Brown, J. W., Leslie, M., Pouget, P., Stuphorn, V., & Schall, J. D. (2008). Performance monitoring local field potentials in the medial frontal cortex of primates: anterior cingulate cortex. *Journal of Neurophysiology, 99*(2), 759-772. https://doi.org/10.1152/jn.00896.2006

Endrass, T., Reuter, B., & Kathmann, N. (2007). ERP correlates of conscious error recognition: Aware and unaware errors in an antisaccade task. *European Journal of Neuroscience, 26*(6), 1714-1720. https://doi.org/10.1111/j.1460-9568.2007.05785.x

Endrass, T., Riesel, A., Kathmann, N., & Buhlmann, U. (2014). Performance monitoring in obsessive–compulsive disorder and social anxiety disorder. *Journal of Abnormal Psychology, 123*(4), 705-714. https://doi.org/10.1037/abn0000012

Eppinger, B., Kray, J., Mock, B., & Mecklinger, A. (2008). Better or worse than expected? Aging, learning, and the ERN. *Neuropsychologia, 46*(2), 521-539. https://doi.org/10.1016/j.neuropsychologia.2007.09.001

Euser, A. S., Evans, B. E., Greaves-Lord, K., Huizink, A. C., & Franken, I. H. A. (2013). Diminished error-related brain activity as a promising endophenotype for substance-use disorders: evidence from high-risk offspring. *Addiction Biology, 18*(6), 970-984. https://doi.org/10.1111/adb.12002

Fairbairn, C. E., Kang, D., & Federmeier, K. D. (2021). Alcohol and neural dynamics: a meta-analysis of acute alcohol effects on event-related brain potentials. *Biological Psychiatry, 89*(10), 990-1000. https://doi.org/10.1016/j.biopsych.2020.11.024

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components: II Error processing in choice reaction tasks. *Electroencephalography & Clinical Neurophysiology, 78*(6), 447-455. https://doi.org/10.1016/0013-4694(91)90062-9

Falkenstein, M., Hoormann, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: A tutorial. *Biological Psychology, 51*(2-3), 87-107. https://doi.org/10.1016/S0301-0511(99)00031-9

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (2001). Changes of error-related ERPs with age. *Experimental Brain Research, 138*(2), 258-262. https://doi.org/10.1007/s002210100712

Filippi, C. A., Subar, A. R., Sachs, J. F., Kircanski, K., Buzzell, G., Pagliaccio, D., Abend, R., Fox, N. A., Leibenluft, E., & Pine, D. S. (2020). Developmental pathways to social anxiety and irritability: The role of the ERN. *Development and Psychopathology, 32*(3), 897-907. https://doi.org/10.1017/S0954579419001329

First, M., Williams, J., Karg, R., & Spitzer, R. (2016). *Structured Clinical Interview for DSM-5 Disorders, Clinician Version (SCID-5-CV)*. American Psychiatric Association.

Fischer, A. G., Danielmeier, C., Villringer, A., Klein, T. A., & Ullsperger, M. (2016). Gender influences on brain responses to errors and post-error adjustments. *Scientific Reports, 6*(1), 24435. https://doi.org/10.1038/srep24435

Foa, E. B., Huppert, J. D., Leiberg, S., Langner, R., Kichic, R., Hajcak, G., & Salkovskis, P. M. (2002). The Obsessive-Compulsive Inventory: Development and validation of a short version. *Psychological Assessment, 14*(4), 485-496. https://doi.org/10.1037/1040-3590.14.4.485

Forbes, M. K., Neo, B., Nezami, O. M., Fried, E. I., Faure, K., Michelsen, B., Twose, M., & Dras, M. (2024). Elemental psychopathology: distilling constituent symptoms and patterns of repetition in the diagnostic criteria of the DSM-5. *Psychological Medicine, 54*(5), 886-894. https://doi.org/10.1017/S0033291723002544

Geburek, A. J., Rist, F., Gediga, G., Stroux, D., & Pedersen, A. (2013). Electrophysiological indices of error monitoring in juvenile and adult attention deficit hyperactivity disorder (ADHD)—A meta-analytic appraisal. *International Journal of Psychophysiology, 87*(3), 349-362. https://doi.org/10.1016/j.ijpsycho.2012.08.006

Gehring, W. J., Goss, B., Coles, M. G., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science, 4*(6), 385-390. https://doi.org/10.1111/j.1467-9280.1993.tb00586.x

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (2018). The error-related negativity. *Perspectives on Psychological Science, 13*(2), 200-204. https://doi.org/10.1177/1745691617715310

Gehring, W. J., & Knight, R. T. (2000). Prefrontal-cingulate interactions in action monitoring. *Nature Neuroscience, 3*(5), 516-520. https://doi.org/10.1038/74899

Gilbertson, H., Fang, L., Andrzejewski, J. A., & Carlson, J. M. (2021). Dorsal anterior cingulate cortex intrinsic functional connectivity linked to electrocortical measures of error monitoring. *Psychophysiology*, e13794. https://doi.org/10.1111/psyp.13794

Glöckner-Rist, A., & Rist, F. (2014). Deutsche Version des Penn State Worry Questionnaire (PSWQ-d) [German version of the Penn State Worry Questionnaire]. *Zusammenstellung sozialwissenschaftlicher Items und Skalen*, 1-26. https://doi.org/10.6102/zis219

Godlove, D. C., Emeric, E. E., Segovis, C. M., Young, M. S., Schall, J. D., & Woodman, G. F. (2011). Event-related potentials elicited by errors during the stop-signal Task. I. Macaque Monkeys. *The Journal of Neuroscience, 31*(44), 15640. https://doi.org/10.1523/JNEUROSCI.3349-11.2011

Gönner, S., Leonhart, R., & Ecker, W. (2007). Das Zwangsinventar OCI-R - Die deutsche Version des Obsessive-Compulsive Inventory-Revised [The German Version of the Obsessive-Compulsive Inventory-Revised]. *Psychotherapie Psychosomatik Medizinische Psychologie, 57*(09/10), 395-404. https://doi.org/10.1055/s-2007-970894

Gorday, J. Y., & Meyer, A. (2018). Linking puberty and error-monitoring: Relationships between self-reported pubertal stages, pubertal hormones, and the error-related negativity in a large sample of children and adolescents. *Developmental Psychobiology, 60*(4), 483-490. https://doi.org/10.1002/dev.21625

Gorka, S. M., Burkhouse, K. L., Klumpp, H., Kennedy, A. E., Afshar, K., Francis, J., Ajilore, O., Mariouw, S., Craske, M. G., Langenecker, S., Shankman, S. A., & Phan, K. L. (2018). Error-related brain activity as a treatment moderator and index of symptom change during cognitive-behavioral therapy or selective serotonin reuptake inhibitors. *Neuropsychopharmacology, 43*(6), 1355-1363. https://doi.org/10.1038/npp.2017.289

Gorka, S. M., MacNamara, A., Aase, D. M., Proescher, E., Greenstein, J. E., Walters, R., Passi, H., Babione, J. M., Levy, D. M., Kennedy, A. E., DiGangi, J. A., Rabinak, C. A., Schroth, C., Afshar, K., Fitzgerald, J., Hajcak, G., & Phan, K. L. (2016). Impact of alcohol use disorder comorbidity on defensive reactivity to errors in veterans with posttraumatic stress disorder. *Psychology of Addictive Behaviors, 30*(7), 733-742. https://doi.org/10.1037/adb0000196

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *The American Journal of Psychiatry, 160*(4), 636-645. https://doi.org/10.1176/appi.ajp.160.4.636

Gulpers, B. J. A., Oude Voshaar, R. C., van Boxtel, M. P. J., Verhey, F. R. J., & Köhler, S. (2019). Anxiety as a risk factor for cognitive decline: a 12-year follow-up cohort study. *The American Journal of Geriatric Psychiatry, 27*(1), 42-52. https://doi.org/10.1016/j.jagp.2018.09.006

Gulpers, B. J. A., Verhey, F. R. J., Eussen, S. J. P. M., Schram, M. T., de Galan, B. E., van Boxtel, M. P. J., Stehouwer, C. D. A., & Köhler, S. (2022). Anxiety and cognitive functioning in the Maastricht study: A cross-sectional population study. *Journal of Affective Disorders, 319*, 570-579. https://doi.org/10.1016/j.jad.2022.09.072

Hajcak, G. (2012). What we've learned from mistakes: Insights from error-related brain activity. *Current Directions in Psychological Science, 21*(2), 101-106. https://doi.org/10.1177/0963721412436809

Hajcak, G., & Foti, D. (2008). Errors are aversive: Defensive motivation and the error-related negativity. *Psychological Science, 19*(2), 103-108. https://doi.org/10.1111/j.1467-9280.2008.02053.x

Hajcak, G., Franklin, M. E., Foa, E. B., & Simons, R. F. (2008). Increased error-related brain activity in pediatric obsessive-compulsive disorder before and after treatment. *American Journal of Psychiatry, 165*(1), 116-123. https://doi.org/10.1176/appi.ajp.2007.07010143

Hajcak, G., Klawohn, J., & Meyer, A. (2019). The utility of event-related potentials in clinical psychology. *Annual Review of Clinical Psychology, 15*(1), 71-95. https://doi.org/10.1146/annurev-clinpsy-050718-095457

Hajcak, G., McDonald, N., & Simons, R. F. (2003). Anxiety and error-related brain activity. *Biological Psychology, 64*(1-2), 77-90. https://doi.org/10.1016/S0301-0511(03)00103-0

Härpfer, K., Carsten, H. P., Kausche, F. M., & Riesel, A. (in revision). Enhanced performance monitoring as a transdiagnostic risk marker of the anxiety and obsessive-compulsive spectrum: The role of disorder category, clinical status, family risk, and anxiety dimensions. *Depression and Anxiety*.

Härpfer, K., Carsten, H. P., Löwisch, K., Westermann, N., & Riesel, A. (2022). Disentangling the effects of trait and state worry on error-related brain activity: Results from a randomized controlled trial using worry manipulations. *Psychophysiology, 59*(9), e14055. https://doi.org/10.1111/psyp.14055

Härpfer, K., Carsten, H. P., Spychalski, D., Kathmann, N., & Riesel, A. (2020). Were we erring? The impact of worry and arousal on error-related negativity in a non-clinical sample. *Psychophysiology, 57*(11), e13661. https://doi.org/10.1111/psyp.13661

Hautzinger, M., Keller, F., & Kühner, C. (2006). *Beck Depressions-Inventar (BDI-II)*. Harcourt Test Services.

Heffer, T., & Willoughby, T. (2021). A person-centered examination of emotion dysregulation, sensitivity to threat, and impulsivity among children and adolescents: An ERP study. *Developmental Cognitive Neuroscience, 47*, 100900. https://doi.org/10.1016/j.dcn.2020.100900

Hill, K. E., Ait Oumeziane, B., Novak, K. D., Rollock, D., & Foti, D. (2018). Variation in reward- and error-related neural measures attributable to age, gender, race, and ethnicity. *International Journal of Psychophysiology, 132*, 353-364. https://doi.org/10.1016/j.ijpsycho.2017.12.009

Hill, K. E., Samuel, D. B., & Foti, D. (2016). Contextualizing individual differences in error monitoring: Links with impulsivity, negative affect, and conscientiousness. *Psychophysiology, 53*(8), 1143-1153. https://doi.org/10.1111/psyp.12671

Hochman, E. Y., Vaidya, A. R., & Fellows, L. K. (2014). Evidence for a role for the dorsal anterior cingulate cortex in disengaging from an incorrect action. *PLOS ONE, 9*(6), e101126. https://doi.org/10.1371/journal.pone.0101126

Hoffmann, S., & Falkenstein, M. (2011). Aging and error processing: Age related increase in the variability of the error-negativity is not accompanied by increase in response variability. *PLOS ONE, 6*(2), e17482. https://doi.org/10.1371/journal.pone.0017482

Hollander, E., Braun, A., & Simeon, D. (2008). Should OCD leave the anxiety disorders in DSM-V? The case for obsessive compulsive-related disorders. *Depression and Anxiety, 25*(4), 317-329. https://doi.org/10.1002/da.20500

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review, 109*(4), 679-709. https://doi.org/10.1037/0033-295X.109.4.679

Hopfinger, J. B., Kiehl, K. A., & Liddle, P. F. (2000). Error processing and the rostral anterior cingulate: An event-related fMRI study. *Psychophysiology, 37*(2), 216-223. https://doi.org/10.1111/1469-8986.3720216

Iannaccone, R., Hauser, T. U., Staempfli, P., Walitza, S., Brandeis, D., & Brem, S. (2015). Conflict monitoring and error processing: New insights from simultaneous EEG–fMRI. *NeuroImage, 105*, 395-407. https://doi.org/10.1016/j.neuroimage.2014.10.028

Imburgio, M. J., Banica, I., Hill, K. E., Weinberg, A., Foti, D., & MacNamara, A. (2020). Establishing norms for error-related brain activity during the arrow Flanker task among young adults. *NeuroImage, 213*, 116694. https://doi.org/10.1016/j.neuroimage.2020.116694

Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K., Sanislow, C., & Wang, P. (2010). Research domain criteria (RDoC): Toward a new classification framework for research on mental disorders. *The American Journal of Psychiatry, 167*(7), 748-751. https://doi.org/10.1176/appi.ajp.2010.09091379

Jackson, F., Nelson, B. D., & Hajcak, G. (2016). The uncertainty of errors: Intolerance of uncertainty is associated with error-related brain activity. *Biological Psychology, 113*, 52-58. https://doi.org/10.1016/j.biopsycho.2015.11.007

Jackson, F., Nelson, B. D., & Proudfit, G. H. (2015). In an uncertain world, errors are more aversive: Evidence from the error-related negativity. *Emotion, 15*(1), 12-16. https://doi.org/10.1037/emo0000020

Kaiser, A., Aggensteiner, P.-M., Baumeister, S., Holz, N. E., Banaschewski, T., & Brandeis, D. (2020). Earlier versus later cognitive event-related potentials (ERPs) in attention-deficit/hyperactivity disorder (ADHD): A meta-analysis. *Neuroscience & Biobehavioral Reviews, 112*, 117-134. https://doi.org/10.1016/j.neubiorev.2020.01.019

Kaufman, J., & Charney, D. (2000). Comorbidity of mood and anxiety disorders. *Depression and Anxiety, 12*(S1), 69-76. https://doi.org/10.1002/1520-6394(2000)12:1+<69::AID-DA9>3.0.CO;2-K

Kessel, E. M., Meyer, A., Hajcak, G., Dougherty, L. R., Torpey-Newman, D. C., Carlson, G. A., & Klein, D. N. (2016). Transdiagnostic factors and pathways to multifinality: The error-related negativity predicts whether preschool irritability is associated with internalizing versus externalizing symptoms at age 9. *Development and Psychopathology, 28*(4), 913-926. https://doi.org/10.1017/S0954579416000626

Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-Month DSM-IV disorders in the national comorbidity survey

replication. *Archives of General Psychiatry, 62*(6), 617-627. https://doi.org/10.1001/archpsyc.62.6.617

Klawohn, J., Endrass, T., Preuss, J., Riesel, A., & Kathmann, N. (2016). Modulation of hyperactive error signals in obsessive–compulsive disorder by dual-task demands. *Journal of Abnormal Psychology, 125*(2), 292-298. https://doi.org/10.1037/abn0000134

Klawohn, J., Hajcak, G., Amir, N., Kathmann, N., & Riesel, A. (2020). Application of attentional bias modification training to modulate hyperactive error-monitoring in OCD. *International Journal of Psychophysiology, 156*, 79-86. https://doi.org/10.1016/j.ijpsycho.2020.07.005

Klawohn, J., Meyer, A., Weinberg, A., & Hajcak, G. (2020). Methodological choices in event-related potential (ERP) research and their impact on internal consistency reliability and individual differences: An examination of the error-related negativity (ERN) and anxiety. *Journal of Abnormal Psychology, 129*(1), 29-37. https://doi.org/10.1037/abn0000458

Knyazev, G. G., Schutter, D. J. L. G., & van Honk, J. (2006). Anxious apprehension increases coupling of delta and beta oscillations. *International Journal of Psychophysiology, 61*(2), 283-287. https://doi.org/10.1016/j.ijpsycho.2005.12.003

Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., Brown, T. A., Carpenter, W. T., Caspi, A., Clark, L. A., Eaton, N. R., Forbes, M. K., Forbush, K. T., Goldberg, D., Hasin, D., Hyman, S. E., Ivanova, M. Y., Lynam, D. R., Markon, K., Miller, J. D., Moffitt, T. E., Morey, L. C., Mullins-Sweatt, S. N., Ormel, J., Patrick, C. J., Regier, D. A., Rescorla, L., Ruggero, C. J., Samuel, D. B., Sellbom, M., Simms, L. J., Skodol, A. E., Slade, T., South, S. C., Tackett, J. L., Waldman, I. D., Waszczuk, M. A., Widiger, T. A., Wright, A. G. C., & Zimmerman, M. (2017). The Hierarchical Taxonomy of Psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology, 126*(4), 454-477. https://doi.org/10.1037/abn0000258

Kozak, M. J., & Cuthbert, B. N. (2016). The NIMH Research Domain Criteria Initiative: Background, issues, and pragmatics. *Psychophysiology, 53*(3), 286-297. https://doi.org/10.1111/psyp.12518

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry, 56*(10), 921-926. https://doi.org/10.1001/archpsyc.56.10.921

Kujawa, A., Weinberg, A., Bunford, N., Fitzgerald, K. D., Hanna, G. L., Monk, C. S., Kennedy, A. E., Klumpp, H., Hajcak, G., & Phan, K. L. (2016). Error-related brain activity in youth and young adults before and after treatment for generalized or social anxiety disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 71*, 162-168. https://doi.org/10.1016/j.pnpbp.2016.07.010

Ladouceur, C. D., Tan, P. Z., Sharma, V., Bylsma, L. M., Silk, J. S., Siegle, G. J., Forbes, E. E., McMakin, D. L., Dahl, R. E., Kendall, P. C., Mannarino, A., & Ryan, N. D. (2018). Error-related brain activity in pediatric anxiety disorders remains elevated

following individual therapy: A randomized clinical trial. *Journal of Child Psychology and Psychiatry, 59*(11), 1152-1161. https://doi.org/10.1111/jcpp.12900

Lahat, A., Lamm, C., Chronis-Tuscano, A., Pine, D. S., Henderson, H. A., & Fox, N. A. (2014). Early behavioral inhibition and increased error monitoring predict later social phobia symptoms in childhood. *Journal of the American Academy of Child & Adolescent Psychiatry, 53*(4), 447-455. https://doi.org/10.1016/j.jaac.2013.12.019

Larson, M. J., Baldwin, S. A., Good, D. A., & Fair, J. E. (2010). Temporal stability of the error-related negativity (ERN) and post-error positivity (Pe): The role of number of trials. *Psychophysiology, 47*(6), 1167-1171. https://doi.org/10.1111/j.1469-8986.2010.01022.x

Larson, M. J., & Clayson, P. E. (2011). The relationship between cognitive performance and electrophysiological indices of performance monitoring. *Cognitive, Affective, & Behavioral Neuroscience, 11*(2), 159-171. https://doi.org/10.3758/s13415-010-0018-6

Larson, M. J., Clayson, P. E., Keith, C. M., Hunt, I. J., Hedges, D. W., Nielsen, B. L., & Call, V. R. A. (2016). Cognitive control adjustments in healthy older and younger adults: Conflict adaptation, the error-related negativity (ERN), and evidence of generalized decline with age. *Biological Psychology, 115*, 50-63. https://doi.org/10.1016/j.biopsycho.2016.01.008

Larson, M. J., South, M., & Clayson, P. E. (2011). Sex differences in error-related performance monitoring. *NeuroReport, 22*(1), 44-48. https://doi.org/10.1097/WNR.0b013e3283427403

Laux, L., Glanzmann, P., Schaffner, P., & Spielberger, C. D. (1981). *Das State-Trait-Angstinventar (STAI)* [The State-Trait Anxiety Inventory (STAI)]. Beltz.

Liebowitz, M. R. (1987). Social phobia. *Modern Problems of Pharmacopsychiatry, 22*, 141-173. https://doi.org/10.1159/000414022

Lijster, J. M., Dierckx, B., Utens, E. M., Verhulst, F. C., Zieldorff, C., Dieleman, G. C., & Legerstee, J. S. (2017). The age of onset of anxiety disorders: a meta-analysis. *The Canadian Journal of Psychiatry, 62*(4), 237-246. https://doi.org/10.1177/0706743716640757

Lin, Y., Moran, T. P., Schroder, H. S., & Moser, J. S. (2015). The role of hand of error and stimulus orientation in the relationship between worry and error-related brain activity: Implications for theory and practice. *Psychophysiology, 52*(10), 1281-1292. https://doi.org/10.1111/psyp.12470

Lindert, J., Paul, K. C., Lachman, M. E., Ritz, B., & Seeman, T. E. (2021). Depression-, anxiety-, and anger and cognitive functions: Findings from a longitudinal prospective study. *Frontiers in Psychiatry, 12*, 665742. https://doi.org/10.3389/fpsyt.2021.665742

Loman, M. M., Johnson, A. E., Westerlund, A., Pollak, S. D., Nelson, C. A., & Gunnar, M. R. (2013). The effect of early deprivation on executive attention in middle childhood. *Journal of Child Psychology and Psychiatry, 54*(1), 37-45. https://doi.org/10.1111/j.1469-7610.2012.02602.x

Lumme, V., Aalto, S., Ilonen, T., Någren, K., & Hietala, J. (2007). Dopamine D2/D3 receptor binding in the anterior cingulate cortex and executive functioning. *Psychiatry Research: Neuroimaging, 156*(1), 69-74. https://doi.org/10.1016/j.pscychresns.2006.12.012

Lutz, M. C., Kok, R., Verveer, I., Malbec, M., Koot, S., van Lier, P. A. C., & Franken, I. H. A. (2021). Diminished error-related negativity and error positivity in children and adults with externalizing problems and disorders: a meta-analysis on error processing. *Journal of Psychiatry and Neuroscience, 46*(6), E615. https://doi.org/10.1503/jpn.200031

Macedo, I., Pasion, R., Barbosa, F., & Ferreira-Santos, F. (2021). A dimensional approach to the neuronal correlates of anxiety, depression, and perfectionism: A transdiagnostic dissociation of error-related brain activity. *Behavioural Brain Research, 408*, 113271. https://doi.org/10.1016/j.bbr.2021.113271

Maier, M. E., Di Gregorio, F., Muricchio, T., & Di Pellegrino, G. (2015). Impaired rapid error monitoring but intact error signaling following rostral anterior cingulate cortex lesions in humans. *Frontiers in Human Neuroscience, 9*(339). https://doi.org/10.3389/fnhum.2015.00339

Manoach, D. S., & Agam, Y. (2013). Neural markers of errors as endophenotypes in neuropsychiatric disorders. *Frontiers in Human Neuroscience, 7*, 350. https://doi.org/10.3389/fnhum.2013.00350

Maron, E., Lan, C.-C., & Nutt, D. (2018). Imaging and genetic approaches to inform biomarkers for anxiety disorders, obsessive–compulsive disorders, and PTSD. In J. Pratt & J. Hall (Eds.), *Biomarkers in Psychiatry* (pp. 219-292). Springer. https://doi.org/10.1007/7854_2018_49

Martin, E. A., McCleery, A., Moore, M. M., Wynn, J. K., Green, M. F., & Horan, W. P. (2018). ERP indices of performance monitoring and feedback processing in psychosis: A meta-analysis. *International Journal of Psychophysiology, 132*, 365-378. https://doi.org/10.1016/j.ijpsycho.2018.08.004

Mathews, C. A., Perez, V. B., Delucchi, K. L., & Mathalon, D. H. (2012). Error-related negativity in individuals with obsessive–compulsive symptoms: Toward an understanding of hoarding behaviors. *Biological Psychology, 89*(2), 487-494. https://doi.org/10.1016/j.biopsycho.2011.12.018

McDermott, J., Troller-Renfree, S., Vanderwert, R., Nelson, C., Zeanah, C., & Fox, N. (2013). Psychosocial deprivation, executive functions, and the emergence of socio-emotional behavior problems. *Frontiers in Human Neuroscience, 7*. https://www.frontiersin.org/articles/10.3389/fnhum.2013.00167

McDermott, J. M., Perez-Edgar, K., Henderson, H. A., Chronis-Tuscano, A., Pine, D. S., & Fox, N. A. (2009). A history of childhood behavioral inhibition and enhanced response monitoring in adolescence are linked to clinical anxiety. *Biological Psychiatry, 65*(5), 445-448. https://doi.org/10.1016/j.biopsych.2008.10.043

McLoughlin, G., Albrecht, B., Banaschewski, T., Rothenberger, A., Brandeis, D., Asherson, P., & Kuntsi, J. (2009). Performance monitoring is altered in adult ADHD: A familial event-related potential investigation. *Neuropsychologia, 47*(14), 3134-3142. https://doi.org/10.1016/j.neuropsychologia.2009.07.013

Mechias, M.-L., Etkin, A., & Kalisch, R. (2010). A meta-analysis of instructed fear studies: Implications for conscious appraisal of threat. *NeuroImage, 49*(2), 1760-1768. https://doi.org/10.1016/j.neuroimage.2009.09.040

Mehra, L. M., Hajcak, G., & Meyer, A. (2022). The relationship between stressful life events and the error-related negativity in children and adolescents. *Developmental Cognitive Neuroscience, 55*, 101110. https://doi.org/10.1016/j.dcn.2022.101110

Mehra, L. M., Hajcak, G., & Meyer, A. (2024). The associations among sleep-related difficulties, anxiety, and error-related brain activity in youth. *Biological Psychology*, 108790. https://doi.org/10.1016/j.biopsycho.2024.108790

Meyer, A. (2016). Developing psychiatric biomarkers: A review focusing on the error-related negativity as a biomarker for anxiety. *Current Treatment Options in Psychiatry, 3*(4), 356-364. https://doi.org/10.1007/s40501-016-0094-5

Meyer, A. (2017). A biomarker of anxiety in children and adolescents: A review focusing on the error-related negativity (ERN) and anxiety across development. *Developmental Cognitive Neuroscience, 27*, 58-68. https://doi.org/10.1016/j.dcn.2017.08.001

Meyer, A. (2022). On the relationship between the error-related negativity and anxiety in children and adolescents: From a neural marker to a novel target for intervention. *Psychophysiology, 59*(6), e14050. https://doi.org/10.1111/psyp.14050

Meyer, A., Carlton, C., Chong, L. J., & Wissemann, K. (2019). The presence of a controlling parent is related to an increase in the error-related negativity in 5–7 year-old children. *Journal of Abnormal Child Psychology, 47*(6), 935-945. https://doi.org/10.1007/s10802-018-0503-x

Meyer, A., Chong, L., Wissemann, K., Mehra, L., & Mirzadegan, I. (2023). An experimental therapeutics approach to the development of a novel computerized treatment targeting error-related brain activity in young children. *Behavior Therapy, 54*(4), 652-665. https://doi.org/10.1016/j.beth.2023.01.005

Meyer, A., Danielson, C. K., Danzig, A. P., Bhatia, V., Black, S. R., Bromet, E., Carlson, G., Hajcak, G., Kotov, R., & Klein, D. N. (2017). Neural biomarker and early temperament predict increased internalizing symptoms after a natural disaster. *Journal of the American Academy of Child & Adolescent Psychiatry, 56*(5), 410-416. https://doi.org/10.1016/j.jaac.2017.02.005

Meyer, A., & Gawlowska, M. (2017). Evidence for specificity of the impact of punishment on error-related brain activity in high versus low trait anxious individuals. *International Journal of Psychophysiology, 120*, 157-163. https://doi.org/10.1016/j.ijpsycho.2017.08.001

Meyer, A., Gibby, B., Wissemann, K., Klawohn, J., Hajcak, G., & Schmidt, N. B. (2020). A brief, computerized intervention targeting error sensitivity reduces the error-related negativity. *Cognitive, Affective, & Behavioral Neuroscience, 20*(1), 172-180. https://doi.org/10.3758/s13415-019-00760-w

Meyer, A., & Hajcak, G. (2019). A review examining the relationship between individual differences in the error-related negativity and cognitive control. *International Journal of Psychophysiology, 144*, 7-13. https://doi.org/10.1016/j.ijpsycho.2019.07.005

Meyer, A., Hajcak, G., Torpey-Newman, D., Kujawa, A., Olino, T. M., Dyson, M., & Klein, D. N. (2018). Early temperamental fearfulness and the developmental trajectory of error-related brain activity. *Developmental Psychobiology, 60*(2), 224-231. https://doi.org/10.1002/dev.21605

Meyer, A., Hajcak, G., Torpey-Newman, D. C., Kujawa, A., & Klein, D. N. (2015). Enhanced error-related brain activity in children predicts the onset of anxiety disorders between the ages of 6 and 9. *Journal of Abnormal Psychology, 124*(2), 266-274. https://doi.org/10.1037/abn0000044

Meyer, A., & Klein, D. N. (2018). Examining the relationships between error-related brain activity (the ERN) and anxiety disorders versus externalizing disorders in young children: Focusing on cognitive control, fear, and shyness. *Comprehensive Psychiatry, 87*, 112-119. https://doi.org/10.1016/j.comppsych.2018.09.009

Meyer, A., Klein, D. N., Torpey, D. C., Kujawa, A. J., Hayden, E. P., Sheikh, H. I., Singh, S. M., & Hajcak, G. (2012). Additive effects of the dopamine D2 receptor and dopamine transporter genes on the error-related negativity in young children. *Genes, Brain and Behavior, 11*(6), 695-703. https://doi.org/10.1111/j.1601-183X.2012.00812.x

Meyer, A., Mehra, L., & Hajcak, G. (2021). Error-related negativity predicts increases in anxiety in a sample of clinically anxious female children and adolescents over 2 years. *Journal of Psychiatry and Neuroscience, 46*(4), E472. https://doi.org/10.1503/jpn.200128

Meyer, A., Nelson, B., Perlman, G., Klein, D. N., & Kotov, R. (2018). A neural biomarker, the error-related negativity, predicts the first onset of generalized anxiety disorder in a large sample of adolescent females. *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 59*(11), 1162-1170. https://doi.org/10.1111/jcpp.12922

Meyer, A., Proudfit, G. H., Bufferd, S. J., Kujawa, A. J., Laptook, R. S., Torpey, D. C., & Klein, D. N. (2015). Self-reported and observed punitive parenting prospectively

predicts increased error-related brain activity in six-year-old children. *Journal of Abnormal Child Psychology, 43*(5), 821-829. https://doi.org/10.1007/s10802-014-9918-1

Meyer, A., Weinberg, A., Klein, D. N., & Hajcak, G. (2012). The development of the error-related negativity (ERN) and its relationship with anxiety: Evidence from 8 to 13 year-olds. *Developmental Cognitive Neuroscience, 2*(1), 152-161. https://doi.org/10.1016/j.dcn.2011.09.005

Meyer, A., & Wissemann, K. (2020). Controlling parenting and perfectionism is associated with an increased error-related negativity (ERN) in young adults. *Social Cognitive and Affective Neuroscience, 15*(1), 87-95. https://doi.org/10.1093/scan/nsaa018

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy, 28*(6), 487-495. https://doi.org/10.1016/0005-7967(90)90135-6

Michael, J. A., Wang, M., Kaur, M., Fitzgerald, P. B., Fitzgibbon, B. M., & Hoy, K. E. (2021). EEG correlates of attentional control in anxiety disorders: A systematic review of error-related negativity and correct-response negativity findings. *Journal of Affective Disorders, 291*, 140-153. https://doi.org/10.1016/j.jad.2021.04.049

Miller, A. E., Watson, J. M., & Strayer, D. L. (2012). Individual differences in working memory capacity predict action monitoring and the error-related negativity. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 38*(3), 757-763. https://doi.org/10.1037/a0026595

Miltner, W. H. R., Lemke, U., Weiss, T., Holroyd, C., Scheffers, M. K., & Coles, M. G. H. (2003). Implementation of error-processing in the human anterior cingulate cortex: a source analysis of the magnetic equivalent of the error-related negativity. *Biological Psychology, 64*(1), 157-166. https://doi.org/10.1016/S0301-0511(03)00107-8

Moran, T. P., Schroder, H. S., Kneip, C., & Moser, J. S. (2017). Meta-analysis and psychophysiology: A tutorial using depression and action-monitoring event-related potentials. *International Journal of Psychophysiology, 111*, 17-32. https://doi.org/10.1016/j.ijpsycho.2016.07.001

Moran, T. P., Taylor, D., & Moser, J. S. (2012). Sex moderates the relationship between worry and performance monitoring brain activity in undergraduates. *International Journal of Psychophysiology, 85*(2), 188-194. https://doi.org/10.1016/j.ijpsycho.2012.05.005

Moriarity, D. P., Joyner, K. J., Slavich, G. M., & Alloy, L. B. (2022). Unconsidered issues of measurement noninvariance in biological psychiatry: A focus on biological phenotypes of psychopathology. *Molecular Psychiatry, 27*(3), 1281-1285. https://doi.org/10.1038/s41380-021-01414-5

Moser, J. S., Hajcak, G., & Simons, R. F. (2005). The effects of fear on performance monitoring and attentional allocation. *Psychophysiology, 42*(3), 261-268. https://doi.org/10.1111/j.1469-8986.2005.00290.x

Moser, J. S., Moran, T. P., & Jendrusina, A. A. (2012). Parsing relationships between dimensions of anxiety and action monitoring brain potentials in female undergraduates. *Psychophysiology, 49*(1), 3-10. https://doi.org/10.1111/j.1469-8986.2011.01279.x

Moser, J. S., Moran, T. P., Kneip, C., Schroder, H. S., & Larson, M. J. (2016). Sex moderates the association between symptoms of anxiety, but not obsessive compulsive disorder, and error-monitoring brain activity: A meta-analytic review. *Psychophysiology, 53*(1), 21-29. https://doi.org/10.1111/psyp.12509

Moser, J. S., Moran, T. P., Schroder, H. S., Donnellan, M. B., & Yeung, N. (2013). On the relationship between anxiety and error monitoring: A meta-analysis and conceptual framework. *Frontiers in Human Neuroscience, 7*, 466. https://doi.org/10.3389/fnhum.2013.00466

Nelson, B. D., Jackson, F., Amir, N., & Hajcak, G. (2015). Single-session attention bias modification and error-related brain activity. *Cognitive, Affective, & Behavioral Neuroscience, 15*(4), 776-786. https://doi.org/10.3758/s13415-015-0365-4

Niessen, E., Fink, G. R., Hoffmann, H. E. M., Weiss, P. H., & Stahl, J. (2017). Error detection across the adult lifespan: Electrophysiological evidence for age-related deficits. *NeuroImage, 152*, 517-529. https://doi.org/10.1016/j.neuroimage.2017.03.015

Nieuwenhuis, S., Ridderinkhof, K. R., Talsma, D., Coles, M. G. H., Holroyd, C. B., Kok, A., & van der Molen, M. W. (2002). A computational account of altered error processing in older age: Dopamine and the error-related negativity. *Cognitive, Affective, & Behavioral Neuroscience, 2*(1), 19-36. https://doi.org/10.3758/CABN.2.1.19

Nigbur, R., & Ullsperger, M. (2020). Funny kittens: Positive mood induced via short video-clips affects error processing but not conflict control. *International Journal of Psychophysiology, 147*, 147-155. https://doi.org/10.1016/j.ijpsycho.2019.11.007

Nitschke, J. B., Heller, W., Palmieri, P. A., & Miller, G. A. (1999). Contrasting patterns of brain activity in anxious apprehension and anxious arousal. *Psychophysiology, 36*(5), 628-637. https://doi.org/10.1017/S0048577299972013

O'Connell, R. G., Dockree, P. M., Bellgrove, M. A., Kelly, S. P., Hester, R., Garavan, H., Robertson, I. H., & Foxe, J. J. (2007). The role of cingulate cortex in the detection of errors with and without awareness: a high-density electrical mapping study. *European Journal of Neuroscience, 25*(8), 2571-2579. https://doi.org/10.1111/j.1460-9568.2007.05477.x

Olver, J. S., O'Keefe, G., Jones, G. R., Burrows, G. D., Tochon-Danguy, H. J., Ackermann, U., Scott, A. M., & Norman, T. R. (2010). Dopamine D1 receptor binding in the

anterior cingulate cortex of patients with obsessive–compulsive disorder. *Psychiatry Research: Neuroimaging, 183*(1), 85-88. https://doi.org/10.1016/j.pscychresns.2010.04.004

Olvet, D. M., & Hajcak, G. (2008). The error-related negativity (ERN) and psychopathology: Toward an endophenotype. *Clinical Psychology Review, 28*(8), 1343-1354. https://doi.org/10.1016/j.cpr.2008.07.003

Olvet, D. M., & Hajcak, G. (2009a). Reliability of error-related brain activity. *Brain Research, 1284*, 89-99. https://doi.org/10.1016/j.brainres.2009.05.079

Olvet, D. M., & Hajcak, G. (2009b). The stability of error-related brain activity with increasing trials. *Psychophysiology, 46*(5), 957-961. https://doi.org/10.1111/j.1469-8986.2009.00848.x

Overbeek, T. J. M., Nieuwenhuis, S., & Ridderinkhof, K. R. (2005). Dissociable components of error processing: On the functional significance of the Pe vis-à-vis the ERN/Ne. *Journal of Psychophysiology, 19*(4), 319-329. https://doi.org/10.1027/0269-8803.19.4.319

Overmeyer, R., Berghäuser, J., Dieterich, R., Wolff, M., Goschke, T., & Endrass, T. (2021). The error-related negativity predicts self-control failures in daily life. *Frontiers in Human Neuroscience, 14*, 616. https://doi.org/10.3389/fnhum.2020.614979

Pasion, R., & Barbosa, F. (2019). ERN as a transdiagnostic marker of the internalizing-externalizing spectrum: A dissociable meta-analytic effect. *Neuroscience and Biobehavioral Reviews, 103*, 133-149. https://doi.org/10.1016/j.neubiorev.2019.06.013

Paul, K., Short, C. A., Beauducel, A., Carsten, H. P., Härpfer, K., Hennig, J., Hewig, J., Hildebrandt, A., Kührt, C., Mueller, E. M., Munk, A., Osinsky, R., Porth, E., Riesel, A., Rodrigues, J., Scheffel, C., Stahl, J., Strobel, A., & Wacker, J. (2022). The methodology and dataset of the CoScience EEG-Personality Project – A large-scale, multi-laboratory project grounded in cooperative forking paths analysis. *Personality Science, 3*, 1-26. https://doi.org/10.5964/ps.7177

Pennebaker, J. W. (2017). Expressive writing in psychological science. *Perspectives on Psychological Science, 13*(2), 226-229. https://doi.org/10.1177/1745691617707315

Peyron, R., Laurent, B., & García-Larrea, L. (2000). Functional imaging of brain responses to pain. A review and meta-analysis (2000). *Neurophysiologie Clinique/Clinical Neurophysiology, 30*(5), 263-288. https://doi.org/10.1016/S0987-7053(00)00227-6

Phillips, K. A., Stein, D. J., Rauch, S. L., Hollander, E., Fallon, B. A., Barsky, A., Fineberg, N., Mataix-Cols, D., Ferrão, Y. A., Saxena, S., Wilhelm, S., Kelly, M. M., Clark, L. A., Pinto, A., Bienvenu, O. J., Farrow, J., & Leckman, J. (2010). Should an obsessive–compulsive spectrum grouping of disorders be included in DSM-V? *Depression and Anxiety, 27*(6), 528-555. https://doi.org/10.1002/da.20705

Pourtois, G., Vocat, R., N'Diaye, K., Spinelli, L., Seeck, M., & Vuilleumier, P. (2010). Errors recruit both cognitive and emotional monitoring systems: Simultaneous intracranial recordings in the dorsal anterior cingulate gyrus and amygdala combined with fMRI. *Neuropsychologia, 48*(4), 1144-1159. https://doi.org/10.1016/j.neuropsychologia.2009.12.020

Proudfit, G. H., Inzlicht, M., & Mennin, D. S. (2013). Anxiety and error monitoring: The importance of motivation and emotion. *Frontiers in Human Neuroscience, 7*, 636. https://doi.org/10.3389/fnhum.2013.00636

Rabinak, C. A., Holman, A., Angstadt, M., Kennedy, A. E., Hajcak, G., & Phan, K. L. (2013). Neural response to errors in combat-exposed returning veterans with and without post-traumatic stress disorder: A preliminary event-related potential study. *Psychiatry Research: Neuroimaging, 213*(1), 71-78. https://doi.org/10.1016/j.pscychresns.2013.01.002

Ramirez, G., & Beilock, S. L. (2011). Writing about testing worries boosts exam performance in the classroom. *Science, 331*(6014), 211-213. https://doi.org/10.1126/science.1199427

Reinhart, R. M. G., & Woodman, G. F. (2014). Causal control of medial-frontal cortex governs electrophysiological and behavioral indices of performance monitoring and learning. *Journal of Neuroscience, 34*(12), 4214-4227. https://doi.org/10.1523/JNEUROSCI.5421-13.2014

Ridderinkhof, K. R., van den Wildenberg, W. P. M., Segalowitz, S. J., & Carter, C. S. (2004). Neurocognitive mechanisms of cognitive control: The role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. *Brain and Cognition, 56*(2), 129-140. https://doi.org/10.1016/j.bandc.2004.09.016

Riesel, A. (2019). The erring brain: Error-related negativity as an endophenotype for OCD—A review and meta-analysis. *Psychophysiology, 56*(4), e13348. https://doi.org/10.1111/psyp.13348

Riesel, A., Endrass, T., Auerbach, L. A., & Kathmann, N. (2015). Overactive performance monitoring as an endophenotype for obsessive-compulsive disorder: Evidence from a treatment study. *American Journal of Psychiatry, 172*(7), 665-673. https://doi.org/10.1176/appi.ajp.2014.14070886

Riesel, A., Endrass, T., Kaufmann, C., & Kathmann, N. (2011). Overactive error-related brain activity as a candidate endophenotype for obsessive-compulsive disorder: Evidence from unaffected first-degree relatives. *The American Journal of Psychiatry, 168*(3), 317-324. https://doi.org/10.1176/appi.ajp.2010.10030416

Riesel, A., Goldhahn, S., & Kathmann, N. (2017). Hyperactive performance monitoring as a transdiagnostic marker: Results from health anxiety in comparison to obsessive–compulsive disorder. *Neuropsychologia, 96*, 1-8. https://doi.org/10.1016/j.neuropsychologia.2016.12.029

Riesel, A., Härpfer, K., Kathmann, N., & Klawohn, J. (2021). In the face of potential harm: The predictive validity of neural correlates of performance monitoring for perceived risk, stress, and internalizing psychopathology during the COVID-19 pandemic. *Biological Psychiatry: Global Open Science, 1*(4), 300-309. https://doi.org/10.1016/j.bpsgos.2021.08.004

Riesel, A., Härpfer, K., Thoma, L., Kathmann, N., & Klawohn, J. (2023). Associations of neural error-processing with symptoms and traits in a dimensional sample recruited across the obsessive–compulsive spectrum. *Psychophysiology, 60*(2), e14164. https://doi.org/10.1111/psyp.14164

Riesel, A., Kathmann, N., & Klawohn, J. (2019). Flexibility of error-monitoring in obsessive-compulsive disorder under speed and accuracy instructions. *Journal of Abnormal Psychology, 128*(7), 671–677. https://doi.org/10.1037/abn0000463

Riesel, A., Kathmann, N., Wüllhorst, V., Banica, I., & Weinberg, A. (2019). Punishment has a persistent effect on error-related brain activity in highly anxious individuals twenty-four hours after conditioning. *International Journal of Psychophysiology, 146*, 63-72. https://doi.org/10.1016/j.ijpsycho.2019.09.014

Riesel, A., Klawohn, J., Grützmann, R., Kaufmann, C., Heinzel, S., Bey, K., Lennertz, L., Wagner, M., & Kathmann, N. (2019). Error-related brain activity as a transdiagnostic endophenotype for obsessive-compulsive disorder, anxiety and substance use disorder. *Psychological Medicine, 49*(7), 1-11. https://doi.org/10.1017/S0033291719000199

Riesel, A., Weinberg, A., Endrass, T., Kathmann, N., & Hajcak, G. (2012). Punishment has a lasting impact on error-related brain activity. *Psychophysiology, 49*(2), 239-247. https://doi.org/10.1111/j.1469-8986.2011.01298.x

Riesel, A., Weinberg, A., Endrass, T., Meyer, A., & Hajcak, G. (2013). The ERN is the ERN is the ERN? Convergent validity of error-related brain activity across different tasks. *Biological Psychology, 93*(3), 377-385. https://doi.org/10.1016/j.biopsycho.2013.04.007

Rollnik, J. D., Schröder, C., Rodríguez-Fornells, A., Kurzbuch, A. R., Däuper, J., Möller, J., & Münte, T. F. (2004). Functional lesions and human action monitoring: combining repetitive transcranial magnetic stimulation and event-related brain potentials. *Clinical Neurophysiology, 115*(1), 145-153. https://doi.org/10.1016/j.clinph.2003.05.001

Ruchsow, M., Spitzer, M., Grön, G., Grothe, J., & Kiefer, M. (2005). Error processing and impulsiveness in normals: evidence from event-related potentials. *Cognitive Brain Research, 24*(2), 317-325. https://doi.org/10.1016/j.cogbrainres.2005.02.003

Ruscio, A. M., Stein, D. J., Chiu, W. T., & Kessler, R. C. (2010). The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. *Molecular Psychiatry, 15*(1), 53-63. https://doi.org/10.1038/mp.2008.94

Russman Block, S. R., Klump, K. L., Beltz, A. M., Burt, S. A., & Moser, J. S. (2024). Ovarian hormones reduce the negative association between worry and cognitive control: A combined neural and behavioral investigation. *Psychoneuroendocrinology, 161*, 106947. https://doi.org/10.1016/j.psyneuen.2023.106947

Rutherford, A. V., Tanovic, E., Bradford, D. E., & Joormann, J. (2020). Psychophysiological correlates of anxious apprehension: Trait worry is associated with startle response to threat. *International Journal of Psychophysiology, 158*, 136-142. https://doi.org/10.1016/j.ijpsycho.2020.09.020

Sandre, A., Banica, I., Riesel, A., Flake, J., Klawohn, J., & Weinberg, A. (2020). Comparing the effects of different methodological decisions on the error-related negativity and its association with behaviour and gender. *International Journal of Psychophysiology, 156*, 18-39. https://doi.org/10.1016/j.ijpsycho.2020.06.016

Santesso, D. L., & Segalowitz, S. J. (2009). The error-related negativity is related to risk taking and empathy in young men. *Psychophysiology, 46*(1), 143-152. https://doi.org/10.1111/j.1469-8986.2008.00714.x

Saunders, B., & Inzlicht, M. (2020). Assessing and adjusting for publication bias in the relationship between anxiety and the error-related negativity. *International Journal of Psychophysiology, 155*, 87-98. https://doi.org/10.1016/j.ijpsycho.2020.05.008

Schoemaker, K., Mulder, H., Deković, M., & Matthys, W. (2013). Executive functions in preschool children with externalizing behavior problems: a meta-analysis. *Journal of Abnormal Child Psychology, 41*(3), 457-471. https://doi.org/10.1007/s10802-012-9684-x

Schreiber, M., Pietschmann, M., Kathmann, N., & Endrass, T. (2011). ERP correlates of performance monitoring in elderly. *Brain and Cognition, 76*(1), 131-139. https://doi.org/10.1016/j.bandc.2011.02.003

Schroder, H. S., Moran, T. P., & Moser, J. S. (2018). The effect of expressive writing on the error-related negativity among individuals with chronic worry. *Psychophysiology, 55*(2), e12990. https://doi.org/10.1111/psyp.12990

Segalowitz, S. J., Santesso, D. L., Murphy, T. I., Homan, D., Chantziantoniou, D. K., & Khan, S. (2010). Retest reliability of medial frontal negativities during performance monitoring. *Psychophysiology, 47*(2), 260-270. https://doi.org/10.1111/j.1469-8986.2009.00942.x

Seow, T. X. F., Benoit, E., Dempsey, C., Jennings, M., Maxwell, A., McDonough, M., & Gillan, C. M. (2020). A dimensional investigation of error-related negativity (ERN) and self-reported psychiatric symptoms. *International Journal of Psychophysiology*. https://doi.org/10.1016/j.ijpsycho.2020.09.019

Shackman, A. J., Salomons, T. V., Slagter, H. A., Fox, A. S., Winter, J. J., & Davidson, R. J. (2011). The integration of negative affect, pain and cognitive control in the

cingulate cortex. *Nature Reviews Neuroscience, 12*(3), 154-167. https://doi.org/10.1038/nrn2994

Shansky, R., & Lipps, J. (2013). Stress-induced cognitive dysfunction: hormone-neurotransmitter interactions in the prefrontal cortex. *Frontiers in Human Neuroscience, 7*, 123. https://doi.org/10.3389/fnhum.2013.00123

Sharma, E., Sharma, L. P., Balachander, S., Lin, B., Manohar, H., Khanna, P., Lu, C., Garg, K., Thomas, T. L., Au, A. C. L., Selles, R. R., Højgaard, D. R. M. A., Skarphedinsson, G., & Stewart, S. E. (2021). Comorbidities in obsessive-compulsive disorder across the lifespan: a systematic review and meta-analysis. *Frontiers in Psychiatry, 12*, 703701. https://doi.org/10.3389/fpsyt.2021.703701

Sharp, P. B., Miller, G. A., & Heller, W. (2015). Transdiagnostic dimensions of anxiety: Neural mechanisms, executive functions, and new directions. *International Journal of Psychophysiology, 98*(2), 365-377. https://doi.org/10.1016/j.ijpsycho.2015.07.001

Shenhav, A., Botvinick, M. M., & Cohen, J. D. (2013). The expected value of control: An integrative theory of anterior cingulate cortex function. *Neuron, 79*(2), 217-240. https://doi.org/10.1016/j.neuron.2013.07.007

Simmonite, M., Bates, A. T., Groom, M. J., Jackson, G. M., Hollis, C., & Liddle, P. F. (2012). Error processing-associated event-related potentials in schizophrenia and unaffected siblings. *International Journal of Psychophysiology, 84*(1), 74-79. https://doi.org/10.1016/j.ijpsycho.2012.01.012

Simmons, J. P., Nelson, L. D., & Simonsohn, U. (2011). False-positive psychology: Undisclosed flexibility in data collection and analysis allows presenting anything as significant. *Psychological Science, 22*(11), 1359-1366. https://doi.org/10.1177/0956797611417632

Simons, R. F. (2010). The way of our errors: Theme and variations. *Psychophysiology, 47*(1), 1-14. https://doi.org/10.1111/j.1469-8986.2009.00929.x

Solmi, M., Radua, J., Olivola, M., Croce, E., Soardo, L., Salazar de Pablo, G., Il Shin, J., Kirkbride, J. B., Jones, P., Kim, J. H., Kim, J. Y., Carvalho, A. F., Seeman, M. V., Correll, C. U., & Fusar-Poli, P. (2022). Age at onset of mental disorders worldwide: large-scale meta-analysis of 192 epidemiological studies. *Molecular Psychiatry, 27*(1), 281-295. https://doi.org/10.1038/s41380-021-01161-7

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the State-Trait Anxiety Inventory*. Consulting Psychologists Press.

Stangier, U., & Heidenreich, T. (2003). Die Liebowitz Soziale Angst‐Skala (LSAS) [The Liebowitz social anxiety scale]. In Collegium Internationale Psychiatriae Scalarum (Ed.), *Internationale Skalen für Psychiatrie*. Hogrefe.

Stein, D. J. (2008a). Is disorder x in category or spectrum y? General considerations and application to the relationship between obsessive–compulsive disorder and anxiety

disorders. *Depression and Anxiety, 25*(4), 330-335. https://doi.org/10.1002/da.20497

Stein, D. J. (2008b). Psychobiology of anxiety disorders and obsessive-compulsive spectrum disorders. *CNS Spectrums, 13*(S14), 23-28. https://doi.org/10.1017/S1092852900026900

Stein, D. J., Fineberg, N. A., Bienvenu, O. J., Denys, D., Lochner, C., Nestadt, G., Leckman, J. F., Rauch, S. L., & Phillips, K. A. (2010). Should OCD be classified as an anxiety disorder in DSM-V? *Depression and Anxiety, 27*(6), 495-506. https://doi.org/10.1002/da.20699

Stemmer, B., Segalowitz, S. J., Witzke, W., & Schönle, P. W. (2004). Error detection in patients with lesions to the medial prefrontal cortex: an ERP study. *Neuropsychologia, 42*(1), 118-130. https://doi.org/10.1016/S0028-3932(03)00121-0

Storch, E. A., Abramowitz, J., & Goodman, W. K. (2008). Where does obsessive–compulsive disorder belong in DSM-V? *Depression and Anxiety, 25*(4), 336-347. https://doi.org/10.1002/da.20488

Suor, J. H., Calentino, A. E., Granros, M., & Burkhouse, K. L. (2022). Maternal error-related negativity relationship with offspring error-related negativity and negative parenting styles: a novel model of internalizing psychopathology risk. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 7*(4), 435-442. https://doi.org/10.1016/j.bpsc.2021.10.007

Swick, D., Turken, & U. (2002). Dissociation between conflict detection and error monitoring in the human anterior cingulate cortex. *Proceedings of the National Academy of Sciences, 99*(25), 16354-16359. https://doi.org/10.1073/pnas.252521499

Tamnes, C. K., Walhovd, K. B., Torstveit, M., Sells, V. T., & Fjell, A. M. (2013). Performance monitoring in children and adolescents: A review of developmental changes in the error-related negativity and brain maturation. *Developmental Cognitive Neuroscience, 6*, 1-13. https://doi.org/10.1016/j.dcn.2013.05.001

Tan, P. Z., Rozenman, M., Chang, S. W., Jurgiel, J., Truong, H. V., Piacentini, J., & Loo, S. K. (2021). The ERN as a neural index of changes in performance monitoring following attention training in pediatric obsessive-compulsive disorder. *Biological Psychology, 166*, 108206. https://doi.org/10.1016/j.biopsycho.2021.108206

Tang, W., Jbabdi, S., Zhu, Z., Cottaar, M., Grisot, G., Lehman, J. F., Yendiki, A., & Haber, S. N. (2019). A connectional hub in the rostral anterior cingulate cortex links areas of emotion and cognitive control. *eLife, 8*, e43761. https://doi.org/10.7554/eLife.43761

Taylor, J. B., Visser, T. A. W., Fueggle, S. N., Bellgrove, M. A., & Fox, A. M. (2018). The error-related negativity (ERN) is an electrophysiological marker of motor impulsiveness on the Barratt Impulsiveness Scale (BIS-11) during adolescence.

Developmental Cognitive Neuroscience, 30, 77-86. https://doi.org/10.1016/j.dcn.2018.01.003

Taylor, S. F., Stern, E. R., & Gehring, W. J. (2007). Neural systems for error monitoring: Recent findings and theoretical perspectives. *The Neuroscientist, 13*(2), 160-172. https://doi.org/10.1177/1073858406298184

Teixeira, A. L., Rocha, N. P., & Berk, M. (2023). Biomarkers in Psychiatry: Conceptual and Methodological Challenges. In A. L. Teixeira, N. P. Rocha, & M. Berk (Eds.), *Biomarkers in Neuropsychiatry: A Primer* (pp. 343-350). Springer International Publishing. https://doi.org/10.1007/978-3-031-43356-6_20

Tetzner, J., & Schuth, M. (2016). Anxiety in late adulthood: Associations with gender, education, and physical and cognitive functioning. *Psychology and Aging, 31*(5), 532-544. https://doi.org/10.1037/pag0000108

Torpey, D. C., Hajcak, G., & Klein, D. N. (2009). An examination of error-related brain activity and its modulation by error value in young children. *Developmental Neuropsychology, 34*(6), 749-761. https://doi.org/10.1080/87565640903265103

Trayvick, J., Adams, E. M., & Nelson, B. D. (2024). Family study of the error-related negativity in adolescent and young adult females and their parents. *Psychophysiology*, e14669. https://doi.org/10.1111/psyp.14669

Troller-Renfree, S., Nelson, C. A., Zeanah, C. H., & Fox, N. A. (2016). Deficits in error monitoring are associated with externalizing but not internalizing behaviors among children with a history of institutionalization. *Journal of Child Psychology and Psychiatry, 57*(10), 1145-1153. https://doi.org/10.1111/jcpp.12604

Turken, A. U., & Swick, D. (2008). The effect of orbitofrontal lesions on the error-related negativity. *Neuroscience Letters, 441*(1), 7-10. https://doi.org/10.1016/j.neulet.2008.05.115

Ullsperger, M., & von Cramon, D. Y. (2006). The role of intact frontostriatal circuits in error processing. *Journal of Cognitive Neuroscience, 18*(4), 651-664. https://doi.org/10.1162/jocn.2006.18.4.651

Ullsperger, M., von Cramon, D. Y., & Müller, N. G. (2002). Interactions of focal cortical lesions with error processing: Evidence from event-related brain potentials. *Neuropsychology, 16*(4), 548-561. https://doi.org/10.1037/0894-4105.16.4.548

Ursu, S., Clark, K. A., Aizenstein, H. J., Stenger, V. A., & Carter, C. S. (2009). Conflict-related activity in the caudal anterior cingulate cortex in the absence of awareness. *Biological Psychology, 80*(3), 279-286. https://doi.org/10.1016/j.biopsycho.2008.10.008

Vallet, W., Neige, C., Mouchet-Mages, S., Brunelin, J., & Grondin, S. (2021). Response-locked component of error monitoring in psychopathy: A systematic review and meta-analysis of error-related negativity/positivity. *Neuroscience & Biobehavioral Reviews, 123*, 104-119. https://doi.org/10.1016/j.neubiorev.2021.01.004

Valt, C., Huber, D., Erhardt, I., & Stürmer, B. (2018). Internal and external signal processing in patients with panic disorder: An event-related potential (ERP) study. *PLOS ONE, 13*(11), e0208257. https://doi.org/10.1371/journal.pone.0208257

van Boxtel, G. J. M., van der Molen, M. W., & Jennings, J. R. (2005). Differential involvement of the anterior cingulate cortex in performance monitoring during a stop-signal task. *Journal of Psychophysiology, 19*(1), 1-10. https://doi.org/10.1027/0269-8803.19.1.1

van Veen, V., & Carter, C. S. (2002). The anterior cingulate as a conflict monitor: fMRI and ERP studies. *Physiology & Behavior, 77*(4), 477-482. https://doi.org/10.1016/S0031-9384(02)00930-7

Verkuil, B., Brosschot, J. F., Putman, P., & Thayer, J. F. (2009). Interacting effects of worry and anxiety on attentional disengagement from threat. *Behaviour Research and Therapy, 47*(2), 146-152. https://doi.org/10.1016/j.brat.2008.11.003

Vidal, F., Burle, B., Bonnet, M., Grapperon, J., & Hasbroucq, T. (2003). Error negativity on correct trials: A reexamination of available data. *Biological Psychology, 64*(3), 265-282. https://doi.org/10.1016/S0301-0511(03)00097-8

Vidal, F., Hasbroucq, T., Grapperon, J., & Bonnet, M. (2000). Is the 'error negativity' specific to errors? *Biological Psychology, 51*(2-3), 109-128. https://doi.org/10.1016/S0301-0511(99)00032-0

Voegler, R., Peterburs, J., Lemke, H., Ocklenburg, S., Liepelt, R., & Straube, T. (2018). Electrophysiological correlates of performance monitoring under social observation in patients with social anxiety disorder and healthy controls. *Biological Psychology, 132*, 71-80. https://doi.org/10.1016/j.biopsycho.2017.11.003

Watson, D., & Clark, L. A. (1991). *The Mood and Anxiety Symptom Questionnaire*. University of Iowa.

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*(1), 3-14. https://doi.org/10.1037/0021-843X.104.1.3

Weaver, M. D., Hickey, C., & van Zoest, W. (2017). The impact of salience and visual working memory on the monitoring and control of saccadic behavior: An eye-tracking and EEG study. *Psychophysiology, 54*(4), 544-554. https://doi.org/10.1111/psyp.12817

Webber, H. E., de Dios, C., Kessler, D. A., Schmitz, J. M., Lane, S. D., & Suchting, R. (2024). A meta-analysis of electrophysiological biomarkers of reward and error monitoring in substance misuse. *Psychophysiology*, e14515. https://doi.org/10.1111/psyp.14515

Weinberg, A., Dieterich, R., & Riesel, A. (2015). Error-related brain activity in the age of RDoC: A review of the literature. *International Journal of Psychophysiology, 98*(2), 276-299. https://doi.org/10.1016/j.ijpsycho.2015.02.029

Weinberg, A., & Hajcak, G. (2011). Longer term test–retest reliability of error-related brain activity. *Psychophysiology, 48*(10), 1420-1425. https://doi.org/10.1111/j.1469-8986.2011.01206.x

Weinberg, A., Kotov, R., & Proudfit, G. H. (2015). Neural indicators of error processing in generalized anxiety disorder, obsessive-compulsive disorder, and major depressive disorder. *Journal of Abnormal Psychology, 124*(1), 172-185. https://doi.org/10.1037/abn0000019

Weinberg, A., Kujawa, A., & Riesel, A. (2022). Understanding trajectories to anxiety and depression: Neural responses to errors and rewards as indices of susceptibility to stressful life events. *Current Directions in Psychological Science*, 1-9. https://doi.org/10.1177/09637214211049228

Weinberg, A., Meyer, A., Hale-Rude, E., Perlman, G., Kotov, R., Klein, D. N., & Hajcak, G. (2016). Error-related negativity (ERN) and sustained threat: Conceptual framework and empirical evaluation in an adolescent sample. *Psychophysiology, 53*(3), 372-385. https://doi.org/10.1111/psyp.12538

Weinberg, A., Olvet, D. M., & Hajcak, G. (2010). Increased error-related brain activity in generalized anxiety disorder. *Biological Psychology, 85*(3), 472-480. https://doi.org/10.1016/j.biopsycho.2010.09.011

Weinberg, A., Riesel, A., & Hajcak, G. (2012). Integrating multiple perspectives on error-related brain activity: The ERN as a neural indicator of trait defensive reactivity. *Motivation and Emotion, 36*(1), 84-100. https://doi.org/10.1007/s11031-011-9269-y

Weissman, M. M., Wickramaratne, P., Adams, P., Wolk, S., Verdeli, H., & Olfson, M. (2000). Brief screening for family psychiatric history: The Family History Screen. *Archives of General Psychiatry, 57*(7), 675-682. https://doi.org/10-1001/pubs.Arch_Gen_Psychiatry-ISSN-0003-990x-57-7-yoa8214

Wessel, J. R. (2012). Error awareness and the error-related negativity: Evaluating the first decade of evidence. *Frontiers in Human Neuroscience, 6*(88), 1-16. https://doi.org/10.3389/fnhum.2012.00088

Wiswede, D., Münte, T. F., Goschke, T., & Rüsseler, J. (2009). Modulation of the error-related negativity by induction of short-term negative affect. *Neuropsychologia, 47*(1), 83-90. https://doi.org/10.1016/j.neuropsychologia.2008.08.016

Wiswede, D., Münte, T. F., & Rüsseler, J. (2009). Negative affect induced by derogatory verbal feedback modulates the neural signature of error detection. *Social Cognitive and Affective Neuroscience, 4*(3), 227-237. https://doi.org/10.1093/scan/nsp015

Wittchen, H. U., Zaudig, M., & Fydrich, T. (1997). *SKID. Strukturiertes Klinisches Interview für DSM-IV. Achse I und II. Handanweisung*. Hogrefe.

Yang, Y., Shields, G. S., Zhang, Y., Wu, H., Chen, H., & Romer, A. L. (2022). Child executive function and future externalizing and internalizing problems: a meta-analysis of prospective longitudinal studies. *Clinical Psychology Review, 97*, 102194. https://doi.org/10.1016/j.cpr.2022.102194

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review, 111*(4), 931-959. https://doi.org/10.1037/0033-295X.111.4.931

Zainal, N. H., & Newman, M. G. (2023). A cross-lagged prospective network analysis of depression and anxiety and cognitive functioning components in midlife community adult women. *Psychological Medicine, 53*(9), 4160-4171. https://doi.org/10.1017/S0033291722000848

Zheng, Y., Sheng, W., Xu, J., & Zhang, Y. (2014). Sensation seeking and error processing. *Psychophysiology, 51*(9), 824-833. https://doi.org/10.1111/psyp.12240

Zirnheld, P. J., Carroll, C. A., Kieffaber, P. D., O'Donnell, B. F., Shekhar, A., & Hetrick, W. P. (2004). Haloperidol impairs learning and error-related negativity in humans. *Journal of Cognitive Neuroscience, 16*(6), 1098-1112. https://doi.org/10.1162/0898929041502779

Appendix

Appendix A1 – Additional Analyses of Study 1

Table A1.1. Results of the Interaction of Trait and State Worry on Alterations of ERN and CRN.

	b	*SE*	*t*	*p*	R^2	*F*	*df*	*p*
ERN Diff. T1 – T0					0.04	1.19	3, 86	.318
Trait Worry	-0.04	0.03	-1.30	.199				
State Worry	0.00	0.07	0.01	.996				
Trait × State Worry	0.00	0.00	0.37	.709				
CRN Diff. T1 – T0					0.01	0.16	3, 86	.922
Trait Worry	-0.01	0.01	-0.58	.561				
State Worry	0.01	0.02	0.26	.792				
Trait × State Worry	0.00	0.00	-0.25	.806				

Note. ERN = error-related negativity (measured at FCz, peak-to-peak) difference between T1 – T0; CRN = correct-response negativity (measured at FCz, peak-to-peak) difference between T1 – T0; trait worry = Penn State Worry Questionnaire; state worry = difference of self-reported state worry between T1 and T0. *N* = 90.

Table A1.2. Results of Potential Intervention Effects on the Pe.

	F	*df*	η_p^2	*p*
Group	1.55	1, 87	0.03	.218
Time	0.46	1, 87	0.01	.498
Group × Time	2.42	2, 87	0.05	.095

Note. Pe = error positivity (measured at CPz, mean amplitude of 200 to 400 ms, baseline corrected for -500 to -300 ms); group (induction, reduction, control); time (T0, T1). Uncorrected separate post-hoc ANOVAs for each group did not reveal a significant effect (all *p*s >.16). *N* = 90.

Table A1.3. Results of the Potential Association of Trait Worry and the Pe.

	b	*SE*	*t*	*p*	R^2	*F*	*df*	*p*
Pe					0.15	5.15	3, 86	**.003**
Gender	1.77	0.80	2.23	**.028**				
PSWQ	-0.01	0.03	-0.27	.790				
Gender × PSWQ	-0.11	0.08	-1.53	.131				

Note. Pe = error positivity (measured at CPz, mean amplitude of 200 to 400 ms, baseline corrected for -500 to -300 ms); PSWQ = Penn State Worry Questionnaire (trait worry) was mean-centered. Significant *p* <.05 printed in bold. *N* = 90.

Appendix A2 – Additional Analyses of Study 2

Table A2.1. ANCOVA Results Regarding Potential Differences of Early and Late Pe Between Diagnostic Groups.

	F	*df*	η_p^2	*p*
Early Pe				
Group	0.91	3, 151	0.02	.439
Late Pe				
Group	0.84	3, 151	0.02	.473

Note. Pe = error positivity (measured at CPz, mean amplitude of 200 to 400 ms (early) and 400 to 600 ms (late), baseline corrected for -500 to -300 ms); group (OCD, SAD, PHOB, CON). Due to behavioral differences between the groups, analyses were controlled for error response times. *N* = 156.

Table A2.2. ANCOVA Results Regarding Potential Differences of Early and Late Pe Between Diagnostic Groups (Including a Strictly Healthy Control Group).

	F	*df*	η_p^2	*p*
Early Pe				
Group	0.83	3, 146	0.02	.479
Late Pe				
Group	0.81	3, 146	0.02	.489

Note. Pe = error positivity (measured at CPz, mean amplitude of 200 to 400 ms (early) and 400 to 600 ms (late), baseline corrected for -500 to -300 ms); group (OCD, SAD, PHOB, HC). Due to behavioral differences between the groups, analyses were controlled for error response times. *N* = 151.

Table A2.3. ANCOVA Results Regarding Potential Effects of Clinical Status of an Internalizing Disorder and Family Risk for Internalizing Psychopathology on Early and Late Pe.

	F	*df*	η_p^2	*p*
Early Pe				
Clinical	0.20	1, 150	0.00	.657
FHS Internalizing	3.13	1, 150	0.02	.079
Clinical × FHS Internalizing	5.04	1, 150	0.03	**.026**
Late Pe				
Clinical	0.13	1, 150	0.00	.909
FHS Internalizing	1.12	1, 150	0.01	.291
Clinical × FHS Internalizing	2.55	1, 150	0.02	.112

Note. Pe = error positivity (measured at CPz, mean amplitude of 200 to 400 ms (early) and 400 to 600 ms (late), baseline corrected for -500 to -300 ms); FHS = Family History Screen (family risk, no family risk); clinical (nonclinical, clinical). Analyses were controlled for error response time and post-error slowing, as these behavioral variables differed between the respective groups. $N = 156$.

Follow-up analyses regarding the interaction effect on the early Pe using Sidak corrected post-hoc comparisons ($p = .022$) revealed smaller amplitudes for nonclinical participants with family risk ($n = 15$; $M = 2.02$, $SD = 0.81$) compared with those without family risk ($n = 19$; $M = 4.59$, $SD = 0.75$), while clinical participants with and without family risk did not differ significantly ($p = .660$).

$p < .05$ are printed in bold

Table A2.4. The Role of Clinical Status, PSWQ, and MASQ-AA on the early and late Pe within the Combined Sample across the Severity Continuum.

	b	SE_{boot}	β	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*
Early Pe						0.09	3.68	9, 239	**<.001**
Gender	1.08	0.43	0.15	2.33	**.014**				
Age	-0.11	0.02	-0.23	-3.68	**<.001**				
Clinical	0.82	0.48	0.13	1.66	.087				
PSWQ	-0.04	0.03	-0.15	-1.04	.251				
MASQ-AA	-0.03	0.05	-0.07	-0.56	.537				
Clinical × PSWQ	-0.02	0.04	-0.05	-0.38	.676				
Clinical × MASQ-AA	0.08	0.06	0.18	1.28	.179				
PSWQ × MASQ-AA	0.00	0.00	0.08	0.60	.509				
Clinical × PSWQ × MASQ-AA	-0.01	0.00	-0.21	-1.37	.115				
Late Pe						0.01	1.31	9, 236	.231
Gender	0.39	0.34	0.07	1.10	.249				
Age	-0.05	0.02	-0.14	-2.18	**.024**				
Clinical	0.17	0.36	0.04	0.44	.662				
PSWQ	-0.02	0.02	-0.08	-0.54	.511				
MASQ-AA	-0.01	0.04	-0.02	-0.16	.863				
Clinical × PSWQ	-0.01	0.03	-0.05	-0.36	.688				
Clinical × MASQ-AA	0.02	0.05	0.04	0.30	.739				
PSWQ × MASQ-AA	0.00	0.00	-0.01	-0.08	.925				
Clinical × PSWQ × MASQ-AA	0.00	0.00	-0.06	-0.40	.605				

Note. Pe = error positivity (measured at CPz, mean amplitude of 200 to 400 ms (early) and 400 to 600 ms (late), baseline corrected for -500 to -300 ms); gender (0 = female, 1 = male); age in years; clinical (0 = nonclinical, 1 = clinical); PSWQ = Penn State Worry Questionnaire; MASQ-AA = Mood and Anxiety Symptom Questionnaire; continuous variables were mean-centered. $N = 246$.

$p < .05$ are printed in bold

Table A2.5. The Role of Gender, Clinical Status, and PSWQ on the early and late Pe within the Combined Sample across the Severity Continuum.

	b	SE_{boot}	*β*	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*
Early Pe						0.10	5.54	6, 239	**<.001**
Gender	2.14	0.80	0.30	2.81	**.005**				
Age	-0.11	0.03	-0.24	-3.76	**<.001**				
Clinical	0.97	0.49	0.15	1.91	**.047**				
PSWQ	-0.05	0.02	-0.21	-2.62	**.007**				
Gender × Clinical	-1.93	0.96	-0.20	-1.83	**.049**				
Gender × PSWQ	0.03	0.04	0.06	0.66	.425				
Late Pe						0.02	1.91	6, 239	.080
Gender	0.75	0.66	0.14	1.29	.256				
Age	-0.05	0.02	-0.14	-2.25	**<.016**				
Clinical	0.26	0.36	0.06	0.67	.468				
PSWQ	-0.03	0.01	-0.15	-1.78	**.042**				
Gender × Clinical	-0.63	0.79	-0.09	-0.77	.407				
Gender × PSWQ	0.02	0.03	0.05	0.56	.509				

Note. Pe = error positivity (measured at CPz, mean amplitude of 200 to 400 ms (early) and 400 to 600 ms (late), baseline corrected for -500 to -300 ms); gender (0 = female, 1 = male); age in years; clinical (0 = nonclinical, 1 = clinical); PSWQ = Penn State Worry Questionnaire. $N = 246$.

The interaction effect of Gender × Clinical on the early Pe was followed-up using an ANOVA with gender and clinical status as between-subject factors. Sidak corrected post-hoc comparisons ($p = .002$) revealed larger amplitudes in non-clinical males ($n = 35$; $M = 5.04$, $SD = 3.61$) compared with non-clinical females ($n = 76$; $M = 2.96$, $SD = 3.02$), while clinical males and females did not differ significantly ($p = .711$).

$p < .05$ are printed in bold

Appendix A3 – Additional Analyses of Study 3

Table A3.1. ANOVA Results Regarding Potential Differences ERPs between First Onset Participants and Those Without a Lifetime History of Internalizing Disorders.

	F	*df*	η_p^2	*p*
ERN/CRN				
Response	133.10	1, 84	0.61	**<.001**
Clinical	0.05	1, 84	0.00	.831
Response × Clinical	0.11	1, 84	0.00	.738
Pe				
Clinical	0.81	1, 84	0.00	.776
N2				
Clinical	0.16	1, 84	0.00	.687

Note. ERN = error-related negativity, CRN = correct-response negativity (both measured at FCz, mean amplitude of 0 to 100 ms, baseline corrected for -500 to -300 ms); Pe = error positivity (measured at Pz, mean amplitude of 200 to 400 ms, baseline corrected for -500 to -300 ms); stimulus-locked N2 (measured at FCz, mean amplitude of 220 to 320 ms, baseline corrected for -200 to 0 ms); response (correct, incorrect); clinical (no lifetime internalizing diagnoses, first onset of internalizing diagnosis after baseline assessment). $N = 86$.

Contributions

Study 1

I actively contributed to the first phase of this project by shaping the initial research idea and formulating precise questions, goals, and objectives for the study (conceptualization). I designed the randomized-controlled longitudinal research approach and ensured its preregistration (methodology). I selected the interventions and provided the utilized study protocols (resources). I customized the flanker task to suit the requirements of this study by programming the task in Presentation (software). Collaboratively with my research team, I dedicated significant efforts to ensure the verification and reproducibility of the experiment (validation). One of my main tasks during the data collection were recruiting participants, interviewing them, and performing the actual EEG experiment with them in the lab (investigation). I supervised undergraduate students and research assistants to maintain high standards during data acquisition (supervision). I kept a constant overview of all ongoing activities and demands regarding the coordination of the project (project administration). I made sure that all data were safely stored and took care of the maintenance of the ongoing data acquisition (data curation). I preprocessed the EEG data and performed the frequentist as well as Bayesian statistical analyses (formal analysis). I visualized both questionnaire and EEG data (visualization). Lastly, I wrote the original draft of the manuscript and edited it based on my co-authors comments before submission (writing and editing).

Study 2

Since the second study was part of a larger project, funded by the German Research Foundation with grants awarded to Anja Riesel, I was not part of the initial funding proposal phase. However, I was actively involved in formulating and adapting the precise questions, goals, and objectives for the study before data collection began (conceptualization). Given the predefined observational research design outlined in the proposal, my contribution involved the augmentation of additional questionnaires, facilitating overlaps with other projects and thereby enabling collaborative investigations across multiple datasets. Eventually, I preregistered the precise study design and analyses (methodology). I provided the utilized study protocols (resources) and ensured to use the identical flanker of the first study that I already programmed (software). Collaboratively with my research team, I dedicated significant efforts to ensure the verification and reproducibility of the experiment (validation). One of my main tasks during the data collection were recruiting participants,

interviewing them, and performing the actual EEG experiment with them in the lab (investigation). I supervised undergraduate students and research assistants to maintain high standards during data acquisition (supervision). I kept a constant overview of all ongoing activities and demands regarding the coordination of the project (project administration). I made sure that all data were safely stored and took care of the maintenance of the ongoing data acquisition (data curation). I preprocessed the EEG data and performed the statistical analyses (formal analysis). I visualized both questionnaire and EEG data (visualization). Lastly, I wrote the original draft of the manuscript and edited it based on my co-authors comments before submission (writing and editing).

Study 3

When I began my PhD, the principal investigator had already developed the overall research objectives (conceptualization) as well as the research design of the third study (methodology). However, I was responsible for implementing the questionnaires for the online study using Limesurvey (resources, software). Collaboratively with my research team, I dedicated significant efforts to ensure the verification and reproducibility of the experiment (validation). One of my main tasks during the data collection were recruiting participants and interviewing them (investigation). I supervised undergraduate students and research assistants to maintain high standards during data acquisition (supervision). I kept a constant overview of all ongoing activities and demands regarding the coordination of the project (project administration). I made sure that all data were safely stored and took care of the maintenance of the ongoing data acquisition (data curation). I preprocessed the EEG data and significantly contributed to the performed statistical analyses (formal analysis). I visualized both questionnaire and EEG data (visualization). Lastly, I wrote the original draft of a substantial amount of paragraphs and reviewed the overall manuscript of the first author (writing and editing).

Materials

Hardware

BrainAmp DC Amplifiers (Brain Products GmbH), were used to record continuous EEG data in all three studies.

BrainCap (EASYCAP GmbH), 64 channels with equidistant and concentric electrode layout, was used to record continuous EEG data in all three studies.

Dell Monitor (Dell, Inc.), 19-inch LCD, was used to display the flanker task in study 1 and 2 (for study 3, monitor specifications were unknown).

Software

BrainVision Recorder (Brain Products GmbH), version 1.22, was used to record continuous EEG data in all three studies (for study 3, version number is unknown).

BrainVision Analyzer (Brain Products GmbH), version 2.2, was used to transform and preprocess continuous EEG data to stimulus- and response-locked ERPs in all three studies.

ChatGPT (OpenAI, Inc.), version GPT 3.5 and GPT 4, was used for language editing purposes and the improvement of readability in study 2 as well as in this dissertation.

Endnote (Clarivate, Plc), version X9.3.3, was used for structuring literature in all three studies as well as in this dissertation.

G*Power (Faul et al., 2009, 2007), version 3.1.9.7, was used to perform power analyses for study 1 and 2.

JASP (JASP Team, 2021), version 0.15.0.0, was used for Bayesian statistical analyses in study 1.

LimeSurvey (LimeSurvey GmbH), version 2, was used for administrating questionnaires in study 1 and 3 (follow-up).

Matlab (The MathWorks, Inc.), version R2019a, was used for creating figures in study 3.

Microsoft Word (Microsoft, Inc.), version 2016, was used for drafting the manuscripts of all three studies as well as this dissertation.

Microsoft PowerPoint (Microsoft, Inc.), version 2016, was used for creating figures in all three studies as well as in this dissertation.

Presentation (Neurobehavioral Systems, Inc.), version 22.1, was used for administrating the flanker tasks in all three studies (for study 3, version number is unknown).

psychoEQ (psychoWare Software GmbH), version 21.01, was used for administrating questionnaires in study 2 and 3 (baseline).

RStudio (Posit Software, PBC), version 2023.06.1, was used for creating figures in study 1 and 2 (for study 1, version number is unknown).

SPSS (IBM, Inc.), version 25.0 and 29.0, was used for frequentist statistical analyses in all three studies and for the explorative analyses of this dissertation.

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Moreover, I would also like to extend my thanks to the second member of my dissertation committee, Prof. Dr. Brigitte Röder. Her insightful comments and encouragement greatly enriched my work. Her distinguished expertise in biological psychology brought a rigorous and valuable perspective to my research projects. This motivated me to refine my methodological approach, deepened my understanding of event-related potentials, and ultimately enhanced the quality of my research.

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Publications

This cumulative dissertation comprises the following three research articles, which can be read in their original form on the subsequent pages:

Study 1: Disentangling Trait and State Worry

Härpfer, K., Carsten, H. P., Löwisch, K., Westermann, N., & Riesel, A. (2022). Disentangling the effects of trait and state worry on error-related brain activity: Results from a randomized controlled trial using worry manipulations. *Psychophysiology, 59*(9), e14055. https://doi.org/10.1111/psyp.14055

Study 2: Enhanced Performance Monitoring as a Transdiagnostic Risk Marker

Härpfer, K., Carsten, H. P., Kausche, F. M., & Riesel, A. (in revision). Enhanced performance monitoring as a transdiagnostic risk marker of the anxiety and obsessive-compulsive spectrum: The role of disorder category, clinical status, family risk, and anxiety dimensions. *Depression and Anxiety*.

Study 3: Translating Risk into Internalizing Psychopathology

Riesel, A., Härpfer, K., Kathmann, N., & Klawohn, J. (2021). In the face of potential harm: The predictive validity of neural correlates of performance monitoring for perceived risk, stress, and internalizing psychopathology during the COVID-19 pandemic. *Biological Psychiatry: Global Open Science, 1*(4), 300-309. https://doi.org/10.1016/j.bpsgos. 2021.08.004

Study 1

Disentangling the Effects of Trait and State Worry on Error-Related Brain Activity: Results from a Randomized Controlled Trial Using Worry Manipulations

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ORIGINAL ARTICLE

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

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Abstract

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

KEYWORDS

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

1 | INTRODUCTION

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

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

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

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

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

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

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

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

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

2 | METHOD

2.1 | Participants

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

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

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

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

2.2 | Procedure

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

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

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

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

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

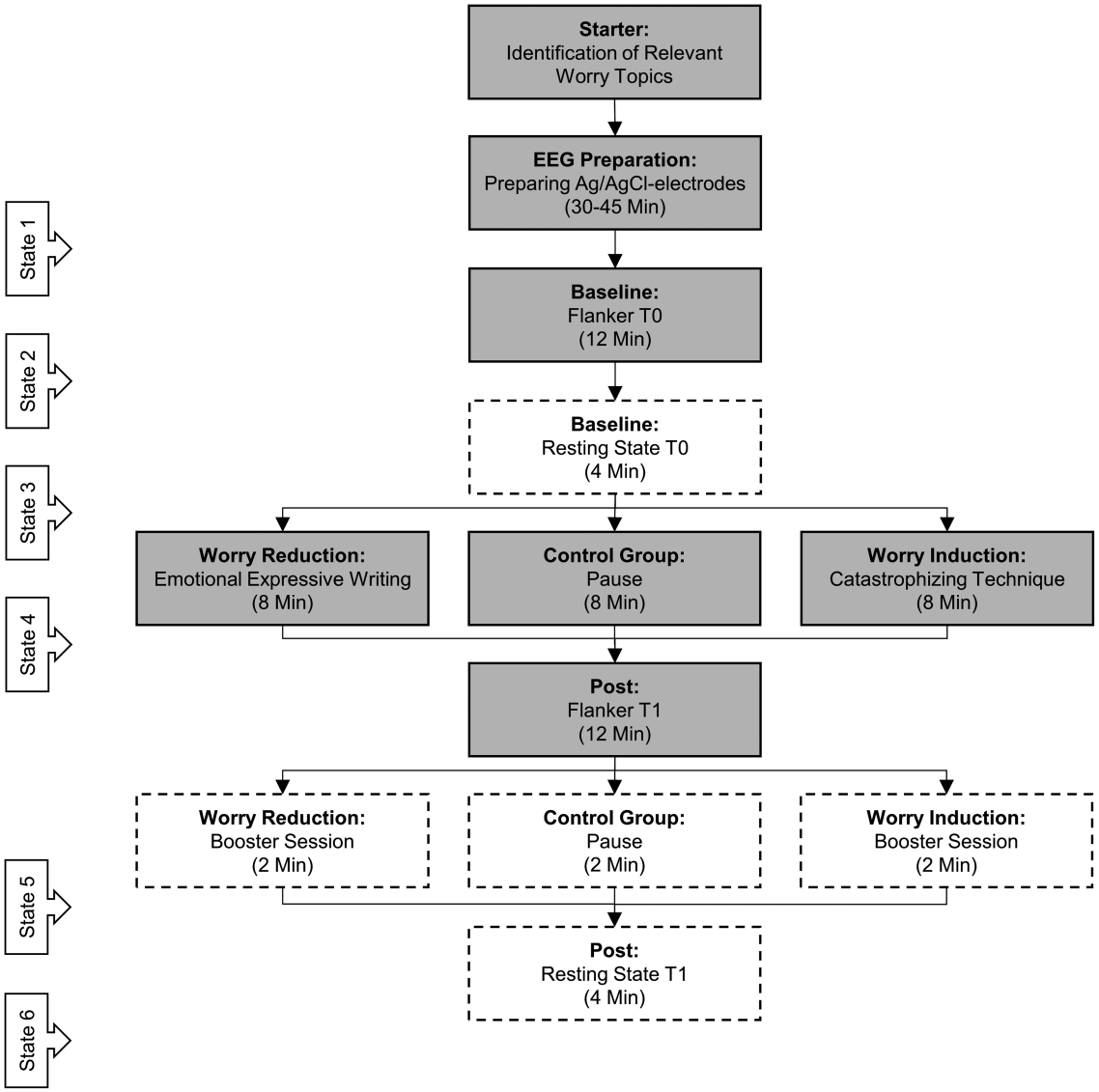

FIGURE 1 Flowchart of the study procedure. Boxes with surrounding dashed lines were part of another research question whose results will be presented elsewhere. Duration of each task is approximative. State measurements throughout the study ensured the tracking of fluctuations in state worry, state arousal,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

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

2.3 | Questionnaires

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

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

2.4 | Task

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

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

2.5 | Electrophysiological recording and processing

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

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

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

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

2.6 | Data analysis

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

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

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

3 | RESULTS

3.1 | Demographic and clinical data

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

3.2 | Manipulation check

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

TABLE 1 Demographical and clinical data of all groups

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

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

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

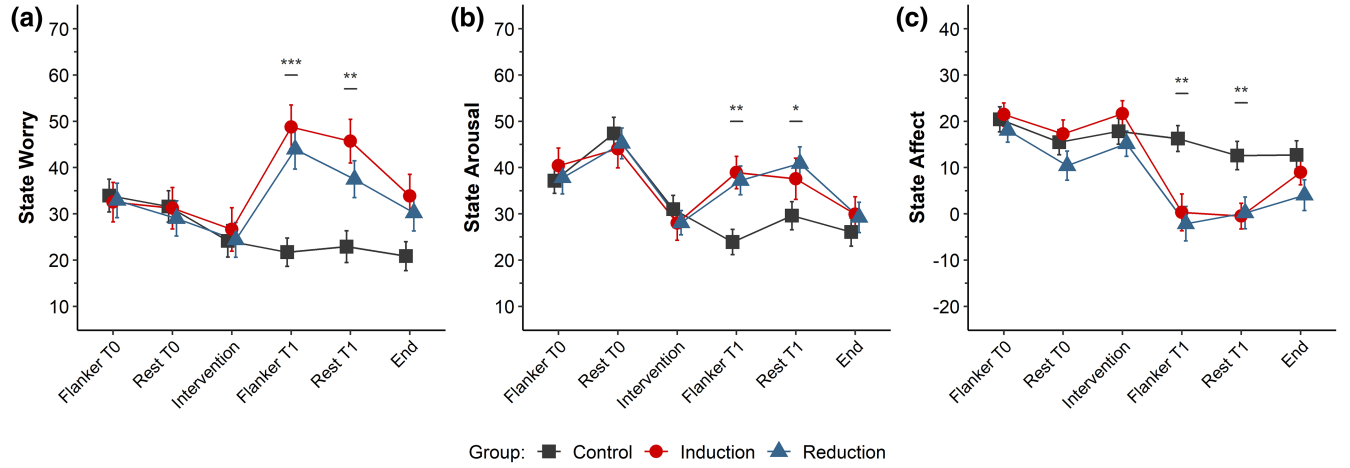

FIGURE 2 State measurements of state worry, state arousal, and state affect. State measurements tracked fluctuations of state worry (a), state arousal (b), and state affect (c) throughout the study and were assessed before each task. Each scale consisted of two items that were rated on a visual analogue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

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

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

3.3 | Event-related potentials

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

3.3.1 | Cross-sectional analyses of trait worry and ERPs

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

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

TABLE 2 Group-specific electrophysiological and behavioral data

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

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

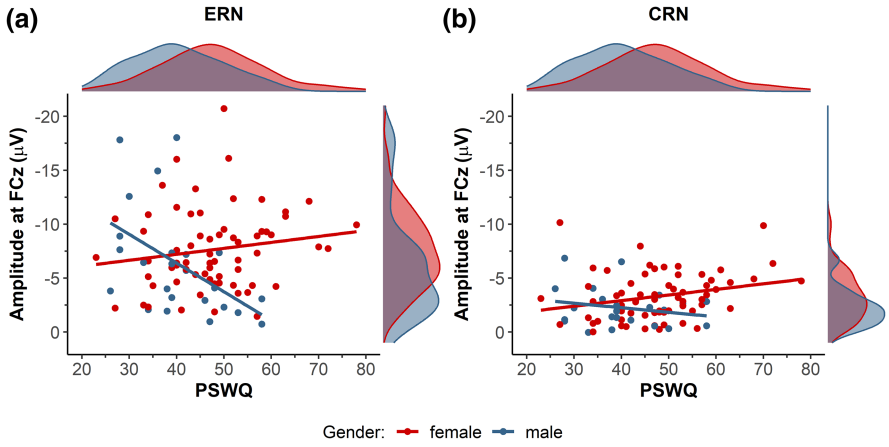

FIGURE 3 Scatter plots depicting the relationship between trait worry and ERN (a) and CRN (b). CRN, correct-response negativity; ERN, error-related negativity; PSWQ, Penn State Worry Questionnaire. Data points, regression lines, and densities were grouped by gender.

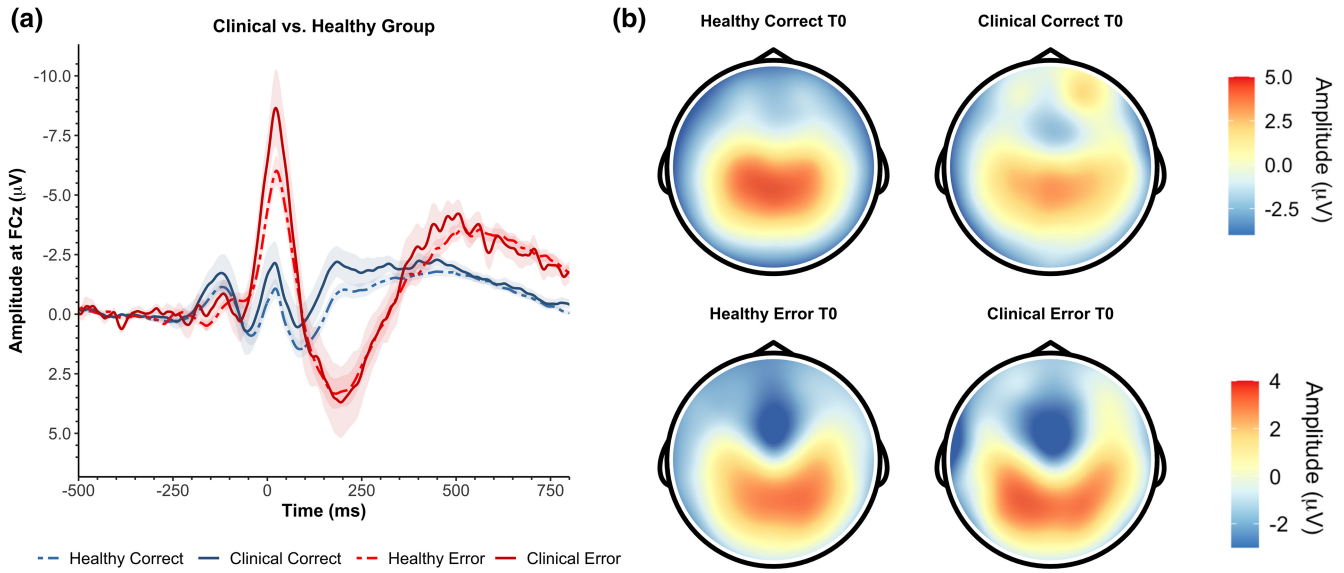

FIGURE 4 Response-locked grand-averaged waveforms of correct and erroneous trials at baseline (T0) for the clinical group with a history of internalizing disorders and the unaffected healthy group (a). Corresponding topographic head maps of the ERN and CRN (b)

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

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

3.3.2 | Longitudinal analyses of state worry and ERPs

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

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

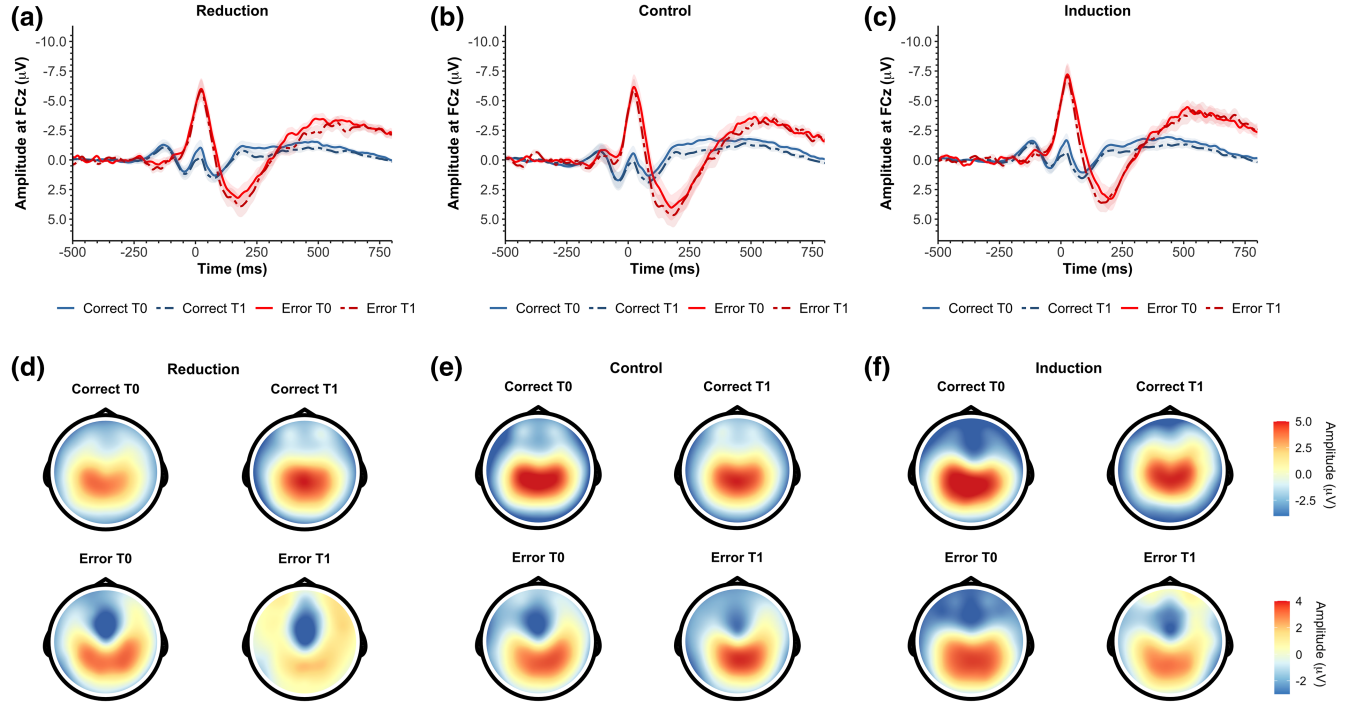

FIGURE 5 Response-locked grand-averaged waveforms of correct and erroneous trials at baseline (T0) and after the intervention (T1) for the worry reduction group (a), passive control group (b), and worry induction group (c). Corresponding topographic head maps of the ERN and CRN for the worry reduction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

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

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

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

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

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

3.4 | Behavioral data

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

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

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

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

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

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

3.4.2 | Longitudinal analyses of state worry and behavioral data

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

4 | DISCUSSION

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

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

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

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

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

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

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

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

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

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

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

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AUTHOR CONTRIBUTIONS

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

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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REFERENCES

Anokhin, A. P., Golosheykin, S., & Heath, A. C. (2008). Heritability of frontal brain function related to action monitoring. *Psychophysiology*, *45*(4), 524–534. https://doi.org/10.1111/j.1469-8986.2008.00664.x

Arch, J. J., & Craske, M. G. (2006). Mechanisms of mindfulness: Emotion regulation following a focused breathing induction. *Behaviour Research and Therapy*, *44*(12), 1849–1858. https://doi.org/10.1016/j.brat.2005.12.007

Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., & Monteiro, M. G. (2001). *AUDIT: The Alcohol Use Disorders Identification Test: Guidelines for use in primary health care* ((2nd ed.) ed.). World Health Organization https://apps.who.int/iris/handle/10665/67205

Baddeley, J. L., & Pennebaker, J. W. (2011). A postdeployment expressive writing intervention for military couples: A randomized controlled trial. *Journal of Traumatic Stress*, *24*(5), 581–585. https://doi.org/10.1002/jts.20679

Bakic, J., Jepma, M., De Raedt, R., & Pourtois, G. (2014). Effects of positive mood on probabilistic learning: Behavioral and electrophysiological correlates. *Biological Psychology*, *103*, 223–232. https://doi.org/10.1016/j.biopsycho.2014.09.012

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory-II*. Psychological Corporation.

Beesdo-Baum, K., Zaudig, M., & Wittchen, H. (2019). *Strukturiertes Klinisches Interview für DSM-5-Störungen—Klinische Version [The Structured Clinical Interview for DSM-5 Disorders—Clinical Version]*. Hogrefe.

Boehnke, K., Schwartz, S., Stromberg, C., & Sagiv, L. (1998). The structure and dynamics of worry: Theory, measurement, and cross-national replications. *Journal of Personality*, *66*(5), 745–782. https://doi.org/10.1111/1467-6494.00031

Borkovec, T. D., & Inz, J. (1990). The nature of worry in generalized anxiety disorder: A predominance of thought activity. *Behaviour Research and Therapy*, *28*(2), 153–158. https://doi.org/10.1016/0005-7967(90)90027-G

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, *108*(3), 624–652. https://doi.org/10.1037/0033-295X.108.3.624

Carlson, J. M., Fang, L., & Andrzejewski, J. A. (2021). No change in electrocortical measures of performance monitoring in high trait anxious individuals following multi-session attention bias modification training. *Neuroimage: Reports*, *1*(4), 100067. https://doi.org/10.1016/j.ynirp.2021.100067

Carrasco, M., Harbin, S. M., Nienhuis, J. K., Fitzgerald, K. D., Gehring, W. J., & Hanna, G. L. (2013). Increased error-related brain activity in youth with obsessive-compulsive disorder and unaffected siblings. *Depression and Anxiety*, *30*(1), 39–46. https://doi.org/10.1002/da.22035

Cavanagh, J. F., & Shackman, A. J. (2015). Frontal midline theta reflects anxiety and cognitive control: Meta-analytic evidence. *Journal of Physiology-Paris*, *109*(1), 3–15. https://doi.org/10.1016/j.jphysparis.2014.04.003

Cohen, J. (1992). A power primer. *Psychological Bulletin*, *112*(1), 155–159. https://doi.org/10.1037/0033-2909.112.1.155

Dutilh, G., van Ravenzwaaij, D., Nieuwenhuis, S., van der Maas, H. L. J., Forstmann, B. U., & Wagenmakers, E.-J. (2012). How to measure post-error slowing: A confound and a simple solution. *Journal of Mathematical Psychology*, *56*(3), 208–216. https://doi.org/10.1016/j.jmp.2012.04.001

Endrass, T., Riesel, A., Kathmann, N., & Buhlmann, U. (2014). Performance monitoring in obsessive–compulsive disorder and social anxiety disorder. *Journal of Abnormal Psychology*, *123*(4), 705–714. https://doi.org/10.1037/abn0000012

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics*, *16*(1), 143–149. https://doi.org/10.3758/BF03203267

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components: II Error processing in choice reaction tasks. *Electroencephalography & Clinical Neurophysiology*, *78*(6), 447–455. https://doi.org/10.1016/0013-4694(91)90062-9

Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods*, *41*, 1149–1160 http://gpower.hhu.de

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*, 175–191 http://gpower.hhu.de

First, M., Williams, J., Karg, R., & Spitzer, R. (2016). *Structured clinical interview for DSM-5 disorders, clinician version (SCID-5-CV)*. American Psychiatric Association.

Fischer, A. G., Danielmeier, C., Villringer, A., Klein, T. A., & Ullsperger, M. (2016). Gender influences on brain responses to errors and post-error adjustments. *Scientific Reports*, *6*(1), 24435. https://doi.org/10.1038/srep24435

Fisher, A. J., & Newman, M. G. (2013). Heart rate and autonomic response to stress after experimental induction of worry versus

relaxation in healthy, high-worry, and generalized anxiety disorder individuals. *Biological Psychology*, *93*(1), 65–74. https://doi.org/10.1016/j.biopsycho.2013.01.012

Foa, E. B., Huppert, J. D., Leiberg, S., Langner, R., Kichic, R., Hajcak, G., & Salkovskis, P. M. (2002). The Obsessive-Compulsive Inventory: Development and validation of a short version. *Psychological Assessment*, *14*(4), 485–496. https://doi.org/10.1037/1040-3590.14.4.485

Frattaroli, J. (2006). Experimental disclosure and its moderators: A meta-analysis. *Psychological Bulletin*, *132*(6), 823–865. https://doi.org/10.1037/0033-2909.132.6.823

Gehring, W. J., Goss, B., Coles, M. G., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science*, *4*(6), 385–390. https://doi.org/10.1111/j.1467-9280.1993.tb00586.x

Glöckner-Rist, A., & Rist, F. (2014). Deutsche Version des Penn State Worry Questionnaire (PSWQ-d) [German version of the Penn State Worry Questionnaire]. *Zusammenstellung sozialwissenschaftlicher Items und Skalen*, 1–26. https://doi.org/10.6102/zis219

Goldman, N., Dugas, M. J., Sexton, K. A., & Gervais, N. J. (2007). The impact of written exposure on worry: A preliminary investigation. *Behavior Modification*, *31*(4), 512–538. https://doi.org/10.1177/0145445506298651

Gönner, S., Leonhart, R., & Ecker, W. (2007). Das Zwangsinventar OCI-R - Die deutsche Version des Obsessive-Compulsive Inventory-Revised [The German Version of the Obsessive-Compulsive Inventory-Revised]. *Psychotherapie Psychosomatik Medizinische Psychologie*, *57*(09/10), 395–404. https://doi.org/10.1055/s-2007-970894

Gorka, S. M., Burkhouse, K. L., Klumpp, H., Kennedy, A. E., Afshar, K., Francis, J., Ajilore, O., Mariouw, S., Craske, M. G., Langenecker, S., Shankman, S. A., & Phan, K. L. (2018). Error-related brain activity as a treatment moderator and index of symptom change during cognitive-behavioral therapy or selective serotonin reuptake inhibitors. *Neuropsychopharmacology*, *43*(6), 1355–1363. https://doi.org/10.1038/npp.2017.289

Gortner, E.-M., Rude, S. S., & Pennebaker, J. W. (2006). Benefits of expressive writing in lowering rumination and depressive symptoms. *Behavior Therapy*, *37*(3), 292–303. https://doi.org/10.1016/j.beth.2006.01.004

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *The American Journal of Psychiatry*, *160*(4), 636–645. https://doi.org/10.1176/appi.ajp.160.4.636

Hajcak, G., Franklin, M. E., Foa, E. B., & Simons, R. F. (2008). Increased error-related brain activity in pediatric obsessive-compulsive disorder before and after treatment. *American Journal of Psychiatry*, *165*(1), 116–123. https://doi.org/10.1176/appi.ajp.2007.07010143

Hajcak, G., Klawohn, J., & Meyer, A. (2019). The utility of event-related potentials in clinical psychology. *Annual Review of Clinical Psychology*, *15*(1), 71–95. https://doi.org/10.1146/annurev-clinpsy-050718-095457

Hajcak, G., McDonald, N., & Simons, R. F. (2003). Anxiety and error-related brain activity. *Biological Psychology*, *64*(1-2), 77–90. https://doi.org/10.1016/S0301-0511(03)00103-0

Härpfer, K., Carsten, H. P., & Riesel, A. (2020, August 31). *The modulating effect of trait and state worry on the error-related negativity (ERN)*. https://osf.io/j8k6z

Härpfer, K., Carsten, H. P., Spychalski, D., Kathmann, N., & Riesel, A. (2020). Were we erring? The impact of worry and arousal on error-related negativity in a non-clinical sample. *Psychophysiology*, *57*(11), e13661. https://doi.org/10.1111/psyp.13661

Hautzinger, M., Keller, F., & Kühner, C. (2006). *Beck Depressions-Inventar (BDI-II)*. Harcourt Test Services.

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review*, *109*(4), 679–709. https://doi.org/10.1037/0033-295X.109.4.679

JASP Team. (2021). *JASP (Version 0.15.0.0) [Computer software]*. https://jasp-stats.org/

Jung, T.-P., Makeig, S., Humphries, C., Lee, T.-W., McKeown, M. J., Iragui, V., & Sejnowski, T. J. (2000). Removing electroencephalographic artifacts by blind source separation. *Psychophysiology*, *37*(2), 163–178. https://doi.org/10.1111/1469-8986.3720163

Keysers, C., Gazzola, V., & Wagenmakers, E.-J. (2020). Using Bayes factor hypothesis testing in neuroscience to establish evidence of absence. *Nature Neuroscience*, *23*(7), 788–799. https://doi.org/10.1038/s41593-020-0660-4

Klawohn, J., Hajcak, G., Amir, N., Kathmann, N., & Riesel, A. (2020). Application of attentional bias modification training to modulate hyperactive error-monitoring in OCD. *International Journal of Psychophysiology*, *156*, 79–86. https://doi.org/10.1016/j.ijpsycho.2020.07.005

Klawohn, J., Meyer, A., Weinberg, A., & Hajcak, G. (2020). Methodological choices in event-related potential (ERP) research and their impact on internal consistency reliability and individual differences: An examination of the error-related negativity (ERN) and anxiety. *Journal of Abnormal Psychology*, *129*(1), 29–37. https://doi.org/10.1037/abn0000458

Kujawa, A., Weinberg, A., Bunford, N., Fitzgerald, K. D., Hanna, G. L., Monk, C. S., Kennedy, A. E., Klumpp, H., Hajcak, G., & Phan, K. L. (2016). Error-related brain activity in youth and young adults before and after treatment for generalized or social anxiety disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *71*, 162–168. https://doi.org/10.1016/j.pnpbp.2016.07.010

Ladouceur, C. D., Tan, P. Z., Sharma, V., Bylsma, L. M., Silk, J. S., Siegle, G. J., Forbes, E. E., McMakin, D. L., Dahl, R. E., Kendall, P. C., Mannarino, A., & Ryan, N. D. (2018). Error-related brain activity in pediatric anxiety disorders remains elevated following individual therapy: A randomized clinical trial. *Journal of Child Psychology and Psychiatry*, *59*(11), 1152–1161. https://doi.org/10.1111/jcpp.12900

Larson, M. J., Gray, A. C., Clayson, P. E., Jones, R., & Kirwan, C. B. (2013). What are the influences of orthogonally-manipulated valence and arousal on performance monitoring processes? The effects of affective state. *International Journal of Psychophysiology*, *87*(3), 327–339. https://doi.org/10.1016/j.ijpsycho.2013.01.005

Larson, M. J., Perlstein, W. M., Stigge-Kaufman, D., Kelly, K. G., & Dotson, V. M. (2006). Affective context-induced modulation of the error-related negativity. *NeuroReport*, *17*(3). https://doi.org/10.1097/01.wnr.0000199461.01542.db

Larson, M. J., South, M., & Clayson, P. E. (2011). Sex differences in error-related performance monitoring. *NeuroReport*, *22*(1), 44–48. https://doi.org/10.1097/WNR.0b013e3283427403

Laux, L., Glanzmann, P., Schaffner, P., & Spielberger, C. D. (1981). *Das State-Trait-Angstinventar (STAI) [The State-Trait Anxiety Inventory (STAI)]*. Beltz.

Lin, Y., Moran, T. P., Schroder, H. S., & Moser, J. S. (2015). The role of hand of error and stimulus orientation in the relationship between worry and error-related brain activity: Implications for theory and practice. *Psychophysiology*, *52*(10), 1281–1292. https://doi.org/10.1111/psyp.12470

Loffing, F., Sölter, F., & Hagemann, N. (2014). Left preference for sport tasks does not necessarily indicate left-handedness: Sport-specific lateral preferences, relationship with handedness and implications for laterality research in behavioural sciences. *PLOS ONE*, *9*(8), e105800. https://doi.org/10.1371/journal.pone.0105800

Luck, S. J. (2014). *An introduction to the event-related potential technique* ((2nd ed.). ed.). MIT Press.

Luijten, M., Machielsen, M. W. J., Veltman, D. J., Hester, R., de Haan, L., & Franken, I. H. A. (2014). Systematic review of ERP and fMRI studies investigating inhibitory control and error processing in people with substance dependence and behavioural addictions. *Journal of Psychiatry & Neuroscience*, *39*(3), 149–169. https://doi.org/10.1503/jpn.130052

Lutz, M. C., Kok, R., Verveer, I., Malbec, M., Koot, S., van Lier, P. A. C., & Franken, I. H. A. (2021). Diminished error-related negativity and error positivity in children and adults with externalizing problems and disorders: A meta-analysis on error processing. *Journal of Psychiatry and Neuroscience*, *46*(6), E615. https://doi.org/10.1503/jpn.200031

Lyonfields, J. D., Borkovec, T. D., & Thayer, J. F. (1995). Vagal tone in generalized anxiety disorder and the effects of aversive imagery and worrisome thinking. *Behavior Therapy*, *26*(3), 457–466. https://doi.org/10.1016/S0005-7894(05)80094-2

Manoach, D. S., & Agam, Y. (2013). Neural markers of errors as endophenotypes in neuropsychiatric disorders. *Frontiers in Human Neuroscience*, *7*, 350. https://doi.org/10.3389/fnhum.2013.00350

Mathews, C. A., Perez, V. B., Delucchi, K. L., & Mathalon, D. H. (2012). Error-related negativity in individuals with obsessive–compulsive symptoms: Toward an understanding of hoarding behaviors. *Biological Psychology*, *89*(2), 487–494. https://doi.org/10.1016/j.biopsycho.2011.12.018

McLaughlin, K. A., Borkovec, T. D., & Sibrava, N. J. (2007). The effects of worry and rumination on affect states and cognitive activity. *Behavior Therapy*, *38*(1), 23–38. https://doi.org/10.1016/j.beth.2006.03.003

Meyer, A., & Gawlowska, M. (2017). Evidence for specificity of the impact of punishment on error-related brain activity in high versus low trait anxious individuals. *International Journal of Psychophysiology*, *120*, 157–163. https://doi.org/10.1016/j.ijpsycho.2017.08.001

Meyer, A., Gibby, B., Wissemann, K., Klawohn, J., Hajcak, G., & Schmidt, N. B. (2020). A brief, computerized intervention targeting error sensitivity reduces the error-related negativity. *Cognitive, Affective, & Behavioral Neuroscience*, *20*(1), 172–180. https://doi.org/10.3758/s13415-019-00760-w

Meyer, A., Hajcak, G., Torpey-Newman, D. C., Kujawa, A., & Klein, D. N. (2015). Enhanced error-related brain activity in children predicts the onset of anxiety disorders between the ages of 6 and 9. *Journal of Abnormal Psychology*, *124*(2), 266–274. https://doi.org/10.1037/abn0000044

Meyer, A., Weinberg, A., Klein, D. N., & Hajcak, G. (2012). The development of the error-related negativity (ERN) and its relationship with anxiety: Evidence from 8 to 13 year-olds. *Developmental Cognitive Neuroscience*, *2*(1), 152–161. https://doi.org/10.1016/j.dcn.2011.09.005

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy*, *28*(6), 487–495. https://doi.org/10.1016/0005-7967(90)90135-6

Moran, T. P., Taylor, D., & Moser, J. S. (2012). Sex moderates the relationship between worry and performance monitoring brain activity in undergraduates. *International Journal of Psychophysiology*, *85*(2), 188–194. https://doi.org/10.1016/j.ijpsycho.2012.05.005

Moser, J. S., Moran, T. P., & Jendrusina, A. A. (2012). Parsing relationships between dimensions of anxiety and action monitoring brain potentials in female undergraduates. *Psychophysiology*, *49*(1), 3–10. https://doi.org/10.1111/j.1469-8986.2011.01279.x

Moser, J. S., Moran, T. P., Kneip, C., Schroder, H. S., & Larson, M. J. (2016). Sex moderates the association between symptoms of anxiety, but not obsessive compulsive disorder, and error-monitoring brain activity: A meta-analytic review. *Psychophysiology*, *53*(1), 21–29. https://doi.org/10.1111/psyp.12509

Moser, J. S., Moran, T. P., Schroder, H. S., Donnellan, M. B., & Yeung, N. (2013). On the relationship between anxiety and error monitoring: A meta-analysis and conceptual framework. *Frontiers in Human Neuroscience*, *7*, 466. https://doi.org/10.3389/fnhum.2013.00466

Nelson, B. D., Jackson, F., Amir, N., & Hajcak, G. (2015). Single-session attention bias modification and error-related brain activity. *Cognitive, Affective, & Behavioral Neuroscience*, *15*(4), 776–786. https://doi.org/10.3758/s13415-015-0365-4

Nigbur, R., & Ullsperger, M. (2020). Funny kittens: Positive mood induced via short video-clips affects error processing but not conflict control. *International Journal of Psychophysiology*, *147*, 147–155. https://doi.org/10.1016/j.ijpsycho.2019.11.007

Oathes, D. J., Ray, W. J., Yamasaki, A. S., Borkovec, T. D., Castonguay, L. G., Newman, M. G., & Nitschke, J. (2008). Worry, generalized anxiety disorder, and emotion: Evidence from the EEG gamma band. *Biological Psychology*, *79*(2), 165–170. https://doi.org/10.1016/j.biopsycho.2008.04.005

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia*, *9*(1), 97–113. https://doi.org/10.1016/0028-3932(71)90067-4

Olvet, D. M., & Hajcak, G. (2008). The error-related negativity (ERN) and psychopathology: Toward an endophenotype. *Clinical Psychology Review*, *28*(8), 1343–1354. https://doi.org/10.1016/j.cpr.2008.07.003

Olvet, D. M., & Hajcak, G. (2009). The stability of error-related brain activity with increasing trials. *Psychophysiology*, *46*(5), 957–961. https://doi.org/10.1111/j.1469-8986.2009.00848.x

Pasion, R., & Barbosa, F. (2019). ERN as a transdiagnostic marker of the internalizing-externalizing spectrum: A dissociable meta-analytic effect. *Neuroscience and Biobehavioral Reviews*, *103*, 133–149. https://doi.org/10.1016/j.neubiorev.2019.06.013

Pennebaker, J. W., & Beall, S. K. (1986). Confronting a traumatic event: Toward an understanding of inhibition and disease. *Journal of Abnormal Psychology*, *95*(3), 274–281. https://doi.org/10.1037//0021-843x.95.3.274

Pennebaker, J. W., & Francis, M. E. (1996). Cognitive, emotional, and language processes in disclosure. *Cognition and Emotion*, *10*(6), 601–626. https://doi.org/10.1080/026999396380079

Pfabigan, D. M., Pintzinger, N. M., Siedek, D. R., Lamm, C., Derntl, B., & Sailer, U. (2013). Feelings of helplessness increase ERN amplitudes in healthy individuals. *Neuropsychologia*, *51*(4), 613–621. https://doi.org/10.1016/j.neuropsychologia.2012.12.008

Proudfit, G. H., Inzlicht, M., & Mennin, D. S. (2013). Anxiety and error monitoring: The importance of motivation and emotion. *Frontiers in Human Neuroscience*, *7*, 636. https://doi.org/10.3389/fnhum.2013.00636

Raftery, A. E. (1995). Bayesian Model Selection in Social Research. *Sociological Methodology*, *25*, 111–163. https://doi.org/10.2307/271063

Ramirez, G., & Beilock, S. L. (2011). Writing about testing worries boosts exam performance in the classroom. *Science*, *331*(6014), 211–213. https://doi.org/10.1126/science.1199427

Ray, W. J., Molnar, C., Aikins, D., Yamasaki, A., Newman, M. G., Castonguay, L., & Borkovec, T. D. (2009). Startle response in generalized anxiety disorder. *Depression and Anxiety*, *26*(2), 147–154. https://doi.org/10.1002/da.20479

Reinhold, M., Bürkner, P.-C., & Holling, H. (2018). Effects of expressive writing on depressive symptoms—A meta-analysis. *Clinical Psychology: Science and Practice*, *25*(1), e12224. https://doi.org/10.1111/cpsp.12224

Riesel, A. (2019). The erring brain: Error-related negativity as an endophenotype for OCD—A review and meta-analysis. *Psychophysiology*, *56*(4), e13348. https://doi.org/10.1111/psyp.13348

Riesel, A., Endrass, T., Auerbach, L. A., & Kathmann, N. (2015). Overactive performance monitoring as an endophenotype for obsessive-compulsive disorder: Evidence from a treatment study. *American Journal of Psychiatry*, *172*(7), 665–673. https://doi.org/10.1176/appi.ajp.2014.14070886

Riesel, A., Endrass, T., Kaufmann, C., & Kathmann, N. (2011). Overactive error-related brain activity as a candidate endophenotype for obsessive-compulsive disorder: Evidence from unaffected first-degree relatives. *The American Journal of Psychiatry*, *168*(3), 317–324. https://doi.org/10.1176/appi.ajp.2010.10030416

Riesel, A., Goldhahn, S., & Kathmann, N. (2017). Hyperactive performance monitoring as a transdiagnostic marker: Results from health anxiety in comparison to obsessive–compulsive disorder. *Neuropsychologia*, *96*, 1–8. https://doi.org/10.1016/j.neuropsychologia.2016.12.029

Riesel, A., Kathmann, N., & Klawohn, J. (2019). Flexibility of error-monitoring in obsessive-compulsive disorder under speed and accuracy instructions. *Journal of Abnormal Psychology*, *128*(7), 671–677. https://doi.org/10.1037/abn0000463

Riesel, A., Klawohn, J., Grützmann, R., Kaufmann, C., Heinzel, S., Bey, K., Lennertz, L., Wagner, M., & Kathmann, N. (2019). Error-related brain activity as a transdiagnostic endophenotype for obsessive-compulsive disorder, anxiety and substance use disorder. *Psychological Medicine*, *49*(7), 1–11. https://doi.org/10.1017/S0033291719000199

Riesel, A., Weinberg, A., Endrass, T., Kathmann, N., & Hajcak, G. (2012). Punishment has a lasting impact on error-related brain activity. *Psychophysiology*, *49*(2), 239–247. https://doi.org/10.1111/j.1469-8986.2011.01298.x

Ruscio, A. M., & Borkovec, T. D. (2004). Experience and appraisal of worry among high worriers with and without generalized anxiety disorder. *Behaviour Research and Therapy*, *42*(12), 1469–1482. https://doi.org/10.1016/j.brat.2003.10.007

Sandre, A., Banica, I., Riesel, A., Flake, J., Klawohn, J., & Weinberg, A. (2020). Comparing the effects of different methodological decisions on the error-related negativity and its association with behaviour and gender. *International Journal of Psychophysiology*, *156*, 18–39. https://doi.org/10.1016/j.ijpsycho.2020.06.016

Saunders, B., & Inzlicht, M. (2020). Assessing and adjusting for publication bias in the relationship between anxiety and the error-related negativity. *International Journal of Psychophysiology*, *155*, 87–98. https://doi.org/10.1016/j.ijpsycho.2020.05.008

Sayer, N. A., Noorbaloochi, S., Frazier, P. A., Pennebaker, J. W., Orazem, R. J., Schnurr, P. P., Murdoch, M., Carlson, K. F., Gravely, A., & Litz, B. T. (2015). Randomized controlled trial of online expressive writing to address readjustment difficulties among U.S. Afghanistan and Iraq war veterans. *Journal of Traumatic Stress*, *28*(5), 381–390. https://doi.org/10.1002/jts.22047

Schroder, H. S., Moran, T. P., & Moser, J. S. (2018). The effect of expressive writing on the error-related negativity among individuals with chronic worry. *Psychophysiology*, *55*(2), e12990. https://doi.org/10.1111/psyp.12990

Shiels, K., & Hawk, L. W. (2010). Self-regulation in ADHD: The role of error processing. *Clinical Psychology Review*, *30*(8), 951–961. https://doi.org/10.1016/j.cpr.2010.06.010

Simons, R. F. (2010). The way of our errors: Theme and variations. *Psychophysiology*, *47*(1), 1–14. https://doi.org/10.1111/j.1469-8986.2009.00929.x

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the State-Trait Anxiety Inventory*. Consulting Psychologists Press.

Suor, J. H., Calentino, A. E., Granros, M., & Burkhouse, K. L. (2021). Maternal error-related negativity relationship with offspring error-related negativity and negative parenting styles: A novel model of internalizing psychopathology risk. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*. https://doi.org/10.1016/j.bpsc.2021.10.007

Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry*, *39*(4), 255–266. https://doi.org/10.1016/0006-3223(95)00136-0

Travagin, G., Margola, D., & Revenson, T. A. (2015). How effective are expressive writing interventions for adolescents? A meta-analytic review. *Clinical Psychology Review*, *36*, 42–55. https://doi.org/10.1016/j.cpr.2015.01.003

Unger, K., Kray, J., & Mecklinger, A. (2012). Worse than feared? Failure induction modulates the electrophysiological signature of error monitoring during subsequent learning. *Cognitive, Affective, & Behavioral Neuroscience*, *12*(1), 34–51. https://doi.org/10.3758/s13415-011-0061-y

van Doorn, J., van den Bergh, D., Böhm, U., Dablander, F., Derks, K., Draws, T., Etz, A., Evans, N. J., Gronau, Q. F., Haaf, J. M., Hinne, M., Kucharský, Š., Ly, A., Marsman, M., Matzke, D., Gupta, A. R. K. N., Sarafoglou, A., Stefan, A., Voelkel, J. G., & Wagenmakers, E.-J. (2021). The JASP guidelines for conducting and reporting a Bayesian analysis. *Psychonomic Bulletin & Review*, *28*(3), 813–826. https://doi.org/10.3758/s13423-020-01798-5

Vasey, M. W., & Borkovec, T. D. (1992). A catastrophizing assessment of worrisome thoughts. *Cognitive Therapy and Research*, *16*(5), 505–520. https://doi.org/10.1007/BF01175138

Verkuil, B., Brosschot, J. F., Putman, P., & Thayer, J. F. (2009). Interacting effects of worry and anxiety on attentional disengagement from threat. *Behaviour Research and Therapy*, *47*(2), 146–152. https://doi.org/10.1016/j.brat.2008.11.003

Vidal, F., Burle, B., Bonnet, M., Grapperon, J., & Hasbroucq, T. (2003). Error negativity on correct trials: A reexamination of available data. *Biological Psychology*, *64*(3), 265–282. https://doi.org/10.1016/S0301-0511(03)00097-8

Vidal, F., Hasbroucq, T., Grapperon, J., & Bonnet, M. (2000). Is the 'error negativity' specific to errors? *Biological Psychology*, *51*(2-3), 109–128. https://doi.org/10.1016/S0301-0511(99)00032-0

Watson, D., & Clark, L. A. (1991). *The Mood and Anxiety Symptom Questionnaire*. University of Iowa.

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology*, *104*(1), 3–14. https://doi.org/10.1037/0021-843X.104.1.3

Weinberg, A., Olvet, D. M., & Hajcak, G. (2010). Increased error-related brain activity in generalized anxiety disorder. *Biological Psychology*, *85*(3), 472–480. https://doi.org/10.1016/j.biopsycho.2010.09.011

Weinberg, A., Riesel, A., & Hajcak, G. (2012). Integrating multiple perspectives on error-related brain activity: The ERN as a neural indicator of trait defensive reactivity. *Motivation and Emotion*, *36*(1), 84–100. https://doi.org/10.1007/s11031-011-9269-y

Wiswede, D., Münte, T. F., Goschke, T., & Rüsseler, J. (2009). Modulation of the error-related negativity by induction of short-term negative affect. *Neuropsychologia*, *47*(1), 83–90. https://doi.org/10.1016/j.neuropsychologia.2008.08.016

Wiswede, D., Münte, T. F., & Rüsseler, J. (2009). Negative affect induced by derogatory verbal feedback modulates the neural signature of error detection. *Social Cognitive and Affective Neuroscience*, *4*(3), 227–237. https://doi.org/10.1093/scan/nsp015

Wolitzky-Taylor, K. B., & Telch, M. J. (2010). Efficacy of self-administered treatments for pathological academic worry: A randomized controlled trial. *Behaviour Research and Therapy*, *48*(9), 840–850. https://doi.org/10.1016/j.brat.2010.03.019

World Medical Association. (2013). World Medical Association declaration of Helsinki: Ethical principles for medical research involving human subjects. *JAMA*, *310*(20), 2191–2194. https://doi.org/10.1001/jama.2013.281053

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review*, *111*(4), 931–959. https://doi.org/10.1037/0033-295X.111.4.931

Zachariae, R., & O'Toole, M. S. (2015). The effect of expressive writing intervention on psychological and physical health outcomes in cancer patients—A systematic review and meta-analysis. *Psycho-Oncology*, *24*(11), 1349–1359. https://doi.org/10.1002/pon.3802

SUPPORTING INFORMATION

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

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

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

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

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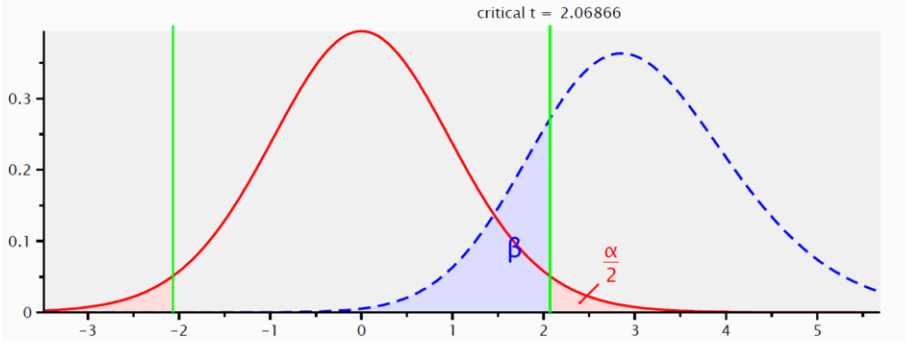
SUPPLEMENTARY MATERIALS

Figures

Figure S1

*Output with Specifications of the A Priori Sample Size Calculation Using G*Power, version 3.1.9.7.*



t tests – Means: Difference between two dependent means (matched pairs)

Analysis:	A priori: Compute required sample size		
Input:	Tail(s)	=	Two
	Effect size dz	=	0.6
	α err prob	=	0.05
	Power (1−β err prob)	=	0.80
Output:	Noncentrality parameter δ	=	2.9393877
	Critical t	=	2.0686576
	Df	=	23
	Total sample size	=	24
	Actual power	=	0.8036714

Tables

Table S1

Effects of Gender, PSWQ, and Gender × PSWQ on the ERN and CRN Using Different Scoring Approaches.

Measure		Variable	*b*	*SE*	*t*	*p*	R^2	*F*	*df*	*p*
ERN	Peak-to-Peak						0.13	4.23	3, 86	**.008**
		Gender	2.65	1.05	2.52	**.014**				
		PSWQ	-0.06	0.04	-1.23	.221				
		Gender × PSWQ	0.32	0.10	3.26	**.002**				
	Mean Amplitude						0.10	3.07	3, 86	**.032**
		Gender	2.33	1.17	1.99	**.049**				
		PSWQ	-0.08	0.05	-1.51	.135				
		Gender × PSWQ	0.30	0.11	2.78	**.007**				
	Area around the peak						0.12	3.76	3, 86	**.014**
		Gender	2.54	1.23	2.06	**.043**				
		PSWQ	-0.09	0.05	-1.64	.105				
		Gender × PSWQ	0.37	0.12	3.17	**.002**				
	ΔERN						0.07	2.14	3, 86	.102
		Gender	0.64	0.88	0.74	.464				
		PSWQ	-0.01	0.04	-0.23	.818				
		Gender × PSWQ	0.19	0.08	2.32	**.023**				
CRN	Peak-to-Peak						0.11	3.44	3, 86	**.020**
		Gender	1.20	0.56	2.16	**.034**				
		PSWQ	-0.05	0.02	-2.19	**.031**				
		Gender × PSWQ	0.09	0.05	1.80	.075				
	Mean Amplitude						0.11	3.60	3, 86	**.017**
		Gender	1.68	0.73	2.29	**.024**				
		PSWQ	-0.07	0.03	-2.12	**.037**				
		Gender × PSWQ	0.11	0.07	1.65	.103				
	Area around the peak						0.13	4.45	3, 86	**.006**
		Gender	1.99	0.80	2.50	**.014**				
		PSWQ	-0.08	0.03	-2.35	**.021**				
		Gender × PSWQ	0.12	0.07	1.57	.120				

Note. Correct-response negativity (CRN), error-related negativity (ERN); mean amplitude (0 – 100 ms); area around the peak (± 20 ms); peak-to-peak (difference between most positive pre-response and most negative post-response peak); ΔERN = error-correct mean amplitude (0–100 ms); all quantifications were based on electrode FCz with an early pre-response baseline of -500 to -300 ms; gender (0 = female, 1 = male); PSWQ = Penn State Worry Questionnaire (mean centered); significant $p < .05$ printed in bold.

Table S2

Results of Frequentist ANOVA for ERN and CRN Varying as Function of Time and Group Using Different Scoring Approaches.

	Peak-to-Peak				Mean Amplitude				Area around the peak				ΔERN			
Variable	*F*	*df*	η_p^2	*p*	*F*	*df*	η_p^2	*p*	*F*	*df*	η_p^2	*p*	*F*	*df*	η_p^2	*p*
Response	155.49	1, 87	0.64	**< .001**	149.28	1, 87	0.63	**< .001**	186.55	1, 87	0.68	**< .001**				
Response × Time	8.92	1, 87	0.09	**.004**	0.18	1, 87	0.00	.673	4.83	1, 87	0.05	**.031**				
Response × Group	0.46	2, 87	0.01	.632	0.26	2, 87	0.01	.774	0.11	2, 87	0.00	.897				
Response × Time × Group	0.77	2, 87	0.02	.465	0.33	2, 87	0.01	.719	0.76	2, 87	0.02	.472				
Time	0.21	1, 87	0.00	.648	15.15	1, 87	0.15	**< .001**	4.98	1, 87	0.05	**.028**	0.18	1, 87	0.00	.673
Time × Group	0.80	2, 87	0.02	.453	0.13	2, 87	0.00	.879	0.00	2, 87	0.00	.998	0.33	2, 87	0.01	.719
Group	0.80	2, 87	0.02	.453	0.82	2, 87	0.02	.446	0.59	2, 87	0.01	.557	0.26	2, 87	0.01	.774

Note. Correct-response negativity (CRN), error-related negativity (ERN), group (induction, reduction, control), response (correct, incorrect), time (T0, T1); Mean amplitude (0 – 100 ms); area around the peak (± 20 ms); peak-to-peak (difference between most positive pre-response and most negative post-response peak); ΔERN = error-correct mean amplitude (0–100 ms); all quantifications were based on electrode FCz with an early pre-response baseline of -500 to -300 ms; significant p <.05 printed in bold.

Study 2

Enhanced Performance Monitoring as a Transdiagnostic Risk Marker of the Anxiety and Obsessive-Compulsive Spectrum: The Role of Disorder Category, Clinical Status, Family Risk, and Anxiety Dimensions

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Enhanced performance monitoring as a transdiagnostic risk marker of the anxiety and obsessive-compulsive spectrum: The role of disorder category, clinical status, family risk, and anxiety dimensions

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Abstract

In this preregistered study, we investigated the relationship between neural correlates of performance monitoring and disorders of the anxiety and obsessive-compulsive spectrum. Specifically, we aimed at understanding the role of disorder category, clinical status, family risk, and the transdiagnostic symptom dimensions anxious apprehension and anxious arousal. To this end, we measured event-related potentials (ERPs) of performance monitoring (i.e., error-related negativity, ERN, and correct-response negativity, CRN) in a large sample of $n = 156$ participants, including groups of patients with obsessive-compulsive disorder, social anxiety disorder, and specific phobia, as well as a naturalistic control group. Contrary to our initial expectations, we did not observe significant differences in ERPs among the clinical groups, nor in comparison to the naturalistic control group. However, after creating a more strictly defined healthy control group, we found larger ERN amplitudes in the specific phobia compared with the healthy control group. In addition, when comparing participants with and without a lifetime clinical diagnosis of any internalizing disorder, regardless of their main diagnosis, as well as when comparing those with or without a family risk for internalizing psychopathology, we observed larger amplitudes for both ERN and CRN. Subsequently, we combined data from this study and a previously published subclinical study to examine the role of transdiagnostic symptom dimensions (i.e., anxious apprehension and anxious arousal) across a wider severity spectrum. In this joint sample of $n = 246$ participants, gender emerged as a moderator of the link between anxious apprehension and enhanced performance monitoring. Specifically, women with increasing anxious apprehension exhibited elevated ERN and CRN amplitudes. In conclusion, our study challenges the notion of a disorder-specific link to performance monitoring. Instead, our findings suggest that enhanced performance monitoring is associated with a higher propensity for anxious

apprehension and acts as a broad risk marker for internalizing psychopathology, reflecting vulnerability beyond diagnostic borders within the anxiety- and obsessive-compulsive spectrum.

Keywords: Anxiety, anxious apprehension, CRN, EEG, ERN, family risk

1. Introduction

Committing an error is a ubiquitous human experience that individuals often try to avoid. This is particularly plausible, given that in many cases, errors signal potential danger or even harm. Nonetheless, human learning is deeply rooted in trial-and-error approaches, and therefore, the commission of an error can be a potential birthplace for the improvement of future performance. As a result, cognitive, motivational, and behavioral adjustments based on past errors allow humans to constantly develop their repertoire of skills and abilities (Botvinick et al., 2001; Cavanagh & Shackman, 2015; Simons, 2010; Weinberg et al., 2012).

Over the past three decades, neuroscientific research has gained deeper insights into the neural correlate of error monitoring in the human electroencephalogram (EEG), that is the event-related potential (ERP) called error-related negativity (ERN; Gehring et al., 1993); or originally referred to as error negativity (N_E; Falkenstein et al., 1991). The ERN is a response-locked fronto-central potential peaking at around 50 ms after erroneous responses and it can be measured over the lifespan in children, adolescents, and adults aged 5 – 80 years (Davies et al., 2004a; Davies et al., 2004b; Nieuwenhuis et al., 2002; Torpey et al., 2009) with high internal consistency across tasks (Larson et al., 2010; Olvet & Hajcak, 2009b; Riesel et al., 2013) and with good test-retest reliability over an interval of two to six weeks (Larson et al., 2010; Olvet & Hajcak, 2009a; Segalowitz et al., 2010) and even up to two years (Weinberg & Hajcak, 2011). Another performance associated ERP is the correct-response negativity (CRN; Vidal et al., 2003; Vidal et al., 2000), elicited within a similar time interval after correct responses as the ERN, albeit with a relatively smaller amplitude. The interplay between ERN and CRN remains a subject of ongoing debate: One approach posits that both ERN and CRN are manifestations of an identical cognitive process (Falkenstein et al., 2000; Hoffmann & Falkenstein, 2010; Vidal et al.,

2000), differing solely in the magnitude of their expression. An alternative perspective assumes the existence of two distinct and independent underlying processes (Yordanova et al., 2004), while a third hypothesis suggests that they represent a shared process, encompassing both a more general cognitive mechanism and an error-specific one (Endrass et al., 2012).

As a result of the long-lasting and ongoing research in this field, an extensive body of theoretically and empirically based frameworks has evolved, aiming at describing the functional role of the ERN within the cognitive system. Early approaches initially conceptualized the ERN as a signal marking a mismatch between the representation of the required and the actual response (Falkenstein et al., 1991; Gehring et al., 1993). This was later superseded by the assumption that the ERN signals the conflict of two simultaneously activated response tendencies (Botvinick et al., 2001; Yeung et al., 2004). A third approach emphasized the importance of the ERN in reinforcement learning, such that the modification of future performance relies on the signaling whenever an outcome differs from what was initially predicted (Holroyd & Coles, 2002). What all three approaches have in common, is that they acknowledge the crucial role of the ERN in cognitive control functions, that the ERN likely signals the need for adjustment in order to fulfill the requirements of a changing environment, and that ERN is the starting point of a potential cascade of processes, ultimately culminating in improved future performance.

Additional informative conclusions for the functional role of the ERN can be derived from its neurobiological underpinnings. Utilizing source localization in both electro- (e.g., Dehaene et al., 1994) and magnetoencephalographic studies (e.g., Miltner et al., 2003), intracerebral single-unit recordings in primates (e.g., Godlove et al., 2011) and humans (e.g., Brázdil et al., 2005), functional magnetic resonance imaging (e.g., Ridderinkhof et al., 2004),

and the combination of neuroscientific methods (e.g., Debener et al., 2005; Grützmann et al., 2016), the anterior cingulate cortex (ACC) has been identified as one of the main generators of the ERN. The ACC consists of a high density of dopaminergic neurons (Lumme et al., 2007; Olver et al., 2010) and is widely connected to both "cognitive" prefrontal and "emotion processing" limbic regions (Bush et al., 2000; Tang et al., 2019). Accordingly, ACC activation is not limited solely to error responses; it extends to instances of fear (Mechias et al., 2010) and pain (Bushnell et al., 2013; Peyron et al., 2000), demonstrating its pivotal regulatory function as an integrating hub of both cognitive and emotional processes (Bush et al., 2000; Cavanagh & Shackman, 2015; Shackman et al., 2011; Shenhav et al., 2013). Because of its connections to other brain regions as well as its responsiveness to reward and punishment, the ACC is considered a central player in behavior regulation and the implementation of adaptive adjustments (Weinberg, Dieterich, et al., 2015).

The involvement of the ACC, and by extension, the ERN, in both cognitive and emotional processes shapes a prevailing perspective on the ERN not only as an index of an individuals' capacity of (i.e., working memory/executive functions; Larson & Clayson, 2011; Miller et al., 2012) and motivation for (i.e., reward/punishment; Riesel, Kathmann, et al., 2019; Riesel et al., 2012) task engagement, but also as an indicator of an individuals' trait-like sensitivity to internal threats (i.e., poor performance; Weinberg, Dieterich, et al., 2015). The role of the ERN as an index of internal threat sensitivity renders it particularly relevant for clinical research, as it can potentially refine our knowledge of the etiopathogenesis of mental disorders (Hajcak et al., 2019). Consequently, error monitoring has been included by the Research Domain Criteria Initiative (RDoC), which offers a framework that centers on neurobiological mechanisms involved in mental health and disorders (Cuthbert, 2014; Insel et al., 2010; Kozak &

Cuthbert, 2016). The RDoC matrix lists different domains of basic cognitive, emotional, motoric, regulatory, and social functions, each of which can be investigated using different units of analysis spanning from molecular and cellular levels to neural circuits, physiological responses, behavioral outcomes, self-reported measures, and experimental paradigms. Reflecting its role as an integrative hub of cognition and emotion, the ERN is listed as a physiological measure within two domains: cognitive systems (cognitive control: performance monitoring) and negative valence systems (sustained threat). Located at the central point of the units of analysis spectrum, stretching from cellular mechanisms to behavioral paradigms, the ERN represents a neurobiological substrate that holds considerable potential for unveiling the pathways leading to psychopathological conditions.

In fact, altered error monitoring has been proposed as a transdiagnostic endophenotype applicable to a range of mental disorders (Manoach & Agam, 2013; Olvet & Hajcak, 2008; Riesel, Klawohn, et al., 2019). An endophenotype represents a quantifiable component aiming to shed light on the complex processes that extend from genetic predisposition to the phenotypical expression of psychopathology (Gottesman & Gould, 2003). In order to qualify as an endophenotype, a neurobiological marker needs to fulfill certain criteria (Gottesman & Gould, 2003). Firstly, it needs to be associated with psychopathology. Previous meta-analytical studies found altered error monitoring to vary along the lines of internalizing and externalizing mental disorders (Lutz et al., 2021; Pasion & Barbosa, 2019). Enhanced ERN amplitudes have consistently been identified in anxiety disorders (Michael et al., 2021; Moser et al., 2013; Saunders & Inzlicht, 2020), obsessive-compulsive disorders (Bellato et al., 2021; Mathews et al., 2012; Riesel, 2019), and depression (Moran et al., 2017), although the relationship with depression is relatively moderate and characterized by considerable between-study heterogeneity.

Conversely, reduced ERN amplitudes have been observed in substance use disorder (Fairbairn et al., 2021; Webber et al., 2024), psychosis (Martin et al., 2018), psychopathy (Vallet et al., 2021), and attention deficit hyperactivity disorder (Bellato et al., 2021; Geburek et al., 2013; but see Kaiser et al., 2020). Secondly, psychopathology and the marker are required to co-segregate within families and to exhibit an increased prevalence among unaffected family members compared with the general population. Investigations involving individuals at risk for psychopathology revealed confirming evidence that unaffected participants with a family history of an anxiety, obsessive-compulsive, substance use, or attention deficit hyperactivity disorder showed a similar altered error monitoring like their (first-degree) relatives (Carrasco, Harbin, et al., 2013; Euser et al., 2013; McLoughlin et al., 2009; Riesel et al., 2011; Riesel, Klawohn, et al., 2019). Thirdly, the marker needs to be independent of symptomatic state, which was found in intervention studies measuring an unchanged increased ERN, despite the successful treatment of anxiety (Kujawa et al., 2016; Ladouceur et al., 2018) and obsessive-compulsive symptoms (Hajcak et al., 2008; Riesel et al., 2015). Lastly, the marker needs to be heritable, which was confirmed in transgenerational studies (Anokhin et al., 2008; Suor et al., 2021). In summary, there is compelling empirical evidence supporting the notion that enhanced error monitoring, as measured by enhanced ERN amplitudes, represents a promising transdiagnostic endophenotype applicable to anxiety and obsessive-compulsive disorders.

However, evidence from primary studies on elevated error monitoring within the anxiety and obsessive-compulsive spectrum mostly rely on case-control designs comparing a clinical and healthy control group. To the best of our knowledge, only few studies compared two or more disorder categories with each other and/ or a control group (e.g., Carrasco, Hong, et al., 2013; Endrass et al., 2014; Kujawa et al., 2016; Riesel et al., 2017; Weinberg, Kotov, et al., 2015).

Regarding meta-analyses in this research field, we do not know of any study that utilized a network approach allowing direct comparisons between different disorder categories. Again, to the best of our knowledge, all of the current available meta-analyses integrated effect sizes from classical case-control studies comparing a single clinical to a healthy control group or associational studies with correlations between symptom dimensions and ERPs of performance monitoring. Furthermore, healthy participants do not necessarily represent a naturalistic control group with characteristics of the general population, bearing the risk of overestimating differences. In summary, the inclusion of various clinical groups and more naturalistic control groups is essential to enhance the research design, to bolster the validity of findings, to facilitate comparisons, and to contribute to a more comprehensive understanding of the neural correlates of performance monitoring in psychopathology.

In addition, studies examining subclinical symptom levels have reported less consistent findings regarding enhanced ERN amplitudes (Clayson et al., 2023; Härpfer et al., 2020; Saunders & Inzlicht, 2020; Seow et al., 2020) and not all disorder categories within the anxiety and obsessive-compulsive spectrum appear to be characterized by enhanced error monitoring (Michael et al., 2021). Enhanced ERN amplitudes have been found in generalized anxiety disorder (GAD; Meyer et al., 2012; Weinberg et al., 2010), social anxiety disorder (SAD; Endrass et al., 2014; Kujawa et al., 2016), health anxiety (Riesel et al., 2017), and obsessive-compulsive disorder (OCD; Bellato et al., 2021; Mathews et al., 2012; Riesel, 2019), but not in post-traumatic stress disorder (PTSD; Gorka et al., 2016; Rabinak et al., 2013) and specific phobia (Hajcak et al., 2003; Moser et al., 2005). Concerning panic disorder, the available literature is limited, with only one study, featuring a relatively small sample size, identifying an enhanced ERN in individuals with a primary diagnosis of panic disorder (Valt et al., 2018).

Finally and to the best of our knowledge, there is no available literature exploring potential alterations of error monitoring in agoraphobia.

Inconsistent findings between disorder categories may stem from variations in latent symptom dimension profiles inherent to anxiety and obsessive-compulsive disorders (Cox et al., 2010; Krueger, 1999). Specifically, the presence of enhanced error monitoring appears to be closely tied to the dimension of anxious apprehension rather than anxious arousal, a notion supported by primary studies (Hajcak et al., 2003; Lin et al., 2015; Moran et al., 2012; Moser et al., 2005; Moser et al., 2012; Weinberg et al., 2010; but see Härpfer et al., 2020; Macedo et al., 2021) as well as meta-analyses (Moser et al., 2013; Saunders & Inzlicht, 2020). Additionally, some of this research suggests that the link between anxious apprehension and enhanced error monitoring is more dominant in women (Moser et al., 2016). These transdiagnostic findings are relevant insofar as they might be translated into diagnostic categories, whereby disorders characterized more prominently by anxious apprehension exhibit stronger associations with ERN variation. For instance, GAD, SAD, and OCD appear to be more closely linked to ERN enhancements, in contrast to PTSD and specific phobia. Consequently, a dimensional and transdiagnostic conceptualization of the anxiety and obsessive-compulsive spectrum may provide a more comprehensive understanding of the transdiagnostic anxiety dimensions (i.e., anxious apprehension and anxious arousal), that are associated with enhanced error monitoring, while broad disorder categories are likely to overshadow specific associations.

In this preregistered study (https://osf.io/kxv5h), our primary objective was to gain a more thorough understanding of the observed associations between ERN and disorders of the anxiety and obsessive-compulsive spectrum, by adopting a transdiagnostic and dimensional approach including various anxiety related disorder categories and dimensions. To this end,

patients with disorders that were likely to have different characteristics in anxious apprehension and anxious arousal were recruited. Specifically, performance monitoring associated ERPs (i.e., ERN and CRN) of n = 156 participants who were diagnosed with obsessive-compulsive disorder (OCD), social anxiety disorder (SAD), or specific phobia (PHOB) as well as control participants (CON) were measured while performing a flanker task. In a subsequent step, we integrated the data from this predominantly clinical sample with data from a subclinical study (Härpfer et al., 2022), resulting in a sample of n = 246 participants with a broad range across the symptom severity spectrum. In our first (preregistered) research question, we aimed to explore the disorder-specific links of the diagnostic groups with alterations in the ERN: We expected that ERN amplitudes of the OCD and SAD group would be larger than those of the PHOB and CON group. Furthermore, we expected no differences between the OCD and SAD group as well as between the PHOB and CON group. In our second (non-preregistered) research question, we were further interested in the role of clinical status of an internalizing disorder as well as family risk for internalizing psychopathology on the ERN: We anticipated that both clinical status and family risk would be associated with larger ERN amplitudes. The third (preregistered) research question aimed at investigating the dimensional relationships between anxiety dimensions and enhanced ERN amplitudes: We predicted anxious apprehension, but not anxious arousal, would be linked to heightened ERN amplitudes, with this association being stronger for clinical participants. The last (non-preregistered) research question focused on the moderating role of gender in the relationship between anxious apprehension and ERN enhancement: We hypothesized that this link would be more pronounced for women compared with men. To test the specificity of the effects on the ERN, we investigated the CRN in the same manner as the ERN for all research questions. However, these CRN analyses were fully exploratory, given the

limited existing evidence regarding the relationship between anxiety and the CRN (Michael et al., 2021).

2. Method

2.1 Participants

Using G*Power, version 3.1.9.7 (Faul et al., 2009; Faul et al., 2007), an a priori sample size calculation was conducted. Based on the results of previous meta-analyses (Moser et al., 2013; Riesel, 2019; Saunders & Inzlicht, 2020), we assumed medium-sized effects in our mixed ANOVA. A within-between interaction of response type (correct, incorrect) × group (OCD, SAD, PHOB, CON) of medium size (Cohen's $f > 0.28$), indicating a group effect on the ERN but not the CRN, can be detected with a sample size of $n = 36$ per group, a power of 80 %, and an alpha of 0.05 (Cohen, 1992). Specifications of the sample size calculation can be found in the supplementary materials (Figure S1). Therefore, we recruited 160 participants including individuals with OCD, SAD, PHOB and control participants ($n = 40$ per group) to ensure a sufficient power while expecting a dropout rate of 10% due to the exclusion criteria.

Our final sample consisted of $n = 156$ right-handed participants ($n = 115$ identified as female) aged 18 to 63 years ($M = 28.51$, $SD = 8.07$). The OCD, SAD, PHOB, and CON group consisted of each $n = 39$ participants with a corresponding main diagnosis. Regarding the clinical status of an internalizing disorder and the family risk for internalizing psychopathology, there were clinical participants with family risk ($n = 88$) and without family risk ($n = 34$), as well as nonclinical participants with family risk ($n = 15$) and without family risk ($n = 19$) in our sample. The group with a clinical status of an internalizing disorder encompassed all participants from the OCD, SAD, and PHOB groups as well as five participants from the control group diagnosed with depression, sleeping disorder, PTSD, and/or skin picking. The group with a family risk for

internalizing psychopathology encompassed participants with a first-degree relative suffering from at least one disorder of the anxiety and obsessive-compulsive spectrum, depression, skin picking, trichotillomania, tics, and/or body dysmorphia.

Participants were recruited via the outpatient unit of the University of Hamburg, student recruiting systems of the university, campus flyers and posters, as well as advertisements placed in local medical and counselling centers, online peer support forums, social media and part-time jobs platforms (Facebook). Participants were screened for eligibility and had to fulfil following criteria: age between 18 and 65 years, at least fluent German language skills, normal or corrected-to-normal vision, and the ability to provide written informed consent. Exclusion criteria for all subjects included a history of any neurological disorder, substance-related disorders (current or lifetime), schizophrenia spectrum disorder (current or lifetime), manic episode in the context of a bipolar disorder (current), use of benzodiazepines during the last week, and neuroleptic medication during the last three months. Further exclusion criteria for the control group included a current or lifetime obsessive-compulsive disorder, social anxiety disorder, or specific phobia. A detailed decomposition of each group's clinical diagnoses is listed in the supplement (Table S1). All participants received either course credit or monetary compensation for their study participation.

Prior to data analysis, two participants from the control group were excluded and replaced because they were mistakenly included in the study even though they fulfilled the criteria of a lifetime diagnosis OCD, SAD, or a specific phobia. One control participant's study participation was canceled because of an unwillingness to follow experimental instructions and was therefore excluded and replaced. Another control participant was excluded due to a low accuracy in the flanker task (i.e., < 50%), likely indicating that this person did not follow the task

instruction. And another three participants, each one of the clinical groups, were excluded due to low EEG quality (i.e., > 25% artifacts). At the time of participation, $n = 47$ participants were currently medicated with at least one drug including antidepressants ($n = 15$), oral contraceptives ($n = 9$), thyroid hormones ($n = 9$), dermatological drugs ($n = 2$), and other drugs of which none was reported more often than by one participant ($n = 12$).

2.2 Procedure

Prior to the laboratory assessment, participants were screened for eligibility using the inclusion and exclusion criteria specified earlier. They received verbal and written information of the objectives and methods of the study and gave written informed consent. Psychopathological symptoms, severity, and clinical diagnoses were assessed by respective questionnaires and structured interviews by trained personnel. After preparing the EEG, participants performed a set of tasks, including the flanker task, which was only preceded by an eight-minute resting state. The study procedure was approved by the local ethics committee and was planned as well as conducted in accordance with the Declaration of Helsinki (World Medical Association, 2013).

2.3 Measures

Clinical diagnoses were assessed using the Structured Clinical Interview for DSM-5 Disorders – Clinical Version (SCID-5-CV; Beesdo-Baum et al., 2019; First et al., 2016). Family risk for internalizing psychopathology was derived via the Family History Screen (Weissman et al., 2000). Obsessive-compulsive symptom severity of OCD patients was determined using the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989; Hand & Büttner-Westphal, 1991). Following these interviews, overall severity of illness was rated based on the Clinical Global Impression Scale (CGI-S; Guy, 1976) and global functioning was scored on the global assessment of functioning (GAF; American Psychiatric Association, 2000).

In addition, a battery of questionnaires were administered: Obsessive-compulsive symptoms were measured by the Obsessive-Compulsive Inventory Revised (OCI-R; 20 items, 5-point Likert scale 0-4; $\alpha = .91$; Foa et al., 2002; Gönner et al., 2007), social phobia symptoms by the self-report version of the Liebowitz Social Anxiety Scale (LSAS-SR; 24 items, 4-point Likert scale 0-3; $\alpha = .98$; Liebowitz, 1987; Stangier & Heidenreich, 2003), specific phobia symptoms by the Severity Measure for Specific Phobia (SMSP; 10 items, 5-point Likert scale 0-4; $\alpha = .85$; Craske et al., 2013), anxious apprehension by the Penn State Worry Questionnaire (PSWQ; 16 items, 5-point Likert scale 1-5; $\alpha = .94$; Glöckner-Rist & Rist, 2014; Meyer et al., 1990), anxious arousal by the respective subscale of the Mood and Anxiety Symptom Questionnaire (MASQ-AA; 17 items, 5-point Likert scale 1-5; $\alpha = .89$; Watson & Clark, 1991; Watson et al., 1995), state and trait anxiety by the respective subscales of the State-Trait-Anxiety Inventory (STAI-S and STAI-T; each 20 items, 4-point Likert scale 1-4; $\alpha = .95$ and $\alpha = .95$; Laux et al., 1981; Spielberger et al., 1970), depression symptoms by the Beck Depression Inventory (BDI-II; 21 items, 4-point Likert scale 0-3; $\alpha = .95$; Beck et al., 1996; Hautzinger et al., 2006), alcohol consumption by the Alcohol Use Disorders Identification Test (AUDIT; 10 items, 5-point Likert scale 0-4; $\alpha = .70$; Babor et al., 2001), and handedness by the modified Edinburgh Handedness Inventory (EHI; 10 items, 5-point Likert scale -10 to +10; $\alpha = .92$; Loffing et al., 2014; Oldfield, 1971). Additional questionnaires were administered for the purpose of investigating other research questions (https://osf.io/zrm2j) and results will be reported elsewhere.

2.4 Flanker Task

Participants performed a speeded arrowhead version of the flanker task (Eriksen & Eriksen, 1974) while they were sitting in a dimly lit, electrically shielded cabin approx. 24 inches in front of a 19-inch LCD monitor with a resolution 1920 × 1080 pixels and a refresh rate of 120

Hz. The task was identical with the one we previously used in another study (Härpfer et al., 2022) and consisted of a set of five horizontally aligned arrows (one target, four flankers) displayed by Presentation Software (Neurobehavioral Systems, Inc., Albany, California). The set of arrows was approx. 6.2° in width and approx. 1.0° in height with an equal number of trials pointing pseudo randomly either into same (<<<<< or >>>>>) or opposite directions (<<><< or >><>>). Participants were instructed to indicate the direction of the center arrow by pressing a key with their respective left or right index finger as quickly and accurately as possible, which was practiced in 20 trials at the beginning of the task. Each trial started with a fixation (200–1200 ms), followed by the presentation of arrow stimuli (100 ms), and a response window (max. 800 ms). The task consisted of five blocks with each 80 trials equaling 400 trials in total and participants received a performance feedback after each block asking them to respond faster, irrespective of their actual response times. The speed performance feedback was implemented to promote a sufficient amount of error trials, to reduce the length of the task to a reasonable extent, and to increase discrimination between groups since group differences of the ERN between OCD patients and healthy control participants were found to be pronounced under speed conditions (Riesel, Kathmann, et al., 2019).

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

2.5 Electrophysiological Recording and Processing

Continuous EEG data were recorded by 61 equidistant and concentric passive Ag/AgCl-electrodes (Easycap, Herrsching, Germany) and two 32-channel BrainAmp amplifiers (Brain Products GmbH, Gilching, Germany). The recording setup encompassed a band-pass filter of 0.01 to 250 Hz, a sampling rate of 1000Hz, a recording reference located between AF3 and Fz, a ground electrode located between AF4 and Fz, and an external electrode placed at the left infraorbital site. During preparation of the electrodes as well as throughout the study, impedances were reduced below a threshold of 5 kΩ.

After the recording of the EEG data, they were processed using Brain Vision Analyzer (Brain Products GmbH, Gilching, Germany). In a first step, we administered a band-pass filter with a low cut-off of 0.1 Hz and a high cut-off of 30 Hz (24 dB/oct roll-off) as well as a notch filter of 50 Hz. In a second step, ocular artifacts were corrected by a semi-automatic independent component analysis (ICA; Jung et al., 2000). This procedure involved the automatic identification of statistically independent components of the EEG signal, the visual inspection of each component's scalp topography and component activation, based on which we qualified those components representing ocular artifacts, and the final inspection of the corrected EEG signal. Subsequent to the ocular correction, continuous data of each electrode were re-referenced to the common average reference of all scalp electrodes, which was followed by the segmentation of response-locked epochs (-500 ms to 800 ms) and the artifact rejection. Segments containing an absolute voltage range exceeding 200 μV, voltage step exceeding 50 μV between consecutive data points, or maximum voltage difference of less than 0.5 μV within 100 ms intervals were removed before averaging each participant's correct and incorrect response segments separately. On average, data loss due to the artifact rejection was small (M = 0.87 %,

SD = 1.82 %, Max. = 11.90 %) in the finally analyzed sample. However, for *n* = 3 participants data loss was more than 25% and they were therefore excluded from data analysis ensuring high data quality as is recommended (Luck, 2014). All participants fulfilled the criterion of at least six erroneous trials to determine a reliable ERN (Olvet & Hajcak, 2009b).

In accordance with recent guidelines for the quantification of the ERN and CRN in anxiety research (Klawohn et al., 2020), we corrected segments for the baseline interval of -500 ms to -300 ms and scored both ERPs as mean amplitudes between 0 and 100 ms post response at electrode FCz, where the signal was maximal after inspecting the grand-averaged waveforms and topographical distributions. Spearman-Brown corrected correlations of odd- and even-numbered trials indicate good psychometric properties for both the ERN (*r* = .94) and CRN (*r* = .98). However, in order to evaluate the impact of different EEG preprocessing decisions on the study results (Klawohn et al., 2020; Sandre et al., 2020; Zhang et al., 2023), we also refer to the multiverse analyses of other commonly used baseline corrections and scoring strategies in the supplementary materials in detail and report the consistency across different alternative analysis paths including the averaged effect size, as well as standard devation and *p*-range of these effect sizes.

2.6 Data Analysis

Data analysis was conducted using IBM SPSS Statistics, version 29.0 (SPSS, Inc., Chicago) with a significance level of $\alpha = .05$. Demographic and clinical data including gender, age, education, as well as the aforementioned questionnaires were used to describe our sample. Differences in gender between groups was tested using a χ^2-test. Differences in demographic (age, education), clinical (questionnaires), and behavioral data (accuracy, PES) between the four diagnostic groups were tested by one-way analyses of variance (ANOVAs) with group (OCD,

SAD, PHOB, CON) as between-subject factor. Hypotheses on ERPs (ERN and CRN) and behavioral data (response times for correct and incorrect response) were analyzed using separate 2 × 4 mixed-measures ANOVAs with response type (correct, incorrect) as within-subject factor and group (OCD, SAD, PHOB, CON) as between-subject factor. Similar analyses were conducted for the influence of clinical status of an internalizing disorder and family risk for internalizing psychopathology, whereby the between-subject factor group was replaced by clinical status (nonclinical, clinical) and family risk (no risk, risk). Whenever preceding behavioral analyses revealed significant differences, the ERP analyses were controlled for the respective variables. Therefore, we slightly deviated from our preregistration by conducting analyses-of-covariance (ANCOVAs). Follow-up analyses were conducted when results revealed significant interactions. All *p*-values were Sidak-corrected for post-hoc comparisons with more than two factor levels.

In order to investigate the potential role of clinical status and the transdiagnostic anxiety dimensions, the sample of the current study was combined with another, subclinical sample (Härpfer et al., 2022) that used identical task and outcome specifications. Using multiple linear regression models (separate for ERN and CRN), models included the predictors gender, age, clinical status, PSWQ, and MASQ-AA as well as the interaction terms clinical status × PSWQ, clinical status × MASQ-AA, PSWQ × MASQ-AA, and clinical status × PSWQ × MASQ-AA. We controlled for the potential effects of gender (Fischer et al., 2016; Härpfer et al., 2020; Larson et al., 2011) and age (Falkenstein et al., 2001; Mathewson et al., 2005; Themanson et al., 2006) since previous showed interindividual differences of these demographic variables. Lastly we were interested in the moderating role of gender on the ERN-PSWQ link. Therefore, we performed multiple linear regression models (separate for ERN and CRN) with the predictors

gender, age, clinical status, PSWQ, gender × clinical, and gender × PSWQ. Continuous variables were mean-centered before they were entered in the regression models in order to reduce multicollinearity due to the interaction terms.

3. Results

3.1 Demographic, Questionnaire, and Clinical Data

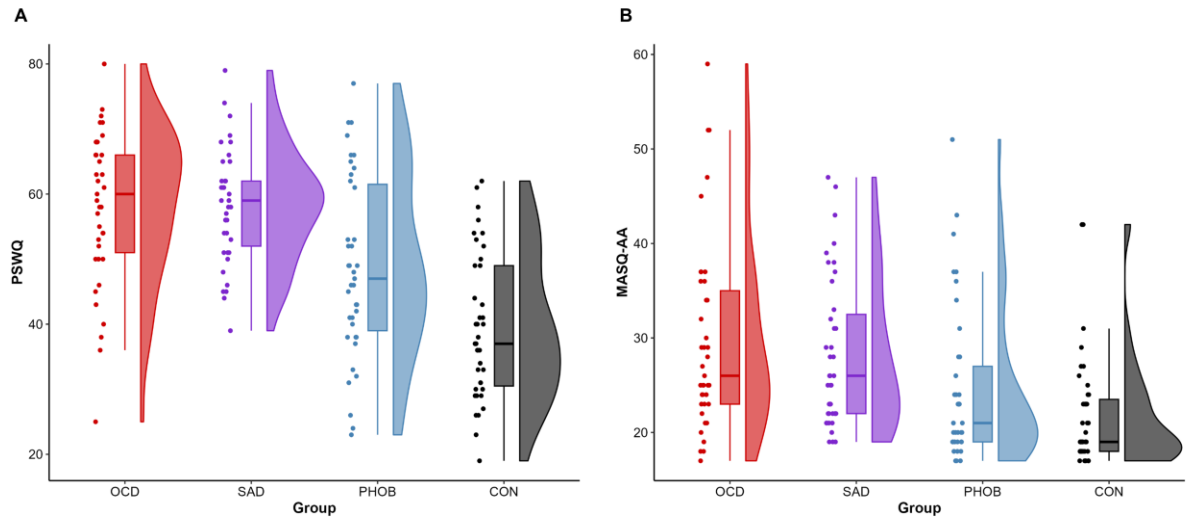
3.1.1 Diagnostic Groups

The four groups showed no significant differences regarding gender, age, or years of education. However, as expected, there were significant variations in questionnaire and clinical data. Specific information and statistics for each group are detailed in Table 1. In alignment with the recruitment strategy, obsessive-compulsive symptoms were most prominent in the OCD group, while social anxiety predominated the SAD group. Phobic symptoms also peaked in the PHOB group, but were not significantly different from the OCD and SAD group. Regarding the transdiagnostic anxiety dimensions, anxious apprehension and anxious arousal were highest in the OCD and SAD group, on intermediate levels in the PHOB group, and lowest in the CON group (Figure 1). State and trait anxiety, as well as depressive symptoms, were most pronounced in the OCD and SAD groups, with the CON group displaying the smallest manifestations, and the PHOB group having intermediate symptom severity. Levels of alcohol consumption did not differ significantly between the groups. Overall, the severity of illness in the clinical global impression rating was most severe for the OCD and SAD groups, followed by the PHOB group, and was inconspicuous for the CON group. A similar pattern emerged for global functioning, which was notably impaired in the OCD and SAD groups, moderately reduced in the PHOB group, and exhibited its highest level of functioning in the CON group. In summary, the four groups revealed varying profiles concerning the symptom severity and the anxiety dimensions.

Regarding the severity of illness and functional impairment, the OCD and SAD groups were more affected than the PHOB and CON groups.

Figure 1

Distribution of Anxious Apprehension (A) and Anxious Arousal (B) across Diagnostic Groups.



Note. OCD = Obsessive-Compulsive Disorder; SAD = Social Anxiety Disorder; PHOB = Specific Phobia; CON = Control Group; PSWQ = Penn State Worry Questionnaire; MASQ-AA = Mood and Anxiety Symptom Questionnaire (Anxious Arousal Subscale). $N = 156$.

Table 1

Demographical, Questionnaire, and Clinical Data across Diagnostic Groups.

	OCD (n = 39)		SAD (n = 39)		PHOB (n = 39)		CON (n = 39)		Group Comparison			
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	χ^2 / *F*	*df*	η^2	*p*
Demographical												
Gender (f/m)	28/11		29/10		30/9		28/11		0.36	3		.948
Age	28.36	6.38	28.38	7.64	27.90	9.80	29.41	8.31	0.24	3, 152	0.01	.868
Education	12.23	1.01	12.23	1.11	12.18	1.02	12.18	0.94	0.03	3, 152	0.00	.992
Questionnaires												
OCI-R	28.31_{a}	11.38	11.82_{b}	8.79	9.21_{bc}	10.11	5.59_{c}	4.77	47.49	3, 152	0.48	**<.001**
LSAS-SR	47.31_{a}	30.33	69.74_{b}	29.01	31.24_{c}	24.15	19.46_{c}	14.94	28.71	3, 151	0.36	**<.001**
SMSP	8.26_{a}	8.38	6.56_{a}	7.38	8.79_{a}	7.86	1.34_{b}	2.34	9.14	3, 150	0.16	**<.001**
PSWQ	58.13_{a}	11.75	58.10_{a}	8.73	48.56_{b}	13.90	39.41_{c}	11.13	23.65	3, 152	0.32	**<.001**
MASQ-AA	29.69_{a}	10.12	28.15_{ab}	7.91	24.62_{bc}	8.20	21.85_{c}	5.98	7.26	3, 152	0.13	**<.001**
STAI-T	52.67_{a}	11.42	53.23_{a}	10.17	40.08_{b}	12.52	34.85_{b}	7.49	29.53	3, 152	0.37	**<.001**
STAI-S	45.28_{a}	13.52	46.03_{a}	11.03	35.05_{b}	9.23	32.42_{b}	5.81	17.55	3, 150	0.26	**<.001**
BDI-II	17.41_{a}	11.70	17.08_{a}	12.44	8.00_{b}	9.62	3.74_{b}	4.61	17.77	3, 152	0.26	**<.001**
AUDIT	4.23	3.74	3.64	2.85	4.51	2.86	3.87	3.11	0.58	3, 152	0.01	.630
EHI	78.72	31.55	75.64	35.47	65.77	46.16	76.92	29.53	1.00	3, 152	0.02	.395
Clinical												
Diagnoses	2.90_{ab}	1.79	3.00_{a}	1.93	2.00_{b}	1.52	0.13_{c}	0.34	29.47	3, 152	0.37	**<.001**
CGI-S	3.59_{a}	0.85	3.58_{a}	1.03	2.79_{b}	1.01	1.00_{c}	0.61	73.18	3, 151	0.59	**<.001**
GAF	56.23_{a}	9.20	58.61_{a}	11.47	75.28_{b}	11.53	88.49_{c}	6.33	91.78	3, 151	0.65	**<.001**
YBOCS	22.03	5.34	-	-	-	-	-	-	-	-	-	-

Note. OCD = Obsessive-Compulsive Disorder; SAD = Social Anxiety Disorder; PHOB = Specific Phobia; CON = Control Group; Gender (f = female, m = male); age and education in years; OCI-R = Obsessive-Compulsive Inventory; LSAS-SR = Liebowitz Social Anxiety Scale – self report; SMSP = Severity Measure for Specific Phobia; PSWQ = Penn State Worry Questionnaire; MASQ-AA = Mood and Anxiety Symptom Questionnaire (Anxious Arousal Subscale); STAI = State-Trait-Anxiety Inventory (Trait and State Subscale); BDI-II = Beck Depression Inventory II; AUDIT = Alcohol Use Disorders Identification Test; EHI = Edinburgh Handedness Inventory (Modified); Diagnoses include index and comorbid diagnoses (n); CGI-S = Clinical Global Impression – Severity of Illness; GAF = Global Assessment of Functioning; YBOCS = Yale-Brown Obsessive Compulsive Scale; degrees of freedom are deviating for some questionnaires due to missing data; means with different subscripts within rows indicate significant differences according to Sidak corrected post-hoc t-tests with p <.05. N = 156.

p < .05 are printed in bold.

3.1.2 Clinical Status and Family Risk

Since we also grouped participants in terms of clinical status of an internalizing disorder and family risk for internalizing psychopathology, we report group-specific means, standard deviations, and statistical results of the group comparison in the supplement (Table S2). Across groups, symptoms displayed a spectrum of severity, with clinical participants with family risk showing the most severe symptoms, succeeded by clinical participants without family risk, non-clinical participants with family risk, and non-clinical participants without family risk.

3.2 Behavioral Data

3.2.1 Diagnostic Groups

The categorical analyses of the diagnostic groups did not reveal any significant differences regarding accuracy, $F(3, 152) = 0.16$, $p = .923$, $\eta_p^2 = 0.00$, or post-error slowing, $F(3, 152) = 0.69$, $p = .558$, $\eta_p^2 = 0.01$. Overall response times were faster on error compared with correct trials across groups, $F(1, 152) = 522.81$, $p < .001$, $\eta_p^2 = 0.78$, but did not differ between groups, $F(3, 152) = 1.70$, $p = .169$, $\eta_p^2 = 0.03$. However, we found an interaction effect of response × group on response times, $F(3, 152) = 5.40$, $p = .001$, $\eta_p^2 = 0.10$. Post-hoc comparisons showed that the PHOB group responded faster on error trials than the CON group ($p = .031$), with no other difference passing the significance threshold (all ps > .48). Group-specific means and standard deviations of the behavioral data can be found in Table 2. In brief, behavioral performance did not differ significantly between the four diagnostic groups, with the exception of error response times, whereby the PHOB group demonstrated faster responses than the CON group.

Table 2

Electrophysiological and Behavioral Data of the Diagnostic Groups.

	OCD (n = 39)		SAD (n = 39)		PHOB (n = 39)		CON (n = 39)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
ERPs								
ERN (μV)	-3.71	3.42	-3.47	3.48	-4.64	4.22	-3.08	2.85
CRN (μV)	-0.01	2.68	0.78	2.44	0.19	2.22	0.49	2.45
Behavior								
Accuracy (%)	86.07	7.80	85.53	8.54	85.50	7.53	84.74	10.07
RT correct (ms)	431.41	47.67	415.30	46.16	420.62	35.84	430.11	48.84
RT incorrect (ms)	359.70	59.74	359.05	64.67	343.27	39.30	380.45	64.52
PES (ms)	43.70	30.12	43.02	24.71	40.30	22.52	35.88	28.54

Note. OCD = Obsessive-Compulsive Disorder; SAD = Social Anxiety Disorder; PHOB = Specific Phobia; CON = Control Group; ERPs = event-related potentials; ERN = error-related negativity; CRN = correct-response negativity; RT = response time; PES = post-error slowing. N = 156.

3.2.2 Clinical Status and Family Risk

Since we also investigated the influence of clinical status of an internalizing disorder and family risk for internalizing psychopathology on ERPs, we shortly report results of participants' behavioral performance here. Regarding accuracy, we did not observe a significant effect of clinical status, $F(1, 152) = 0.42$, $p = .516$, $\eta_p^2 = 0.00$, family risk, $F(1, 152) = 2.05$, $p = .155$, $\eta_p^2 = 0.01$, or clinical status × family risk, $F(1, 152) = 0.01$, $p = .915$, $\eta_p^2 = 0.00$. However, post-error slowing differed as a result of family risk, $F(1, 152) = 6.94$, $p = .009$, $\eta_p^2 = 0.04$, with prolonged post-error slowing for the risk group, while there was no significant effect of clinical status, $F(1, 152) = 0.27$, $p = .607$, $\eta_p^2 = 0.00$, or clinical status × family risk, $F(1, 152) = 2.28$, $p = .133$, $\eta_p^2 = 0.02$. Overall response times were faster on error compared with correct trials, $F(1, 152) = 269.51$, $p < .001$, $\eta_p^2 = 0.64$. In addition, there was a significant interaction effect of response × clinical status on response times, $F(1, 152) = 5.36$, $p = .022$, $\eta_p^2 = 0.03$, which was qualified by faster response times for error trials ($p = .087$) but not for correct trials ($p = .682$) for the clinical compared with the nonclinical group, albeit closely missing the significance threshold for error

trials in the post-hoc comparison. No other significant effects on response times were found (all *p*s > .11). Group-specific means and standard deviations of the behavioral data can be found in the supplement (Table S3). Taken together, family risk was associated with prolonged post-error slowing and, on a trend level, clinical status with faster error response times.

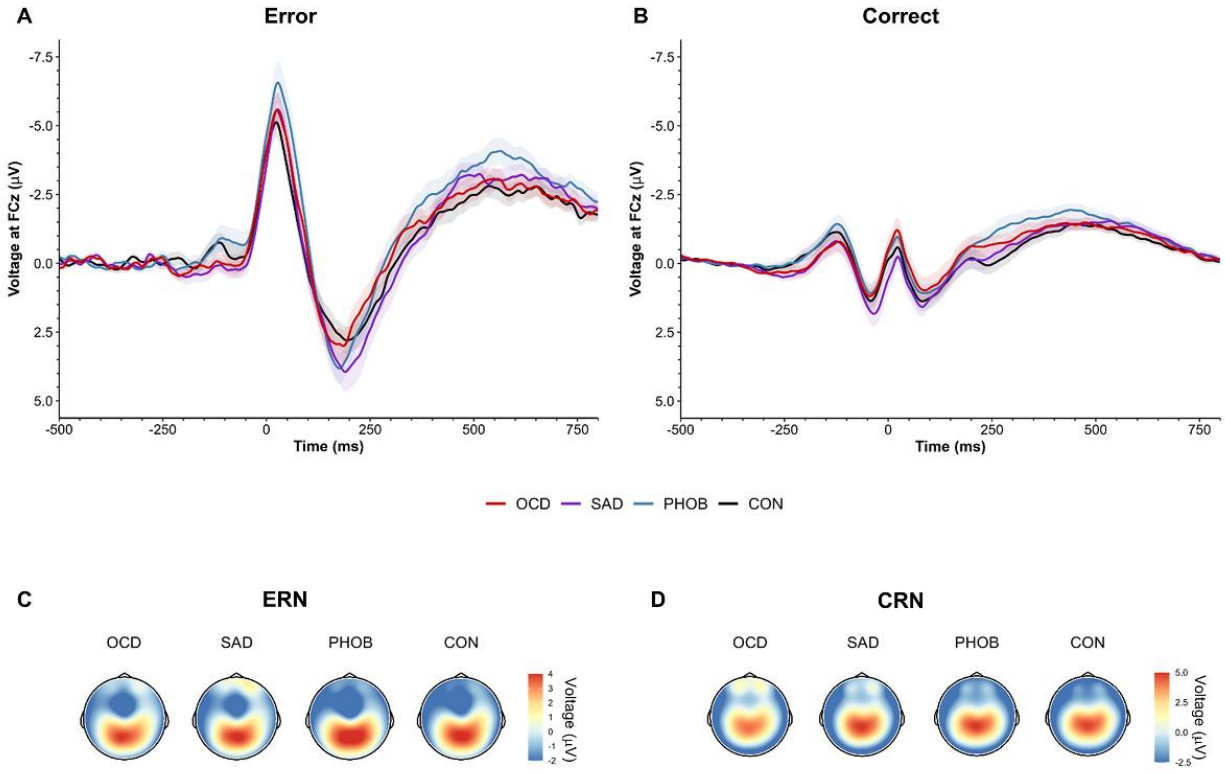
3.3 Event-Related Potentials

3.3.1 Diagnostic Groups

In the categorical analysis of the four diagnostic groups, we slightly deviated from our preregistered model (ANOVA) by controlling for error response times (ANCOVA), given that the PHOB group showed faster error response times compared with the CON group. The ANCOVA revealed a main effect of response type indicating larger ERN compared with CRN amplitudes, $F(1, 151) = 19.89$, $p < .001$, $\eta_p^2 = 0.12$. However, we did not find a significant group effect, $F(3, 151) = 1.56$, $p = .201$, $\eta_p^2 = 0.03$, nor an interaction of response × group, $F(3, 151) = 0.95$, $p = .418$, $\eta_p^2 = 0.02$, meaning we could not find evidence for differences in ERN or CRN amplitudes between the diagnostic groups. Table 2 lists group-specific means and standard deviations of the ERN and CRN. Figure 2 shows grand-averaged potentials and topographies of each group. This result pattern was robust against different quantification approaches of the ERN and CRN (Table S4 in the supplement). Across scoring strategies, the average effect size was $M_{\eta_p^2} = 0.114$, $SD_{\eta_p^2} = 0.026$ (*p*-range = <.001 - .001) for the response effect, $M_{\eta_p^2} = 0.020$, $SD_{\eta_p^2} = 0.007$ (*p*-range = .244 - .696) for the group effect, and $M_{\eta_p^2} = 0.021$, $SD_{\eta_p^2} = 0.004$ (*p*-range = .242 - .479) for the response × group interaction.

Figure 2

Grand-Averaged Waveforms and Corresponding Topographies for Error and Correct Trials of the Diagnostic Groups.



Note. Response-locked grand-averaged waveforms of erroneous and correct trials of each group (A, B). OCD = Obsessive-Compulsive Disorder; SAD = Social Anxiety Disorder; PHOB = Specific Phobia; CON = Control Group. Corresponding topographic head maps of the ERN (C) and CRN (D). ERN = error-related negativity; CRN = correct-response negativity. $N = 156$.

In order to examine the influence of our naturalistic control group on ERP differences, we repeated the categorical analyses by excluding five participants with a current or lifetime internalizing diagnosis from our control group. In this ANCOVA involving a strictly healthy control group, the group factor narrowly missed the significance threshold, $F(3, 146) = 2.49$, $p = .062$, $\eta_p^2 = 0.05$. Consequently, post-hoc comparisons also fell short of significance, although the comparison related to ERN differences between PHOB group ($M = -4.64$, $SD = 4.22$) and HC group ($M = -2.58$, $SD = 2.68$) approached the threshold ($p = .070$). Additionally, there was a

main effect of response type, $F(1, 146) = 18.05$, $p < .001$, $\eta_p^2 = 0.11$, but no interaction effect of response × group, $F(3, 146) = 1.03$, $p = .379$, $\eta_p^2 = 0.02$. Grand-averaged potentials of the three clinical groups compared with the healthy control group can be found in Figure S2 of the supplementary materials. Across scoring strategies, the average effect size was $M_{\eta_p^2} = 0.109$, $SD_{\eta_p^2} = 0.025$ (p-range = <.001 - .002) for the response effect, $M_{\eta_p^2} = 0.046$, $SD_{\eta_p^2} = 0.010$ (p-range = .033 - .265) for the group effect, and $M_{\eta_p^2} = 0.017$, $SD_{\eta_p^2} = 0.002$ (p-range = .379 - .520) for the response × group interaction. For more statistical details, please see Table S5 in the supplementary materials.

In essence, our investigation did not reveal evidence for variations in ERN or CRN amplitudes among the clinical groups, nor in comparison to the naturalistic control group. However, upon refining the control group by excluding participants with a current or lifetime internalizing disorder, creating a more strictly defined healthy control group, trend-level significant differences emerged between the PHOB and HC group.

3.3.2 Clinical Status and Family Risk

In order to investigate the role of clinical status of an internalizing disorder and family risk for internalizing psychopathology, we conducted a non-preregistered categorical analysis (ANCOVA) after reclassifying participants (i.e., clinical/nonclinical participants with/without family risk), while controlling for post-error slowing and error response times (see behavioral data for details). Detailed results of the ANCOVA are listed in Table 3 and illustrated in Figure 3. We found a main effect of response with larger ERN compared with CRN, but clinical status closely missed the significance threshold ($p = .071$), although there were descriptive differences in both ERPs, with larger ERN (nonclinical: $M = -2.58$, $SD = 2.68$ vs. clinical: $M = -4.04$, $SD = 3.69$) as well as CRN amplitudes (nonclinical: $M = 0.91$, $SD = 2.27$ vs. clinical: $M = 0.21$, $SD =$

2.48). Instead, family risk was associated with higher ERN (no risk: *M* = -2.79, *SD* = 3.00 versus risk: *M* = -4.21, *SD* = 3.71) and CRN amplitudes (no risk: *M* = 0.94, *SD* = 2.02 versus risk: *M* = 0.06, *SD* = 2.60). Notably, the result pattern for the family risk effect was heterogeneous across scoring strategies, while the clinical effect was significant in all other scoring strategies (Table S6 in the supplement): The average effect size was $M_{\eta_p^2}$ = 0.035, $SD_{\eta_p^2}$ = 0.012 (*p*-range = .004 - .071) for the clinical effect, $M_{\eta_p^2}$ = 0.014, $SD_{\eta_p^2}$ = 0.011 (*p*-range = .039 - .861) for the family risk effect, and $M_{\eta_p^2}$ = 0.000, $SD_{\eta_p^2}$ = 0.000 (*p*-range = .652 - .902) for the clinical × family risk interaction. In summary, both clinical status of an internalizing disorder and family risk for internalizing psychopathology seem to be associated with an enhanced ERN as well as CRN, although statistical significance of these effects were partially depending on the chosen scoring strategy.

Table 3

The Effects of Clinical Status of an Internalizing Disorder and Family Risk for Internalizing Psychopathology on the ERN and CRN.

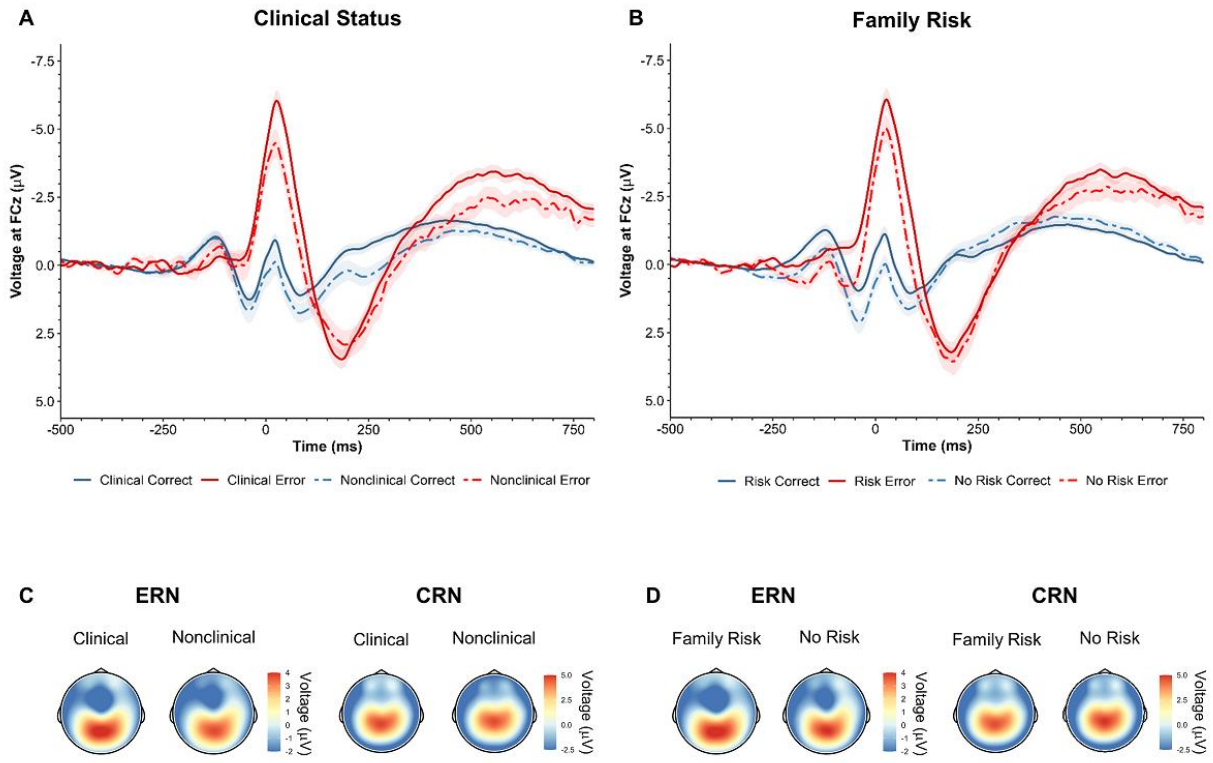
	F	*df*	η_p^2	*p*
Response	19.10	1, 150	0.11	**<.001**
Response × Clinical	0.69	1, 150	0.01	.406
Response × FHS Internalizing	0.54	1, 150	0.00	.466
Response × Clinical × FHS Internalizing	0.03	1, 150	0.00	.860
Clinical	3.32	1, 150	0.02	.071
Clinical × FHS Internalizing	0.12	1, 150	0.00	.731
FHS Internalizing	4.35	1, 150	0.03	**.039**

Note. ERN = error-related negativity; CRN = correct-response negativity; FHS = Family History Screen (family risk, no family risk); clinical (nonclinical, clinical); response (correct, incorrect). Analyses were controlled for error response time and post-error slowing, as these behavioral variables differed between the respective groups. *N* = 156.

p < .05 are printed in bold

Figure 3

Grand-Averaged Waveforms and Corresponding Topographies of Error and Correct Trials for Participants with or without a Clinical Status of an Internalizing Disorder or Family Risk for Internalizing Psychopathology.



Note. Response-locked grand-averaged waveforms of correct and erroneous trials for participants with or without a clinical status of an internalizing disorder (A) as well as for participants with or without a family risk for internalizing psychopathology (B). Corresponding topographic head maps of the ERN and CRN (C, D). ERN = error-related negativity; CRN = correct-response negativity. $N = 156$.

3.3.3 Clinical Status and Anxiety Dimensions

In accordance with our preregistration, we also combined data of the present clinical study with data from a previously published subclinical study (Härpfer et al., 2022) to investigate the impact of clinical status as well as the role of transdiagnostic anxiety dimensions across a wider spectrum of symptom severity. This yielded a joint sample consisting of $n = 246$ individuals affected by at least one lifetime internalizing disorder ($n = 136$) and individuals without any lifetime history of psychopathology ($n = 110$). Unfortunately, data concerning family risk for internalizing psychopathology were not collected in the previous study and could

therefore not be analyzed in this larger, combined sample. Since clinical status, PSWQ, and MASQ-AA were our predictors of main interest, we will focus on these in the following description of results. However, detailed results of the linear regression models of the ERN and CRN including all variables are listed in Table 4. Results of different scoring strategies are summarized in the supplement (Table S7).

In the preregistered ERN model, clinical status closely missed the significance threshold (p = .069), but was significant for all other scoring strategies. The average effect size of clinical status was M_β = -0.18, SD_β = 0.04 (p-range = .003 - .069) across scoring strategies. No other predictor of main interest (i.e., PSWQ or MASQ-AA) was significant. In the preregistered CRN model, we found a significant interaction of clinical status × PSWQ (p = .032; Figure 4) which was qualified by larger CRN amplitudes with increasing anxious apprehension in nonclinical (r = -.27, p = .005) but not clinical participants (r = -.03, p = .722). Across scoring strategies, the average effect size of clinical × PSWQ was M_β = 0.31, SD_β = 0.04 (p-range = .004 - .040). The average Pearson correlation between PSWQ and the CRN across scoring strategies was M_r = -.26, SD_r = 0.04 (p-range = <.001 - .054) in nonclinical participants and M_r = -.01, SD_r = 0.05 (p-range = .372 - .958) in clinical participants. Consequently, depending on the scoring approach, larger ERN amplitudes were linked to clinical status mirroring the effects observed in the categorical analysis. For the CRN, we found a differential effect regarding the PSWQ-CRN link between nonclinical and clinical participants, such that only nonclinical participants showed larger CRN amplitudes with increasing anxious apprehension.

Table 4

The Role of Clinical Status, PSWQ, and MASQ-AA on the ERN and CRN within the Combined Sample across the Severity Continuum.

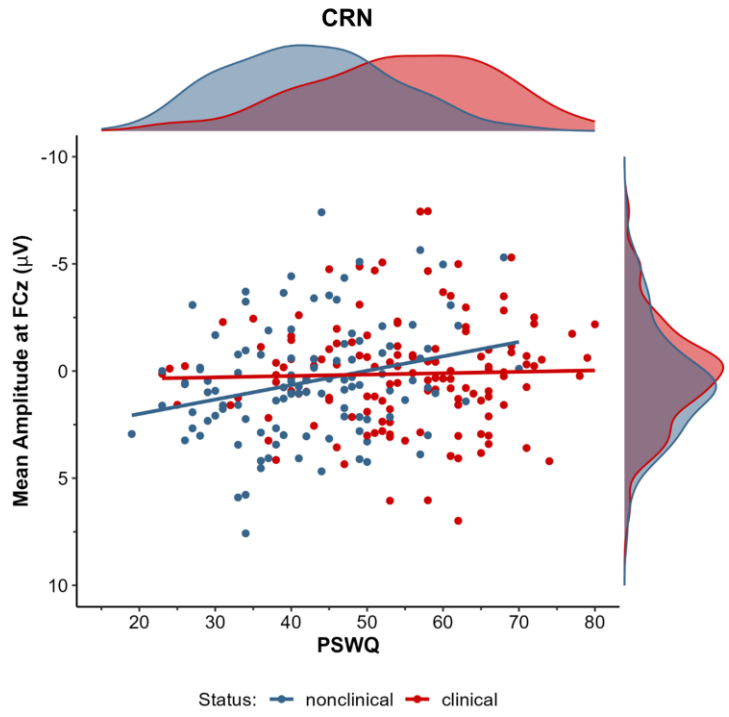
	ERN									CRN								
	b	SE_{boot}	β	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*	*b*	SE_{boot}	β	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*
Model						0.02	1.62	9, 236	.110						0.08	3.35	9, 236	**<.001**
Gender	0.78	0.61	0.09	1.33	.198					1.08	0.34	0.18	2.85	**.002**				
Age	0.07	0.03	0.13	1.97	**.017**					0.02	0.02	0.06	0.87	.320				
Clinical	-1.07	0.59	-0.14	-1.70	.069					0.05	0.41	0.01	0.13	.907				
PSWQ	-0.04	0.05	-0.13	-0.89	.366					-0.06	0.03	-0.28	-1.90	.060				
MASQ-AA	0.03	0.06	0.07	0.48	.569					-0.01	0.04	-0.04	-0.31	.766				
Clinical × PSWQ	0.04	0.06	0.10	0.71	.476					0.08	0.04	0.27	2.12	**.032**				
Clinical × MASQ-AA	-0.09	0.08	-0.17	-1.16	.244					-0.02	0.05	-0.06	-0.42	.668				
PSWQ × MASQ-AA	-0.01	0.01	-0.20	-1.36	.294					0.00	0.00	-0.15	-1.04	.351				
Clinical × PSWQ × MASQ-AA	0.01	0.01	0.24	1.54	.194					0.00	0.00	0.01	0.05	.969				

Note. ERN = error-related negativity; CRN = correct-response negativity; Gender (0 = female, 1 = male); age in years; clinical (0 = nonclinical, 1 = clinical); PSWQ = Penn State Worry Questionnaire; MASQ-AA = Mood and Anxiety Symptom Questionnaire; continuous variables were mean-centered. $N = 246$.

$p < .05$ are printed in bold

Figure 4

Clinical Status Moderating the Link between Anxious Apprehension and CRN.



Note. CRN = correct-response negativity. PSWQ = Penn State Worry Questionnaire. $N = 246$.

As preregistered, we also investigated the role of the transdiagnostic anxiety dimensions within the initial sample ($n = 156$). Detailed results of these models (with smaller statistical power) can be found in the supplement (Table S8). For the ERN, we found partial support of an association with PSWQ, depending on the scoring strategy, with an average effect size of $M_\beta = -0.17$, $SD_\beta = 0.03$ (p-range = .044 - .248). No association was found for the MASQ-AA (all ps > .26). For the CRN, we could not find any significant prediction by PSWQ or MASQ-AA (all ps > .16).

3.3.4 Anxious Apprehension and Gender

Prior research has indicated that gender may act as a moderating factor in the relationship between anxiety and enhanced error monitoring, with women displaying a stronger association between anxious apprehension and increased ERN amplitudes (Moser et al., 2016). In order to investigate the moderating role of gender, we conducted explorative multiple linear regression models using the combined sample (n = 246) including n = 181 women and n = 65 men. The results demonstrated that the interaction term gender × PSWQ was significant for both the ERN and CRN indicating a differential effect, whereby only women exhibited a larger ERN (females: r = -.15, p = .049 vs. males: r = .07, p = .584) and CRN (females: r = -.17, p = .023 vs. males: r = .14, p = .261) with increasing anxious apprehension (Table 5, Figure 5). Detailed results across various scoring strategies are available in the supplementary materials (Table S9). Across scoring strategies, the average effect size of gender × PSWQ was M_β = 0.61, SD_β = 0.10 (p-range = .002 - .035) for the ERN and M_β = 0.60, SD_β = 0.04 (p-range = .005 - .016) for the CRN. The average Pearson correlation between PSWQ and the ERN in women was M_r = -.16, SD_r = 0.02 (p-range = .016 - .071) and M_r = .13, SD_r = 0.04 (p-range = .139 - .584) in men. For the PSWQ-CRN link, the average correlation was M_r = -.15, SD_r = 0.02 (p-range = .015 - .104) for women and M_r = .13, SD_r = 0.04 (p-range = .160 - .530) for men. Taken together, women but not men showed increasing ERN and CRN amplitudes with increasing anxious apprehension.

Table 5

The Role of Gender and PSWQ on the ERN and CRN within the Combined Sample across the Severity Continuum.

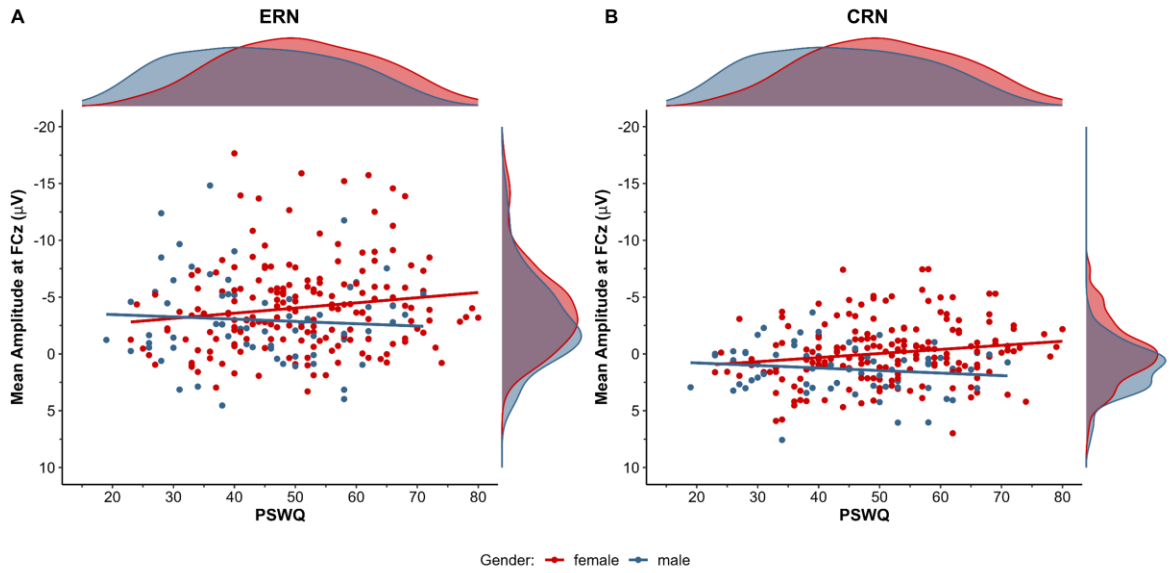
	ERN									CRN								
	b	SE_{boot}	β	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*	*b*	SE_{boot}	β	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*
Model						0.03	2.41	6, 239	**.028**						0.06	3.79	6, 239	**.001**
Gender	1.30	0.93	0.15	1.34	.155					1.76	0.63	0.30	2.78	**.002**				
Age	0.08	0.03	0.15	2.30	**.008**					0.02	0.02	0.06	0.99	.289				
Clinical	-0.67	0.62	-0.09	-1.05	.284					0.00	0.42	0.00	-0.01	.994				
PSWQ	-0.04	0.02	-0.12	-1.46	.135					-0.04	0.02	-0.18	-2.22	**.010**				
Gender × Clinical	-0.40	1.18	-0.03	-0.30	.715					-0.57	0.87	-0.07	-0.66	.461				
Gender × PSWQ	0.09	0.05	0.16	1.74	**.048**					0.07	0.03	0.20	2.19	**.003**				

Note. ERN = error-related negativity; CRN = correct-response negativity; gender (0 = female, 1 = male); age in years; clinical (0 = nonclinical, 1 = clinical); PSWQ = Penn State Worry Questionnaire. $N = 246$.

$p < .05$ are printed in bold

Figure 5

Gender Moderating the Link between Anxious Apprehension and (A) ERN as well as (B) CRN.



Note. ERN = error-related negativity; CRN = correct-response negativity. PSWQ = Penn State Worry Questionnaire. *N* = 246.

4. Discussion

By elucidating the neural underpinnings of anxiety and obsessive-compulsive disorders in past research, the prevailing understanding emerged that enhanced error-related brain activity represents a potential neural risk marker. However, previous investigations have not uniformly linked all disorder categories within this spectrum to ERN variations, which was attributed to different underlying symptom dimensions and varying degrees of symptom severity. The current study aimed to fill this research gap by simultaneously investigating multiple disorders and associated dimensions within the anxiety and obsessive-compulsive spectrum. Specifically, we focused on the role of disorder category (i.e., OCD, SAD, specific phobia, and a control group), clinical status of a lifetime internalizing disorder (i.e. nonclinical and clinical), family risk for internalizing psychopathology (i.e., no risk and risk), and transdiagnostic anxiety dimensions

(i.e., anxious apprehension and anxious arousal). In the categorical analysis, we did not observe significant differences in ERPs among the clinical groups, nor in comparison to the naturalistic control group. However, after creating a more strictly defined healthy control group, we found larger ERN amplitudes in the specific phobia compared with the healthy control group. In addition, when comparing clinical participants with a lifetime history of an internalizing disorder to those without any past or present diagnoses, both ERN and CRN were larger in the clinical group. Moreover, our data also suggest an association of increased ERN and CRN amplitudes with family risk for internalizing psychopathology. Lastly, the dimensional analyses did not reveal an overall association of anxious apprehension and performance monitoring. Instead, we could identify gender as a moderating factor of this relationship, indicating that higher anxious apprehension was associated with larger ERN and CRN amplitudes in women but not in men.

In a broader context, our findings show partial alignment with the existing body of literature. Previous research has not yet suggested a link between specific phobia and aberrant error monitoring (Hajcak et al., 2003; Moser et al., 2005), contrary to what our results unexpectedly reveal. Furthermore, we could not replicate ERN differences between the control group and OCD or SAD, respectively, as indicated by prior studies (e.g., Endrass et al., 2014; Riesel, 2019), even when compared with a strictly defined healthy control group. However it is crucial to consider the possibility that these findings may, in part, be attributed to insufficient statistical power, as the disorder-specific analysis was only able to detect medium or large sized effects. By reclassifying all clinical participants within one group and combining data with a previous subclinical study, we were able to conduct better powered analyses revealing larger ERN and CRN amplitudes in the clinical group. Consistent with prior investigations, our results undermine the importance of clinical status (Saunders & Inzlicht, 2020), particularly the role of

lifetime internalizing disorders (Härpfer et al., 2022), for the link between anxiety and enhanced performance monitoring. Individuals with internalizing psychopathology seem to share a predisposition for a cognitive system that is more wired to monitor performance compared with those without any clinically relevant symptomatology. Thus, enhanced performance monitoring might contribute to explain the trajectories to clinical anxiety and obsessive-compulsive symptoms (Weinberg, Dieterich, et al., 2015; Weinberg et al., 2022) and an elevated ERN may serve as a broad risk marker (or even endophenotype, as posited by other studies) for the anxiety and obsessive-compulsive spectrum, rather than being indicative for specific disorders within that spectrum. This observation aligns with findings from a prospective study predicting the initial onset of anxiety disorders in children exhibiting larger error-related brain activity, but failing to differentiate among specific types of anxiety disorders (Meyer, Hajcak, et al., 2015). However, when employing the ERN as a prognostic tool among already clinically anxious children and adolescents over a two-year period, enhanced error-related brain activity was related to increased generalized anxiety, social anxiety, and harm avoidance, but not with physical anxiety symptoms or panic (Meyer et al., 2021). This suggests that the predictive value of the ERN may change over time, with its contribution varying in terms of the trajectories leading to the initial development of a disorder and the trajectories over the course of the disorder.

In addition to clinical status, family risk for internalizing psychopathology also help us understand the role of performance monitoring in the development of anxiety and obsessive-compulsive symptoms. A limitation inherent to cross-sectional studies, including our own, lies in the inability to establish causal relationships between enhanced performance monitoring and the development of symptoms or vice versa. Nonetheless, our research contributes substantively to the notion that enhanced performance monitoring, as a vulnerability marker, might be

transmitted across generations, indicating a predisposition toward internalizing disorders (Carrasco, Harbin, et al., 2013; Riesel et al., 2011; Riesel, Klawohn, et al., 2019). This conclusion was drawn from our finding that both clinical status and family risk independently contributed to an increased ERN and CRN. We also conclude that clinical status and family risk do not yield an additive effect on the magnitude of performance monitoring; instead, either factor independently proves sufficient to be associated with heightened performance monitoring. In this context, it is crucial to differentiate between the trajectories leading to an enhanced trait-like ERN and its role in the etiopathogenesis of clinical anxiety and obsessive-compulsive symptoms. On the one hand, an increased ERN is potentially shaped by a complex interplay of multiple early acting factors during infancy and adolesence, such as genetics (Anokhin et al., 2008; Suor et al., 2022), gender (Fischer et al., 2016; Hill et al., 2018; Imburgio et al., 2020; Larson et al., 2011), cognitive control (Meyer & Hajcak, 2019), temperament (Brooker & Buss, 2014; Lahat et al., 2014; McDermott et al., 2009; Meyer, Hajcak, et al., 2018), and parenting style (Banica et al., 2019; Brooker & Buss, 2014; Meyer et al., 2019; Meyer, Proudfit, et al., 2015; Suor et al., 2022). On the other hand, an increased ERN represents a risk marker for anxiety and obsessive-compulsive disorders, regardless of the specific etiological factors behind its enhancement. Consequently, while some individuals may have inherited an enhanced ERN, posing a risk for clinical anxiety and obsessive-compulsive symptoms, this aspect may only account for the phenomenon in a subset of cases. In essence, an individual exposed to a punitive parenting style that results in an enhanced ERN may harbor a comparable risk for clinical symptoms as individuals who have inherited an elevated ERN. Yet, the complex interplay of these influencing factors contributing to an elevated trait-like ERN remains widely unexplored. Consequently,

future investigations should prioritize a comprehensive examination of their combined effects to gain deeper insights into the genesis of enhanced error-related brain activity.

Another potential explanation for previously observed differential associations within the anxiety and obsessive-compulsive spectrum highlights the pivotal role of the transdiagnostic dimension anxious apprehension. Previous research found the ERN to predict the onset of a generalized anxiety disorder (Meyer, Nelson, et al., 2018) as well as generalized and social anxiety symptoms (Lahat et al., 2014; Meyer et al., 2021), but found no cross-sectional or prospective association with other dimensions including panic or physical symptoms (Hajcak et al., 2003; Meyer et al., 2021; Moser et al., 2005). This differential links indicate a certain specificity of the ERN to the anxious apprehension dimension and is also in line with cross-sectional findings from meta-analyses (Moser et al., 2013; Saunders & Inzlicht, 2020). As a result, an enhanced ERN does not seem to generally indicate risk for all disorder categories of the anxiety and obsessive-compulsive spectrum; instead, enhanced error monitoring likely contributes only to the development of disorders characterized by anxious apprehension. However, an important remark to consider is that the relationship between elevated error monitoring and anxious apprehension is, to a significant extent, relying on the retrospective classification of samples (such as worry versus mixed anxiety) in meta-analyses. Consequently, the direct dimensional association between ERN amplitudes and anxious apprehension vs. anxious arousal has received comparatively limited attention, with only some exceptions (e.g., Härpfer et al., 2022; Härpfer et al., 2020; Lin et al., 2015; Moser et al., 2012). To the best of our knowledge, our study is among the first employing a transdiagnostic dimensional approach within a widely transdiagnostic dataset including different clinical disorder categories.

Aligning with findings of previous research, our results support the notion that the link between heightened anxious apprehension and enhanced error monitoring is gender-specific, such that it predominantly manifests in women (Moser et al., 2016). Extending previous research findings, we found evidence that the gender-specific link of anxious apprehension extends beyond error monitoring to encompass performance monitoring in general (i.e., ERN and CRN). Overall, these findings might imply differential trajectories of anxiety in women and men, wherein enhanced performance monitoring appears to only play a contributory role in the development of worry among women, not men. Within the existing literature, two explanatory approaches have been advanced to elucidate the gender-specific link between anxious apprehension and increased error monitoring (Moser et al., 2016). One perspective posits that the amplification of verbal worries exerts a more pronounced impact on cognitive performance in women. Extensive research demonstrates that anxiety is cross-sectionally and prospectively linked to impairments in cognitive functioning (Gulpers et al., 2022; Lindert et al., 2021; Tetzner & Schuth, 2016) with some studies indicating greater impairment in women (de Visser et al., 2010; Gulpers et al., 2019; Zainal & Newman, 2023). This may be attributable to the inclination of women, relative to men, to employ verbal processes during problem-solving, thereby fostering subvocal articulation as a response tendency to threatening or uncertain situations (Moser et al., 2016). Alternatively, another perspective underscores the organizational and regulatory impact of ovarian hormones on frontal brain regions associated with cognitive control in women (Beltz & Moser, 2020; Russman Block et al., 2024; Shansky & Lipps, 2013). Moser et al. (2016) argue that the elevated concentration of estradiol in regions linked to error monitoring, coupled with estradiol's modulatory influence on dopamine—a primary neurotransmitter thought to contribute to the generation of the ERN—may offer a promising neurobiological rationale for the observed

connection between anxious apprehension and the ERN in women. A recent study suggests that estradiol and progesterone may represent protective factors, wherein women exhibiting relatively elevated levels of these hormones show a weaker association of worry and error-related brain activity in the ACC (Russman Block et al., 2024).While our study design cannot offer support for either explanation regarding the gender specificity of this connection, we assert that it is imperative to consider gender effects when investigating the etiological pathways of the neural foundations in clinical anxiety. Additionally, a more thorough understanding of the mechanisms contributing to development of anxiety and obsessive-compulsive symptoms in women and men is essential.

However, even though our study found that anxious apprehension was dimensionally linked to enhanced error monitoring (in women), this did not directly translate into ERN differences among the investigated clinical groups. This challenges the assumption that variations in latent symptom profiles, specifically anxious apprehension, underlying anxiety and obsessive-compulsive disorders can singularly account for driving this link. It is plausible that another transdiagnostic and more general phenotype, such as threat sensitivity (Proudfit et al., 2013) trait defense reactivity (Weinberg et al., 2012), or anxious misery (Riesel et al., 2023), that is on the one hand partially associated with anxious apprehension but potentially more evenly distributed across internalizing disorder categories, might drive variations in ERN amplitudes, thus, explaining that we did not find ERN differences between disorder categories. While an augmented ERN is commonly conceptualized as the sensitivity to internal threat (i.e., concerns about the consequences of one's performance and potentially the social evaluation by others) rather than external threats (i.e., fear of specific objects or situations) and thereby linked to disorders, such as OCD, SAD, and GAD, it might also be adaptive for individuals with fear-

related disorders, such as specific phobia or agoraphobia, to monitor their behavior carefully. Nevertheless, examining how dimensional constructs translate into differences between disorder categories requires future well-powered studies involving multiple disorders categories with larger samples sizes for each group.

In evaluating our results, it is essential to consider certain limitations. Firstly, despite our aim for a transdiagnostic and dimensional approach to unravel the link between performance monitoring and the anxiety and obsessive-compulsive spectrum, we did not include the full range of potentially relevant disorders. Of particular interest would be the additional inclusion of generalized anxiety disorder, panic disorder, and agoraphobia to cover the complete transdiagnostic anxiety and obsessive-compulsive spectrum. In fact, this subsequent research project is currently ongoing in our lab. Furthermore, many previous studies also used healthy control participants for their case-control design. In contrast, we followed a more conservative and naturalistic approach whereby control participants did not need to be fully free of any mental disorder (except the index diagnoses). Nonetheless, a rather small amount of only five participants of the control group were diagnosed with a lifetime disorder. Another limitation is that we utilized a speeded version of the flanker task; initially in order to promote potential group differences between clinical and control participants. In a previous study (Riesel, Kathmann, et al., 2019), OCD patients showed impaired flexibility under speed conditions, whereas healthy control participants seem capable to up- and downregulate their error monitoring depending on the speed vs. accuracy demand of a task. However, this pattern was not yet investigated for SAD or specific phobia and it remains unclear whether the inflexibility of OCD patients also applies to these disorders. As already demonstrated in the early years of ERN research, speed instructions generally cause smaller ERN amplitudes (Falkenstein et al., 2000; Gehring et al., 1993). This

might have led to floor effects limiting the range of variance available to analyze. Lastly, we wanted to account for the variance of potential forking paths in ERP quantification by demonstrating the sensitivity of the results to commonly used scoring strategies. Most of our findings were robust against different scoring approaches, however, some of the analyses (e.g., family risk) displayed substantial variation in the final outcome. Over the past years of anxiety research, the potential influence of methodological decisions in EEG preprocessing on study results has received increasing attention (Klawohn et al., 2020; Sandre et al., 2020; Zhang et al., 2023). However, final guidelines that stand on a clear and unequivocal foundation of empirical evidence are yet to formulate (Paul et al., 2022).

Finally, we want to emphasize the importance of further investigating the mechanisms involved in translating neural indices of vulnerability into clinical anxiety and its diverse facets. In this context, previous research (e.g., Meyer, 2017; Weinberg et al., 2022) has frequently invoked the diathesis-stress model, describing psychopathology (i.e., anxiety and obsessive-compulsive symptoms) as a result of the interplay between predispositional vulnerability (i.e., enhanced ERN) and stress (i.e., adverse life events). Although heightened error monitoring unequivocally indicates risk for clinical anxiety, obsessions, and compulsions (e.g., Riesel, Klawohn, et al., 2019; Saunders & Inzlicht, 2020), only a subset of individuals with an enhanced ERN develops a disorder. Building on the diathesis-stress model, instances of adverse life events, such as interpersonal stress (Banica et al., 2020), a natural disaster (Meyer et al., 2017), or a pandemic (Riesel et al., 2021) appear to moderate the association between enhanced error monitoring and the development of anxiety and obsessive-compulsive symptoms. As previous research suggests, heightened error monitoring may specifically contribute to the development of certain anxiety and obsessive-compulsive disorders, particularly those characterized by anxious

apprehension (Moser et al., 2013; Saunders & Inzlicht, 2020). Moreover, there is evidence that not only stressful life events (i.e., environmental factors) serve as moderators but also other features of a person (i.e., individual factors), such as a behavioral inhibition (Lahat et al., 2014; McDermott et al., 2009) or sex (Moser et al., 2016), in conjunction with enhanced error monitoring, play a role in the emergence of clinical anxiety. In light of these observations, we believe that the research field would notably benefit from the formulation of a more detailed and integrative etiological model delineating the trajectories to psychopathology with testable predictions. In our opinion, this model should consider (a) the general formation of an increased or decreased trait-like ERN by various early-acting factors (e.g., genetics, cognitive control, or parenting style), (b) the interplay of an enhanced or diminished trait-like ERN with environmental (e.g., adverse life events) and individual factors (e.g., sex or behavioral inhibition) resulting in either internalizing or externalizing disorders, and (c) the characterization of these disorders by specific facets (e.g., impulsivity or anxious apprehension).

In conclusion, results of the present study challenge the assumption of a disorder-specific association with enhanced error monitoring. Instead, our data indicate that enhanced performance monitoring is more broadly associated with internalizing psychopathology. Moreover, in contrast to the limited existing literature, our results suggest that enhanced error monitoring may also play a role in specific phobia. Our data further endorse the suggested role of elevated performance monitoring as a neural risk maker reflecting vulnerability for internalizing disorders, as reflected by the association of increased ERN and CRN amplitudes with family risk for internalizing psychopathology. Additionally, our findings indicate heightened performance monitoring is linked to anxious apprehension, particularly in women, while no such association was observed in men or with anxious arousal. Notably, this association is not contingent upon

clinical status, implying that the strength of this link remains consistent across both clinical and nonclinical individuals. In essence, our study supports the notion that enhanced neural indices of performance monitoring serve as a broad risk marker for internalizing psychopathology, particularly within the anxiety- and obsessive-compulsive spectrum, without specifying particular disorder categories within that spectrum. Dimensional analyses also suggest a link to the transdiagnostic anxiety dimension of anxious apprehension in women, spanning across diagnostic categories. Future research endeavors should aim at unravelling the trajectories leading to internalizing psychopathology by examining the complex interplay between the predisposition for enhanced performance monitoring and various biological, psychological, and environmental factors.

References

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR)*.

Anokhin, A. P., Golosheykin, S., & Heath, A. C. (2008). Heritability of frontal brain function related to action monitoring. *Psychophysiology, 45*(4), 524-534. https://doi.org/10.1111/j.1469-8986.2008.00664.x

Babor, T. F., Higgins-Biddle, J. C., Saunders, J. B., & Monteiro, M. G. (2001). *AUDIT: The Alcohol Use Disorders Identification Test: Guidelines for use in primary health care* (2nd ed.). World Health Organization. https://apps.who.int/iris/handle/10665/67205

Banica, I., Sandre, A., Shields, G. S., Slavich, G. M., & Weinberg, A. (2020). The error-related negativity (ERN) moderates the association between interpersonal stress and anxiety symptoms six months later. *International Journal of Psychophysiology, 153*, 27-36. https://doi.org/10.1016/j.ijpsycho.2020.03.006

Banica, I., Sandre, A., & Weinberg, A. (2019). Overprotective/authoritarian maternal parenting is associated with an enhanced error-related negativity (ERN) in emerging adult females. *International Journal of Psychophysiology, 137*, 12-20. https://doi.org/10.1016/j.ijpsycho.2018.12.013

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory-II*. Psychological Corporation.

Beesdo-Baum, K., Zaudig, M., & Wittchen, H. (2019). *Strukturiertes Klinisches Interview für DSM-5-Störungen – Klinische Version* [The Structured Clinical Interview for DSM-5 Disorders – Clinical Version]. Hogrefe.

Bellato, A., Norman, L., Idrees, I., Ogawa, C. Y., Waitt, A., Zuccolo, P. F., Tye, C., Radua, J., Groom, M. J., & Shephard, E. (2021). A systematic review and meta-analysis of altered electrophysiological markers of performance monitoring in Obsessive-Compulsive Disorder (OCD), Gilles de la Tourette Syndrome (GTS), Attention-Deficit/Hyperactivity disorder (ADHD) and Autism. *Neuroscience & Biobehavioral Reviews, 131*, 964-987. https://doi.org/10.1016/j.neubiorev.2021.10.018

Beltz, A. M., & Moser, J. S. (2020). Ovarian hormones: a long overlooked but critical contributor to cognitive brain structures and function. *Annals of the New York Academy of Sciences, 1464*(1), 156-180. https://doi.org/10.1111/nyas.14255

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review, 108*(3), 624-652. https://doi.org/10.1037/0033-295X.108.3.624

Brázdil, M., Roman, R., Daniel, P., & Rektor, I. (2005). Intracerebral error-related negativity in a simple Go/NoGo task. *Journal of Psychophysiology, 19*(4), 244-255. https://doi.org/10.1027/0269-8803.19.4.244

Brooker, R. J., & Buss, K. A. (2014). Harsh parenting and fearfulness in toddlerhood interact to predict amplitudes of preschool error-related negativity. *Developmental Cognitive Neuroscience, 9*, 148-159. https://doi.org/10.1016/j.dcn.2014.03.001

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4*(6), 215-222. https://doi.org/10.1016/S1364-6613(00)01483-2

Bushnell, M. C., Čeko, M., & Low, L. A. (2013). Cognitive and emotional control of pain and its disruption in chronic pain. *Nature Reviews Neuroscience, 14*(7), 502-511. https://doi.org/10.1038/nrn3516

Carrasco, M., Harbin, S. M., Nienhuis, J. K., Fitzgerald, K. D., Gehring, W. J., & Hanna, G. L. (2013). Increased error-related brain activity in youth with obsessive-compulsive disorder and unaffected siblings. *Depression and Anxiety, 30*(1), 39-46. https://doi.org/10.1002/da.22035

Carrasco, M., Hong, C., Nienhuis, J. K., Harbin, S. M., Fitzgerald, K. D., Gehring, W. J., & Hanna, G. L. (2013). Increased error-related brain activity in youth with obsessive-compulsive disorder and other anxiety disorders. *Neuroscience Letters, 541*, 214-218. https://doi.org/10.1016/j.neulet.2013.02.017

Cavanagh, J. F., & Shackman, A. J. (2015). Frontal midline theta reflects anxiety and cognitive control: Meta-analytic evidence. *Journal of Physiology-Paris, 109*(1), 3-15. https://doi.org/10.1016/j.jphysparis.2014.04.003

Clayson, P. E., McDonald, J. B., Park, B., Holbrook, A., Baldwin, S. A., Riesel, A., & Larson, M. J. (2023). Registered replication report of the construct validity of the error-related negativity (ERN): A multi-site study of task-specific ERN correlations with internalizing and externalizing symptoms. *Psychophysiology*, e14496. https://doi.org/10.1111/psyp.14496

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*(1), 155-159. https://doi.org/10.1037/0033-2909.112.1.155

Cox, B. J., Clara, I. P., Hills, A. L., & Sareen, J. (2010). Obsessive-compulsive disorder and the underlying structure of anxiety disorders in a nationally representative sample: Confirmatory factor analytic findings from the German health survey. *Journal of Anxiety Disorders, 24*(1), 30-33. https://doi.org/10.1016/j.janxdis.2009.08.003

Craske, M., Wittchen, H. U., Bogels, S., Stein, M., Adrews, G., & Lebeu, R. (2013). *Severity Measure for Specific Phobia—Adult*. American Psychiatric Association.

Cuthbert, B. N. (2014). Translating intermediate phenotypes to psychopathology: The NIMH Research Domain Criteria. *Psychophysiology, 51*(12), 1205-1206. https://doi.org/10.1111/psyp.12342

Davies, P. L., Segalowitz, S. J., & Gavin, W. J. (2004a). Development of error-monitoring event-related potentials in adolescents. *Annals of the New York Academy of Sciences, 1021*(1), 324-328. https://doi.org/10.1196/annals.1308.039

Davies, P. L., Segalowitz, S. J., & Gavin, W. J. (2004b). Development of response-monitoring ERPs in 7- to 25-year-olds. *Developmental Neuropsychology, 25*(3), 355-376. https://doi.org/10.1207/s15326942dn2503_6

de Visser, L., van der Knaap, L. J., van de Loo, A. J. A. E., van der Weerd, C. M. M., Ohl, F., & van den Bos, R. (2010). Trait anxiety affects decision-making differently in healthy men and women: Towards gender-specific endophenotypes of anxiety. *Neuropsychologia, 48*(6), 1598-1606. https://doi.org/10.1016/j.neuropsychologia.2010.01.027

Debener, S., Ullsperger, M., Siegel, M., Fiehler, K., von Cramon, D. Y., & Engel, A. K. (2005). Trial-by-trial coupling of concurrent electroencephalogram and functional magnetic resonance imaging identifies the dynamics of performance monitoring. *The Journal of Neuroscience, 25*(50), 11730-11737. https://doi.org/10.1523/JNEUROSCI.3286-05.2005

Dehaene, S., Posner, M. I., & Tucker, D. M. (1994). Localization of a neural system for error detection and compensation. *Psychological Science, 5*(5), 303-305. https://doi.org/10.1111/j.1467-9280.1994.tb00630.x

Dutilh, G., van Ravenzwaaij, D., Nieuwenhuis, S., van der Maas, H. L. J., Forstmann, B. U., & Wagenmakers, E.-J. (2012). How to measure post-error slowing: A confound and a simple solution. *Journal of Mathematical Psychology, 56*(3), 208-216. https://doi.org/10.1016/j.jmp.2012.04.001

Endrass, T., Klawohn, J., Gruetzmann, R., Ischebeck, M., & Kathmann, N. (2012). Response-related negativities following correct and incorrect responses: Evidence from a temporospatial principal component analysis. *Psychophysiology, 49*(6), 733-743. https://doi.org/10.1111/j.1469-8986.2012.01365.x

Endrass, T., Riesel, A., Kathmann, N., & Buhlmann, U. (2014). Performance monitoring in obsessive–compulsive disorder and social anxiety disorder. *Journal of Abnormal Psychology, 123*(4), 705-714. https://doi.org/10.1037/abn0000012

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception & Psychophysics, 16*(1), 143-149. https://doi.org/10.3758/BF03203267

Euser, A. S., Evans, B. E., Greaves-Lord, K., Huizink, A. C., & Franken, I. H. A. (2013). Diminished error-related brain activity as a promising endophenotype for substance-use disorders: evidence from high-risk offspring. *Addiction Biology, 18*(6), 970-984. https://doi.org/10.1111/adb.12002

Fairbairn, C. E., Kang, D., & Federmeier, K. D. (2021). Alcohol and neural dynamics: a meta-analysis of acute alcohol effects on event-related brain potentials. *Biological Psychiatry, 89*(10), 990-1000. https://doi.org/10.1016/j.biopsych.2020.11.024

Falkenstein, M., Hohnsbein, J., Hoormann, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components: II Error processing in choice reaction tasks. *Electroencephalography & Clinical Neurophysiology, 78*(6), 447-455. https://doi.org/10.1016/0013-4694(91)90062-9

Falkenstein, M., Hoormann, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: A tutorial. *Biological Psychology, 51*(2-3), 87-107. https://doi.org/10.1016/S0301-0511(99)00031-9

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (2001). Changes of error-related ERPs with age. *Experimental Brain Research, 138*(2), 258-262. https://doi.org/10.1007/s002210100712

Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods, 41*, 1149-1160. http://gpower.hhu.de

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*, 175-191. http://gpower.hhu.de

First, M., Williams, J., Karg, R., & Spitzer, R. (2016). *Structured Clinical Interview for DSM-5 Disorders, Clinician Version (SCID-5-CV)*. American Psychiatric Association.

Fischer, A. G., Danielmeier, C., Villringer, A., Klein, T. A., & Ullsperger, M. (2016). Gender influences on brain responses to errors and post-error adjustments. *Scientific Reports, 6*(1), 24435. https://doi.org/10.1038/srep24435

Foa, E. B., Huppert, J. D., Leiberg, S., Langner, R., Kichic, R., Hajcak, G., & Salkovskis, P. M. (2002). The Obsessive-Compulsive Inventory: Development and validation of a short version. *Psychological Assessment, 14*(4), 485-496. https://doi.org/10.1037/1040-3590.14.4.485

Geburek, A. J., Rist, F., Gediga, G., Stroux, D., & Pedersen, A. (2013). Electrophysiological indices of error monitoring in juvenile and adult attention deficit hyperactivity disorder (ADHD)—A meta-analytic appraisal. *International Journal of Psychophysiology, 87*(3), 349-362. https://doi.org/10.1016/j.ijpsycho.2012.08.006

Gehring, W. J., Goss, B., Coles, M. G., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science, 4*(6), 385-390. https://doi.org/10.1111/j.1467-9280.1993.tb00586.x

Glöckner-Rist, A., & Rist, F. (2014). Deutsche Version des Penn State Worry Questionnaire (PSWQ-d) [German version of the Penn State Worry Questionnaire]. *Zusammenstellung sozialwissenschaftlicher Items und Skalen*, 1-26. https://doi.org/10.6102/zis219

Godlove, D. C., Emeric, E. E., Segovis, C. M., Young, M. S., Schall, J. D., & Woodman, G. F. (2011). Event-related potentials elicited by errors during the stop-signal Task. I. Macaque Monkeys. *The Journal of Neuroscience, 31*(44), 15640. https://doi.org/10.1523/JNEUROSCI.3349-11.2011

Gönner, S., Leonhart, R., & Ecker, W. (2007). Das Zwangsinventar OCI-R - Die deutsche Version des Obsessive-Compulsive Inventory-Revised [The German Version of the Obsessive-Compulsive Inventory-Revised]. *Psychotherapie Psychosomatik Medizinische Psychologie, 57*(09/10), 395-404. https://doi.org/10.1055/s-2007-970894

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., Heninger, G. R., & Charney, D. S. (1989). The Yale-Brown Obsessive Compulsive Scale: I. Development, Use, and Reliability. *Archives of General Psychiatry, 46*(11), 1006-1011. https://doi.org/10.1001/archpsyc.1989.01810110048007

Gorka, S. M., MacNamara, A., Aase, D. M., Proescher, E., Greenstein, J. E., Walters, R., Passi, H., Babione, J. M., Levy, D. M., Kennedy, A. E., DiGangi, J. A., Rabinak, C. A., Schroth, C., Afshar, K., Fitzgerald, J., Hajcak, G., & Phan, K. L. (2016). Impact of alcohol use disorder comorbidity on defensive reactivity to errors in veterans with posttraumatic stress disorder. *Psychology of Addictive Behaviors, 30*(7), 733-742. https://doi.org/10.1037/adb0000196

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *The American Journal of Psychiatry, 160*(4), 636-645. https://doi.org/10.1176/appi.ajp.160.4.636

Grützmann, R., Endrass, T., Kaufmann, C., Allen, E., Eichele, T., & Kathmann, N. (2016). Presupplementary motor area contributes to altered error monitoring in obsessive-compulsive disorder. *Biological Psychiatry, 80*(7), 562-571. https://doi.org/10.1016/j.biopsych.2014.12.010

Gulpers, B. J. A., Oude Voshaar, R. C., van Boxtel, M. P. J., Verhey, F. R. J., & Köhler, S. (2019). Anxiety as a risk factor for cognitive decline: a 12-year follow-up cohort study. *The American Journal of Geriatric Psychiatry, 27*(1), 42-52. https://doi.org/10.1016/j.jagp.2018.09.006

Gulpers, B. J. A., Verhey, F. R. J., Eussen, S. J. P. M., Schram, M. T., de Galan, B. E., van Boxtel, M. P. J., Stehouwer, C. D. A., & Köhler, S. (2022). Anxiety and cognitive functioning in

the Maastricht study: A cross-sectional population study. *Journal of Affective Disorders, 319*, 570-579. https://doi.org/10.1016/j.jad.2022.09.072

Guy, W. (1976). *ECDEU assessment manual for psychopharmacology*. U.S. Department of Health, Education, and Welfare.

Hajcak, G., Franklin, M. E., Foa, E. B., & Simons, R. F. (2008). Increased error-related brain activity in pediatric obsessive-compulsive disorder before and after treatment. *American Journal of Psychiatry, 165*(1), 116-123. https://doi.org/10.1176/appi.ajp.2007.07010143

Hajcak, G., Klawohn, J., & Meyer, A. (2019). The utility of event-related potentials in clinical psychology. *Annual Review of Clinical Psychology, 15*(1), 71-95. https://doi.org/10.1146/annurev-clinpsy-050718-095457

Hajcak, G., McDonald, N., & Simons, R. F. (2003). Anxiety and error-related brain activity. *Biological Psychology, 64*(1-2), 77-90. https://doi.org/10.1016/S0301-0511(03)00103-0

Hand, I., & Büttner-Westphal, H. (1991). Die Yale-Brown Obsessive Compulsive Scale (Y-BOCS): Ein halbstrukturiertes Interview zur Beurteilung des Schweregrades von Denk- und Handlungszwängen [The Yale-Brown Obsessive Compulsive Scale (Y-BOCS): A semi-structured interview to assess the severity of obsessions and compulsions.]. *Verhaltenstherapie, 1*(3), 223-225. https://doi.org/10.1159/000257972

Härpfer, K., Carsten, H. P., Löwisch, K., Westermann, N., & Riesel, A. (2022). Disentangling the effects of trait and state worry on error-related brain activity: Results from a randomized controlled trial using worry manipulations. *Psychophysiology, 59*(9), e14055. https://doi.org/10.1111/psyp.14055

Härpfer, K., Carsten, H. P., Spychalski, D., Kathmann, N., & Riesel, A. (2020). Were we erring? The impact of worry and arousal on error-related negativity in a non-clinical sample. *Psychophysiology, 57*(11), e13661. https://doi.org/10.1111/psyp.13661

Hautzinger, M., Keller, F., & Kühner, C. (2006). *Beck Depressions-Inventar (BDI-II)*. Harcourt Test Services.

Hill, K. E., Ait Oumeziane, B., Novak, K. D., Rollock, D., & Foti, D. (2018). Variation in reward- and error-related neural measures attributable to age, gender, race, and ethnicity. *International Journal of Psychophysiology, 132*, 353-364. https://doi.org/10.1016/j.ijpsycho.2017.12.009

Hoffmann, S., & Falkenstein, M. (2010). Independent component analysis of erroneous and correct responses suggests online response control. *Human Brain Mapping, 31*(9), 1305-1315. https://doi.org/10.1002/hbm.20937

Holroyd, C. B., & Coles, M. G. H. (2002). The neural basis of human error processing: Reinforcement learning, dopamine, and the error-related negativity. *Psychological Review, 109*(4), 679-709. https://doi.org/10.1037/0033-295X.109.4.679

Imburgio, M. J., Banica, I., Hill, K. E., Weinberg, A., Foti, D., & MacNamara, A. (2020). Establishing norms for error-related brain activity during the arrow Flanker task among young adults. *NeuroImage, 213*, 116694. https://doi.org/https://doi.org/10.1016/j.neuroimage.2020.116694

Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K., Sanislow, C., & Wang, P. (2010). Research domain criteria (RDoC): Toward a new classification framework for research on mental disorders. *The American Journal of Psychiatry, 167*(7), 748-751. https://doi.org/10.1176/appi.ajp.2010.09091379

Jung, T.-P., Makeig, S., Humphries, C., Lee, T.-W., McKeown, M. J., Iragui, V., & Sejnowski, T. J. (2000). Removing electroencephalographic artifacts by blind source separation. *Psychophysiology, 37*(2), 163-178. https://doi.org/10.1111/1469-8986.3720163

Kaiser, A., Aggensteiner, P.-M., Baumeister, S., Holz, N. E., Banaschewski, T., & Brandeis, D. (2020). Earlier versus later cognitive event-related potentials (ERPs) in attention-deficit/hyperactivity disorder (ADHD): A meta-analysis. *Neuroscience & Biobehavioral Reviews, 112*, 117-134. https://doi.org/10.1016/j.neubiorev.2020.01.019

Klawohn, J., Meyer, A., Weinberg, A., & Hajcak, G. (2020). Methodological choices in event-related potential (ERP) research and their impact on internal consistency reliability and individual differences: An examination of the error-related negativity (ERN) and anxiety. *Journal of Abnormal Psychology, 129*(1), 29-37. https://doi.org/10.1037/abn0000458

Kozak, M. J., & Cuthbert, B. N. (2016). The NIMH Research Domain Criteria Initiative: Background, issues, and pragmatics. *Psychophysiology, 53*(3), 286-297. https://doi.org/10.1111/psyp.12518

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry, 56*(10), 921-926. https://doi.org/10.1001/archpsyc.56.10.921

Kujawa, A., Weinberg, A., Bunford, N., Fitzgerald, K. D., Hanna, G. L., Monk, C. S., Kennedy, A. E., Klumpp, H., Hajcak, G., & Phan, K. L. (2016). Error-related brain activity in youth and young adults before and after treatment for generalized or social anxiety disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 71*, 162-168. https://doi.org/10.1016/j.pnpbp.2016.07.010

Ladouceur, C. D., Tan, P. Z., Sharma, V., Bylsma, L. M., Silk, J. S., Siegle, G. J., Forbes, E. E., McMakin, D. L., Dahl, R. E., Kendall, P. C., Mannarino, A., & Ryan, N. D. (2018). Error-related brain activity in pediatric anxiety disorders remains elevated following individual therapy: A randomized clinical trial. *Journal of Child Psychology and Psychiatry, 59*(11), 1152-1161. https://doi.org/10.1111/jcpp.12900

Lahat, A., Lamm, C., Chronis-Tuscano, A., Pine, D. S., Henderson, H. A., & Fox, N. A. (2014). Early behavioral inhibition and increased error monitoring predict later social phobia symptoms in childhood. *Journal of the American Academy of Child & Adolescent Psychiatry, 53*(4), 447-455. https://doi.org/10.1016/j.jaac.2013.12.019

Larson, M. J., Baldwin, S. A., Good, D. A., & Fair, J. E. (2010). Temporal stability of the error-related negativity (ERN) and post-error positivity (Pe): The role of number of trials. *Psychophysiology, 47*(6), 1167-1171. https://doi.org/10.1111/j.1469-8986.2010.01022.x

Larson, M. J., & Clayson, P. E. (2011). The relationship between cognitive performance and electrophysiological indices of performance monitoring. *Cognitive, Affective, & Behavioral Neuroscience, 11*(2), 159-171. https://doi.org/10.3758/s13415-010-0018-6

Larson, M. J., South, M., & Clayson, P. E. (2011). Sex differences in error-related performance monitoring. *NeuroReport, 22*(1), 44-48. https://doi.org/10.1097/WNR.0b013e3283427403

Laux, L., Glanzmann, P., Schaffner, P., & Spielberger, C. D. (1981). *Das State-Trait-Angstinventar (STAI)* [The State-Trait Anxiety Inventory (STAI)]. Beltz.

Liebowitz, M. R. (1987). Social phobia. *Modern Problems of Pharmacopsychiatry, 22*, 141-173. https://doi.org/10.1159/000414022

Lin, Y., Moran, T. P., Schroder, H. S., & Moser, J. S. (2015). The role of hand of error and stimulus orientation in the relationship between worry and error-related brain activity: Implications for theory and practice. *Psychophysiology, 52*(10), 1281-1292. https://doi.org/10.1111/psyp.12470

Lindert, J., Paul, K. C., Lachman, M. E., Ritz, B., & Seeman, T. E. (2021). Depression-, anxiety-, and anger and cognitive functions: Findings from a longitudinal prospective study. *Frontiers in Psychiatry, 12*, 665742. https://doi.org/10.3389/fpsyt.2021.665742

Loffing, F., Sölter, F., & Hagemann, N. (2014). Left preference for sport tasks does not necessarily indicate left-handedness: Sport-specific lateral preferences, relationship with handedness and implications for laterality research in behavioural sciences. *PLOS ONE, 9*(8), e105800. https://doi.org/10.1371/journal.pone.0105800

Luck, S. J. (2014). *An introduction to the event-related potential technique* (2nd ed.). MIT Press.

Lumme, V., Aalto, S., Ilonen, T., Någren, K., & Hietala, J. (2007). Dopamine D2/D3 receptor binding in the anterior cingulate cortex and executive functioning. *Psychiatry Research: Neuroimaging, 156*(1), 69-74. https://doi.org/https://doi.org/10.1016/j.pscychresns.2006.12.012

Lutz, M. C., Kok, R., Verveer, I., Malbec, M., Koot, S., van Lier, P. A. C., & Franken, I. H. A. (2021). Diminished error-related negativity and error positivity in children and adults with externalizing problems and disorders: a meta-analysis on error processing. *Journal of Psychiatry and Neuroscience, 46*(6), E615. https://doi.org/10.1503/jpn.200031

Macedo, I., Pasion, R., Barbosa, F., & Ferreira-Santos, F. (2021). A dimensional approach to the neuronal correlates of anxiety, depression, and perfectionism: A transdiagnostic dissociation of error-related brain activity. *Behavioural Brain Research, 408*, 113271. https://doi.org/10.1016/j.bbr.2021.113271

Manoach, D. S., & Agam, Y. (2013). Neural markers of errors as endophenotypes in neuropsychiatric disorders. *Frontiers in Human Neuroscience, 7*, 350. https://doi.org/10.3389/fnhum.2013.00350

Martin, E. A., McCleery, A., Moore, M. M., Wynn, J. K., Green, M. F., & Horan, W. P. (2018). ERP indices of performance monitoring and feedback processing in psychosis: A meta-analysis. *International Journal of Psychophysiology, 132*, 365-378. https://doi.org/10.1016/j.ijpsycho.2018.08.004

Mathews, C. A., Perez, V. B., Delucchi, K. L., & Mathalon, D. H. (2012). Error-related negativity in individuals with obsessive–compulsive symptoms: Toward an understanding of hoarding behaviors. *Biological Psychology, 89*(2), 487-494. https://doi.org/10.1016/j.biopsycho.2011.12.018

Mathewson, K. J., Dywan, J., & Segalowitz, S. J. (2005). Brain bases of error-related ERPs as influenced by age and task. *Biological Psychology, 70*(2), 88-104. https://doi.org/10.1016/j.biopsycho.2004.12.005

McDermott, J. M., Perez-Edgar, K., Henderson, H. A., Chronis-Tuscano, A., Pine, D. S., & Fox, N. A. (2009). A history of childhood behavioral inhibition and enhanced response monitoring in adolescence are linked to clinical anxiety. *Biological Psychiatry, 65*(5), 445-448. https://doi.org/10.1016/j.biopsych.2008.10.043

McLoughlin, G., Albrecht, B., Banaschewski, T., Rothenberger, A., Brandeis, D., Asherson, P., & Kuntsi, J. (2009). Performance monitoring is altered in adult ADHD: A familial event-related potential investigation. *Neuropsychologia, 47*(14), 3134-3142. https://doi.org/https://doi.org/10.1016/j.neuropsychologia.2009.07.013

Mechias, M.-L., Etkin, A., & Kalisch, R. (2010). A meta-analysis of instructed fear studies: Implications for conscious appraisal of threat. *NeuroImage, 49*(2), 1760-1768. https://doi.org/10.1016/j.neuroimage.2009.09.040

Meyer, A. (2017). A biomarker of anxiety in children and adolescents: A review focusing on the error-related negativity (ERN) and anxiety across development. *Developmental Cognitive Neuroscience, 27*, 58-68. https://doi.org/10.1016/j.dcn.2017.08.001

Meyer, A., Carlton, C., Chong, L. J., & Wissemann, K. (2019). The presence of a controlling parent is related to an increase in the error-related negativity in 5–7 year-old children. *Journal of Abnormal Child Psychology, 47*(6), 935-945. https://doi.org/10.1007/s10802-018-0503-x

Meyer, A., Danielson, C. K., Danzig, A. P., Bhatia, V., Black, S. R., Bromet, E., Carlson, G., Hajcak, G., Kotov, R., & Klein, D. N. (2017). Neural biomarker and early temperament predict increased internalizing symptoms after a natural disaster. *Journal of the American Academy of Child & Adolescent Psychiatry, 56*(5), 410-416. https://doi.org/https://doi.org/10.1016/j.jaac.2017.02.005

Meyer, A., & Hajcak, G. (2019). A review examining the relationship between individual differences in the error-related negativity and cognitive control. *International Journal of Psychophysiology, 144*, 7-13. https://doi.org/10.1016/j.ijpsycho.2019.07.005

Meyer, A., Hajcak, G., Torpey-Newman, D., Kujawa, A., Olino, T. M., Dyson, M., & Klein, D. N. (2018). Early temperamental fearfulness and the developmental trajectory of error-related brain activity. *Developmental Psychobiology, 60*(2), 224-231. https://doi.org/10.1002/dev.21605

Meyer, A., Hajcak, G., Torpey-Newman, D. C., Kujawa, A., & Klein, D. N. (2015). Enhanced error-related brain activity in children predicts the onset of anxiety disorders between the ages of 6 and 9. *Journal of Abnormal Psychology, 124*(2), 266-274. https://doi.org/10.1037/abn0000044

Meyer, A., Mehra, L., & Hajcak, G. (2021). Error-related negativity predicts increases in anxiety in a sample of clinically anxious female children and adolescents over 2 years. *Journal of Psychiatry and Neuroscience, 46*(4), E472. https://doi.org/10.1503/jpn.200128

Meyer, A., Nelson, B., Perlman, G., Klein, D. N., & Kotov, R. (2018). A neural biomarker, the error-related negativity, predicts the first onset of generalized anxiety disorder in a large sample of adolescent females. *Journal of Child Psychology and Psychiatry, and Allied Disciplines, 59*(11), 1162-1170. https://doi.org/10.1111/jcpp.12922

Meyer, A., Proudfit, G. H., Bufferd, S. J., Kujawa, A. J., Laptook, R. S., Torpey, D. C., & Klein, D. N. (2015). Self-reported and observed punitive parenting prospectively predicts increased error-related brain activity in six-year-old children. *Journal of Abnormal Child Psychology, 43*(5), 821-829. https://doi.org/10.1007/s10802-014-9918-1

Meyer, A., Weinberg, A., Klein, D. N., & Hajcak, G. (2012). The development of the error-related negativity (ERN) and its relationship with anxiety: Evidence from 8 to 13 year-olds. *Developmental Cognitive Neuroscience, 2*(1), 152-161. https://doi.org/10.1016/j.dcn.2011.09.005

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy, 28*(6), 487-495. https://doi.org/10.1016/0005-7967(90)90135-6

Michael, J. A., Wang, M., Kaur, M., Fitzgerald, P. B., Fitzgibbon, B. M., & Hoy, K. E. (2021). EEG correlates of attentional control in anxiety disorders: A systematic review of error-related negativity and correct-response negativity findings. *Journal of Affective Disorders, 291*, 140-153. https://doi.org/10.1016/j.jad.2021.04.049

Miller, A. E., Watson, J. M., & Strayer, D. L. (2012). Individual differences in working memory capacity predict action monitoring and the error-related negativity. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 38*(3), 757-763. https://doi.org/10.1037/a0026595

Miltner, W. H. R., Lemke, U., Weiss, T., Holroyd, C., Scheffers, M. K., & Coles, M. G. H. (2003). Implementation of error-processing in the human anterior cingulate cortex: a source analysis of the magnetic equivalent of the error-related negativity. *Biological Psychology, 64*(1), 157-166. https://doi.org/10.1016/S0301-0511(03)00107-8

Moran, T. P., Schroder, H. S., Kneip, C., & Moser, J. S. (2017). Meta-analysis and psychophysiology: A tutorial using depression and action-monitoring event-related potentials. *International Journal of Psychophysiology, 111*, 17-32. https://doi.org/10.1016/j.ijpsycho.2016.07.001

Moran, T. P., Taylor, D., & Moser, J. S. (2012). Sex moderates the relationship between worry and performance monitoring brain activity in undergraduates. *International Journal of Psychophysiology, 85*(2), 188-194. https://doi.org/10.1016/j.ijpsycho.2012.05.005

Moser, J. S., Hajcak, G., & Simons, R. F. (2005). The effects of fear on performance monitoring and attentional allocation. *Psychophysiology, 42*(3), 261-268. https://doi.org/10.1111/j.1469-8986.2005.00290.x

Moser, J. S., Moran, T. P., & Jendrusina, A. A. (2012). Parsing relationships between dimensions of anxiety and action monitoring brain potentials in female undergraduates. *Psychophysiology, 49*(1), 3-10. https://doi.org/10.1111/j.1469-8986.2011.01279.x

Moser, J. S., Moran, T. P., Kneip, C., Schroder, H. S., & Larson, M. J. (2016). Sex moderates the association between symptoms of anxiety, but not obsessive compulsive disorder, and error-monitoring brain activity: A meta-analytic review. *Psychophysiology, 53*(1), 21-29. https://doi.org/10.1111/psyp.12509

Moser, J. S., Moran, T. P., Schroder, H. S., Donnellan, M. B., & Yeung, N. (2013). On the relationship between anxiety and error monitoring: A meta-analysis and conceptual framework. *Frontiers in Human Neuroscience, 7*, 466. https://doi.org/10.3389/fnhum.2013.00466

Nieuwenhuis, S., Ridderinkhof, K. R., Talsma, D., Coles, M. G. H., Holroyd, C. B., Kok, A., & van der Molen, M. W. (2002). A computational account of altered error processing in older age: Dopamine and the error-related negativity. *Cognitive, Affective, & Behavioral Neuroscience, 2*(1), 19-36. https://doi.org/10.3758/CABN.2.1.19

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia, 9*(1), 97-113. https://doi.org/10.1016/0028-3932(71)90067-4

Olver, J. S., O'Keefe, G., Jones, G. R., Burrows, G. D., Tochon-Danguy, H. J., Ackermann, U., Scott, A. M., & Norman, T. R. (2010). Dopamine D1 receptor binding in the anterior cingulate cortex of patients with obsessive–compulsive disorder. *Psychiatry Research: Neuroimaging, 183*(1), 85-88. https://doi.org/https://doi.org/10.1016/j.pscychresns.2010.04.004

Olvet, D. M., & Hajcak, G. (2008). The error-related negativity (ERN) and psychopathology: Toward an endophenotype. *Clinical Psychology Review, 28*(8), 1343-1354. https://doi.org/10.1016/j.cpr.2008.07.003

Olvet, D. M., & Hajcak, G. (2009a). Reliability of error-related brain activity. *Brain Research, 1284*, 89-99. https://doi.org/10.1016/j.brainres.2009.05.079

Olvet, D. M., & Hajcak, G. (2009b). The stability of error-related brain activity with increasing trials. *Psychophysiology, 46*(5), 957-961. https://doi.org/10.1111/j.1469-8986.2009.00848.x

Pasion, R., & Barbosa, F. (2019). ERN as a transdiagnostic marker of the internalizing-externalizing spectrum: A dissociable meta-analytic effect. *Neuroscience and Biobehavioral Reviews, 103*, 133-149. https://doi.org/10.1016/j.neubiorev.2019.06.013

Paul, K., Short, C. A., Beauducel, A., Carsten, H. P., Härpfer, K., Hennig, J., Hewig, J., Hildebrandt, A., Kührt, C., Mueller, E. M., Munk, A., Osinsky, R., Porth, E., Riesel, A., Rodrigues, J., Scheffel, C., Stahl, J., Strobel, A., & Wacker, J. (2022). The methodology and dataset of the CoScience EEG-Personality Project – A large-scale, multi-laboratory project grounded in cooperative forking paths analysis. *Personality Science, 3*, 1-26. https://doi.org/10.5964/ps.7177

Peyron, R., Laurent, B., & García-Larrea, L. (2000). Functional imaging of brain responses to pain. A review and meta-analysis (2000). *Neurophysiologie Clinique/Clinical Neurophysiology, 30*(5), 263-288. https://doi.org/10.1016/S0987-7053(00)00227-6

Proudfit, G. H., Inzlicht, M., & Mennin, D. S. (2013). Anxiety and error monitoring: The importance of motivation and emotion. *Frontiers in Human Neuroscience, 7*, 636. https://doi.org/10.3389/fnhum.2013.00636

Rabinak, C. A., Holman, A., Angstadt, M., Kennedy, A. E., Hajcak, G., & Phan, K. L. (2013). Neural response to errors in combat-exposed returning veterans with and without post-

traumatic stress disorder: A preliminary event-related potential study. *Psychiatry Research: Neuroimaging, 213*(1), 71-78. https://doi.org/10.1016/j.pscychresns.2013.01.002

Ridderinkhof, K. R., van den Wildenberg, W. P. M., Segalowitz, S. J., & Carter, C. S. (2004). Neurocognitive mechanisms of cognitive control: The role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. *Brain and Cognition, 56*(2), 129-140. https://doi.org/10.1016/j.bandc.2004.09.016

Riesel, A. (2019). The erring brain: Error-related negativity as an endophenotype for OCD—A review and meta-analysis. *Psychophysiology, 56*(4), e13348. https://doi.org/10.1111/psyp.13348

Riesel, A., Endrass, T., Auerbach, L. A., & Kathmann, N. (2015). Overactive performance monitoring as an endophenotype for obsessive-compulsive disorder: Evidence from a treatment study. *American Journal of Psychiatry, 172*(7), 665-673. https://doi.org/10.1176/appi.ajp.2014.14070886

Riesel, A., Endrass, T., Kaufmann, C., & Kathmann, N. (2011). Overactive error-related brain activity as a candidate endophenotype for obsessive-compulsive disorder: Evidence from unaffected first-degree relatives. *The American Journal of Psychiatry, 168*(3), 317-324. https://doi.org/10.1176/appi.ajp.2010.10030416

Riesel, A., Goldhahn, S., & Kathmann, N. (2017). Hyperactive performance monitoring as a transdiagnostic marker: Results from health anxiety in comparison to obsessive–compulsive disorder. *Neuropsychologia, 96*, 1-8. https://doi.org/10.1016/j.neuropsychologia.2016.12.029

Riesel, A., Härpfer, K., Kathmann, N., & Klawohn, J. (2021). In the face of potential harm: The predictive validity of neural correlates of performance monitoring for perceived risk, stress, and internalizing psychopathology during the COVID-19 pandemic. *Biological Psychiatry: Global Open Science, 1*(4), 300-309. https://doi.org/10.1016/j.bpsgos.2021.08.004

Riesel, A., Härpfer, K., Thoma, L., Kathmann, N., & Klawohn, J. (2023). Associations of neural error-processing with symptoms and traits in a dimensional sample recruited across the obsessive–compulsive spectrum. *Psychophysiology, 60*(2), e14164. https://doi.org/10.1111/psyp.14164

Riesel, A., Kathmann, N., & Klawohn, J. (2019). Flexibility of error-monitoring in obsessive-compulsive disorder under speed and accuracy instructions. *Journal of Abnormal Psychology, 128*(7), 671–677. https://doi.org/10.1037/abn0000463

Riesel, A., Klawohn, J., Grützmann, R., Kaufmann, C., Heinzel, S., Bey, K., Lennertz, L., Wagner, M., & Kathmann, N. (2019). Error-related brain activity as a transdiagnostic

endophenotype for obsessive-compulsive disorder, anxiety and substance use disorder. *Psychological Medicine, 49*(7), 1-11. https://doi.org/10.1017/S0033291719000199

Riesel, A., Weinberg, A., Endrass, T., Kathmann, N., & Hajcak, G. (2012). Punishment has a lasting impact on error-related brain activity. *Psychophysiology, 49*(2), 239-247. https://doi.org/10.1111/j.1469-8986.2011.01298.x

Riesel, A., Weinberg, A., Endrass, T., Meyer, A., & Hajcak, G. (2013). The ERN is the ERN is the ERN? Convergent validity of error-related brain activity across different tasks. *Biological Psychology, 93*(3), 377-385. https://doi.org/10.1016/j.biopsycho.2013.04.007

Russman Block, S. R., Klump, K. L., Beltz, A. M., Burt, S. A., & Moser, J. S. (2024). Ovarian hormones reduce the negative association between worry and cognitive control: A combined neural and behavioral investigation. *Psychoneuroendocrinology, 161*, 106947. https://doi.org/10.1016/j.psyneuen.2023.106947

Sandre, A., Banica, I., Riesel, A., Flake, J., Klawohn, J., & Weinberg, A. (2020). Comparing the effects of different methodological decisions on the error-related negativity and its association with behaviour and gender. *International Journal of Psychophysiology, 156*, 18-39. https://doi.org/10.1016/j.ijpsycho.2020.06.016

Saunders, B., & Inzlicht, M. (2020). Assessing and adjusting for publication bias in the relationship between anxiety and the error-related negativity. *International Journal of Psychophysiology, 155*, 87-98. https://doi.org/10.1016/j.ijpsycho.2020.05.008

Segalowitz, S. J., Santesso, D. L., Murphy, T. I., Homan, D., Chantziantoniou, D. K., & Khan, S. (2010). Retest reliability of medial frontal negativities during performance monitoring. *Psychophysiology, 47*(2), 260-270. https://doi.org/10.1111/j.1469-8986.2009.00942.x

Seow, T. X. F., Benoit, E., Dempsey, C., Jennings, M., Maxwell, A., McDonough, M., & Gillan, C. M. (2020). A dimensional investigation of error-related negativity (ERN) and self-reported psychiatric symptoms. *International Journal of Psychophysiology*. https://doi.org/10.1016/j.ijpsycho.2020.09.019

Shackman, A. J., Salomons, T. V., Slagter, H. A., Fox, A. S., Winter, J. J., & Davidson, R. J. (2011). The integration of negative affect, pain and cognitive control in the cingulate cortex. *Nature Reviews Neuroscience, 12*(3), 154-167. https://doi.org/10.1038/nrn2994

Shansky, R., & Lipps, J. (2013). Stress-induced cognitive dysfunction: hormone-neurotransmitter interactions in the prefrontal cortex. *Frontiers in Human Neuroscience, 7*, 123. https://doi.org/10.3389/fnhum.2013.00123

Shenhav, A., Botvinick, M. M., & Cohen, J. D. (2013). The expected value of control: An integrative theory of anterior cingulate cortex function. *Neuron, 79*(2), 217-240. https://doi.org/10.1016/j.neuron.2013.07.007

Simons, R. F. (2010). The way of our errors: Theme and variations. *Psychophysiology, 47*(1), 1-14. https://doi.org/10.1111/j.1469-8986.2009.00929.x

Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the State-Trait Anxiety Inventory*. Consulting Psychologists Press.

Stangier, U., & Heidenreich, T. (2003). Die Liebowitz Soziale Angst‐Skala (LSAS) [The Liebowitz social anxiety scale]. In Collegium Internationale Psychiatriae Scalarum (Ed.), *Internationale Skalen für Psychiatrie*. Hogrefe.

Suor, J. H., Calentino, A. E., Granros, M., & Burkhouse, K. L. (2021). Maternal error-related negativity relationship with offspring error-related negativity and negative parenting styles: a novel model of internalizing psychopathology risk. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*. https://doi.org/10.1016/j.bpsc.2021.10.007

Suor, J. H., Calentino, A. E., Granros, M., & Burkhouse, K. L. (2022). Maternal error-related negativity relationship with offspring error-related negativity and negative parenting styles: a novel model of internalizing psychopathology risk. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 7*(4), 435-442. https://doi.org/10.1016/j.bpsc.2021.10.007

Tang, W., Jbabdi, S., Zhu, Z., Cottaar, M., Grisot, G., Lehman, J. F., Yendiki, A., & Haber, S. N. (2019). A connectional hub in the rostral anterior cingulate cortex links areas of emotion and cognitive control. *eLife, 8*, e43761. https://doi.org/10.7554/eLife.43761

Tetzner, J., & Schuth, M. (2016). Anxiety in late adulthood: Associations with gender, education, and physical and cognitive functioning. *Psychology and Aging, 31*(5), 532-544. https://doi.org/10.1037/pag0000108

Themanson, J. R., Hillman, C. H., & Curtin, J. J. (2006). Age and physical activity influences on action monitoring during task switching. *Neurobiology of Aging, 27*(9), 1335-1345. https://doi.org/10.1016/j.neurobiolaging.2005.07.002

Torpey, D. C., Hajcak, G., & Klein, D. N. (2009). An examination of error-related brain activity and its modulation by error value in young children. *Developmental Neuropsychology, 34*(6), 749-761. https://doi.org/10.1080/87565640903265103

Vallet, W., Neige, C., Mouchet-Mages, S., Brunelin, J., & Grondin, S. (2021). Response-locked component of error monitoring in psychopathy: A systematic review and meta-analysis of error-related negativity/positivity. *Neuroscience & Biobehavioral Reviews, 123*, 104-119. https://doi.org/10.1016/j.neubiorev.2021.01.004

Valt, C., Huber, D., Erhardt, I., & Stürmer, B. (2018). Internal and external signal processing in patients with panic disorder: An event-related potential (ERP) study. *PLOS ONE, 13*(11), e0208257. https://doi.org/10.1371/journal.pone.0208257

Vidal, F., Burle, B., Bonnet, M., Grapperon, J., & Hasbroucq, T. (2003). Error negativity on correct trials: A reexamination of available data. *Biological Psychology, 64*(3), 265-282. https://doi.org/10.1016/S0301-0511(03)00097-8

Vidal, F., Hasbroucq, T., Grapperon, J., & Bonnet, M. (2000). Is the 'error negativity' specific to errors? *Biological Psychology, 51*(2-3), 109-128. https://doi.org/10.1016/S0301-0511(99)00032-0

Watson, D., & Clark, L. A. (1991). *The Mood and Anxiety Symptom Questionnaire*. University of Iowa.

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*(1), 3-14. https://doi.org/10.1037/0021-843X.104.1.3

Webber, H. E., de Dios, C., Kessler, D. A., Schmitz, J. M., Lane, S. D., & Suchting, R. (2024). A meta-analysis of electrophysiological biomarkers of reward and error monitoring in substance misuse. *Psychophysiology, n/a*(n/a), e14515. https://doi.org/10.1111/psyp.14515

Weinberg, A., Dieterich, R., & Riesel, A. (2015). Error-related brain activity in the age of RDoC: A review of the literature. *International Journal of Psychophysiology, 98*(2, Part 2), 276-299. https://doi.org/10.1016/j.ijpsycho.2015.02.029

Weinberg, A., & Hajcak, G. (2011). Longer term test–retest reliability of error-related brain activity. *Psychophysiology, 48*(10), 1420-1425. https://doi.org/10.1111/j.1469-8986.2011.01206.x

Weinberg, A., Kotov, R., & Proudfit, G. H. (2015). Neural indicators of error processing in generalized anxiety disorder, obsessive-compulsive disorder, and major depressive disorder. *Journal of Abnormal Psychology, 124*(1), 172-185. https://doi.org/10.1037/abn0000019

Weinberg, A., Kujawa, A., & Riesel, A. (2022). Understanding trajectories to anxiety and depression: Neural responses to errors and rewards as indices of susceptibility to stressful life events. *Current Directions in Psychological Science*, 1-9. https://doi.org/10.1177/09637214211049228

Weinberg, A., Olvet, D. M., & Hajcak, G. (2010). Increased error-related brain activity in generalized anxiety disorder. *Biological Psychology, 85*(3), 472-480. https://doi.org/10.1016/j.biopsycho.2010.09.011

Weinberg, A., Riesel, A., & Hajcak, G. (2012). Integrating multiple perspectives on error-related brain activity: The ERN as a neural indicator of trait defensive reactivity. *Motivation and Emotion, 36*(1), 84-100. https://doi.org/10.1007/s11031-011-9269-y

Weissman, M. M., Wickramaratne, P., Adams, P., Wolk, S., Verdeli, H., & Olfson, M. (2000). Brief screening for family psychiatric history: The Family History Screen. *Archives of General Psychiatry, 57*(7), 675-682. https://doi.org/10-1001/pubs.Arch_Gen_Psychiatry-ISSN-0003-990x-57-7-yoa8214

World Medical Association. (2013). World Medical Association declaration of Helsinki: Ethical principles for medical research involving human subjects. *JAMA, 310*(20), 2191-2194. https://doi.org/10.1001/jama.2013.281053

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review, 111*(4), 931-959. https://doi.org/10.1037/0033-295X.111.4.931

Yordanova, J., Falkenstein, M., Hohnsbein, J., & Kolev, V. (2004). Parallel systems of error processing in the brain. *NeuroImage, 22*(2), 590-602. https://doi.org/10.1016/j.neuroimage.2004.01.040

Zainal, N. H., & Newman, M. G. (2023). A cross-lagged prospective network analysis of depression and anxiety and cognitive functioning components in midlife community adult women. *Psychological Medicine, 53*(9), 4160-4171. https://doi.org/10.1017/S0033291722000848

Zhang, G., Garrett, D. R., & Luck, S. J. (2023). Optimal filters for ERP research II: Recommended settings for seven common ERP components. *bioRxiv*. https://doi.org/10.1101/2023.06.13.544794

Preregistration

The research objectives and methodology of this study were preregistered prior to data collection using the Open Science Framework (OSF) platform (https://osf.io/kxv5h).

Data Availability

The dataset and analytic code used to support the findings of this study have been deposited in the OSF repository (https://doi.org/10.17605/OSF.IO/7DCM3).

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

Funding Statement

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CRediT

Kai Härpfer: Conceptualization, data curation, formal analysis, investigation, methodology, project administration, resources, software, validation, visualization, writing – original draft, writing – review and editing

Hannes Per Carsten: Conceptualization, data curation, investigation, methodology, project administration, resources, validation, visualization, writing – review and editing

Franziska Magdalena Kausche: Conceptualization, investigation, validation, writing – review and editing

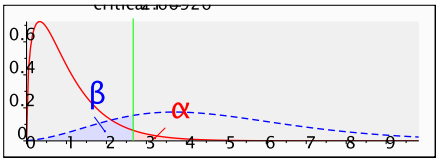
Anja Riesel: Conceptualization, funding acquisition, methodology, project administration, resources, validation, writing – review and editing

SUPPLEMENTARY MATERIALS

Figures

Figure S1

*Output with Specifications of the A Priori Sample Size Calculation Using G*Power, version 3.1.9.6.*

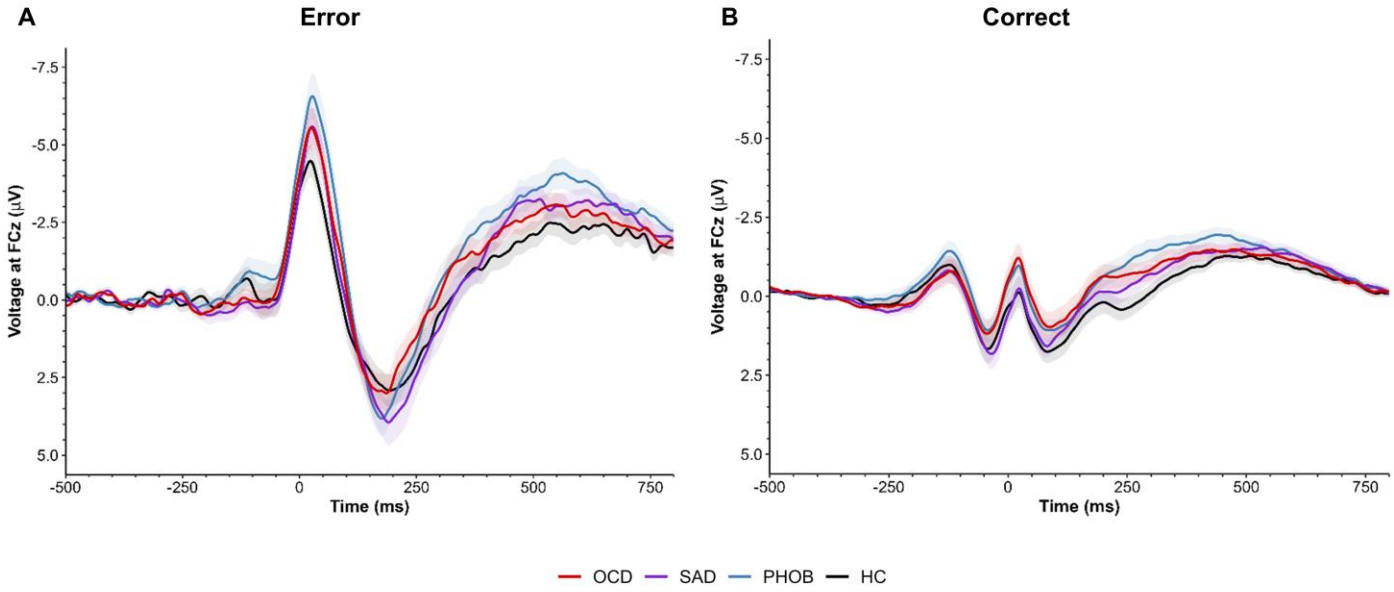


F tests – ANOVA: Repeated measures, within-between interaction

Analysis:	A priori: Compute required sample size		
Input:	Effect size f(V)	=	0.28
	α err prob	=	0.05
	Power (1-β err prob)	=	0.80
	Number of groups	=	4
	Number of measurements	=	2
	Nonsphericity correction ε	=	1
Output:	Noncentrality parameter λ	=	11.2896000
	Critical F	=	2.6692564
	Numerator df	=	3.0000000
	Denominator df	=	140
	Total sample size	=	144
	Actual power	=	0.8030278

Figure S2

Grand-Averaged Waveforms and Corresponding Topographies for Error and Correct Trials of the Clinical Groups and the Healthy Control Group.



Note. Response-locked grand-averaged waveforms of erroneous and correct trials of each group (A, B). OCD = Obsessive-Compulsive Disorder; SAD = Social Anxiety Disorder; PHOB = Specific Phobia; HC = Healthy Control Group. $N = 151$.

Tables

Table S1

Decomposition of Current Clinical Diagnoses across the Diagnostic Groups.

	Overall (*n*=156)	OCD (*n*=39)	SAD (*n*=39)	PHOB (*n*=39)	CON (*n*=39)	Comparison		
	N	*n*	*n*	*n*	*n*	χ^2	*df*	*p*
Depression	**64**	22	25	15	2	33.28	3	**<.001**
Specific Phobia	**49**	5	5	39	0	115.54	3	**<.001**
Social Anxiety Disorder	**46**	6	39	1	0	126.90	3	**<.001**
Obsessive-Compulsive Disorder	**40**	39	1	0	0	150.89	3	**<.001**
Sleeping Disorder	**20**	9	4	6	1	7.80	3	.050
Post-Traumatic Stress Disorder	**11**	3	5	2	1	3.42	3	.331
Generalized Anxiety Disorder	**10**	3	4	3	0	3.85	3	.279
Agoraphobia	**6**	2	3	1	0	3.47	3	.325
Somatisation & Health Anxiety	**5**	4	1	0	0	8.89	3	**.031**
Panic Disorder	**5**	2	1	2	0	2.27	3	.518
Tic Spectrum	**4**	0	3	0	1	6.16	3	.104
Externalizing Spectrum	**4**	0	2	2	0	4.11	3	.250
Eating Disorder & Body Dysmorphia	**3**	3	0	0	0	9.18	3	**.027**
Separation Anxiety	**1**	0	1	0	0	3.02	3	.389
Total Number of Diagnoses	**268**	**98**	**94**	**71**	**5**			

Note. Sorted by overall frequency; obsessive-compulsive Disorder (OCD), social anxiety disorder (SAD), specific phobia (PHOB), control group (CON); tic spectrum includes skin picking, trichotillomania, and hoarding; externalizing spectrum includes attention deficit hyperactivity disorder and intermittent explosive disorder. $N = 156$.

$p < .05$ printed in bold

Table S2

Demographical, Questionnaire, and Clinical Data of the Clinical and Family Risk Groups.

	Nonclinical + No Risk (n = 19)		Nonclinical + Risk (n = 15)		Clinical + No Risk (n = 34)		Clinical + Risk (n =88)		Group Comparison			
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	χ^2 / *F*	*df*	η^2	*p*
Demographical												
Gender (f/m)	10/9		13/2		26/8		66/22		5.87	3		.118
Age	30.32	8.53	30.60	8.36	29.35	10.25	27.44	6.85	1.30	3, 152	0.03	.277
Education	12.32	1.00	12.00	1.00	11.97	1.31	12.31	0.88	1.19	3, 152	0.02	.317
Questionnaires												
OCI-R	4.16_a	3.59	6.00_a	5.68	10.68_a	10.56	18.30_b	13.25	12.36	3, 152	0.20	**<.001**
LSAS-SR	16.32_a	9.98	17.53_a	10.32	38.50_b	28.55	53.21_b	32.22	13.84	3, 151	0.22	**<.001**
SMSP	2.17_a	3.13	0.60_a	0.83	4.27_a	4.91	8.80_b	8.39	10.45	3, 150	0.17	**<.001**
PSWQ	34.63_a	8.53	40.80_{ab}	11.00	49.09_b	13.57	57.10_c	10.97	26.06	3, 152	0.34	**<.001**
MASQ-AA	19.42_a	2.81	21.80_a	4.33	24.00_a	6.87	29.05_b	9.37	10.76	3, 152	0.18	**<.001**
STAI-T	32.63_a	6.99	35.33_{ab}	7.68	42.79_b	12.69	50.53_c	12.07	18.42	3, 152	0.27	**<.001**
STAI-S	31.53_a	6.46	32.86_a	5.64	38.33_{ab}	11.04	43.07_b	12.45	8.05	3, 150	0.14	**<.001**
BDI-II	3.16_a	4.37	2.80_a	4.16	9.79_a	11.07	15.55_b	11.83	12.05	3, 152	0.19	**<.001**
AUDIT	3.74	2.79	4.00	3.64	4.29	3.14	4.06	3.19	0.13	3, 152	0.00	.943
EHI	72.89	38.56	84.67	14.82	73.24	34.79	73.18	38.97	0.45	3, 152	0.01	.717
Clinical												
Diagnoses	0.00_a	0.00	0.00_a	0.00	1.91_b	1.58	2.82_c	1.81	26.88	3, 152	0.35	**<.001**
CGI-S	0.95_a	0.62	0.87_a	0.52	2.82_b	0.90	3.41_c	1.08	55.67	3, 151	0.53	**<.001**
GAF	90.42_a	4.44	87.67_a	7.16	70.62_b	13.77	61.76_c	13.45	40.73	3, 151	0.45	**<.001**

Note. Gender (f = female, m = male); age and education in years; PSWQ = Penn State Worry Questionnaire; MASQ-AA = Mood and Anxiety Symptom Questionnaire (Anxious Arousal Subscale); OCI-R = Obsessive-Compulsive Inventory; LSAS-SR = Liebowitz Social Anxiety Scale – self report; SMSP = Severity Measure for Specific Phobia; STAI = State-Trait-Anxiety Inventory (Trait and State Subscale); BDI-II = Beck Depression Inventory II; AUDIT = Alcohol Use Disorders Identification Test; EHI = Edinburgh Handedness Inventory (Modified); Diagnoses include index and comorbid diagnoses (n); CGI-S = Clinical Global Impression – Severity of Illness; GAF = Global Assessment of Functioning; degrees of freedom are deviating for some questionnaires due to missing data; means with different subscripts within rows indicate significant differences according to Sidak corrected post-hoc t-tests with p <.05. N = 156.

p < .05 are printed in bold.

Table S3

Electrophysiological and Behavioral Data of the Clinical and Family Risk Groups.

	Nonclinical + No Risk (n = 19)		Nonclinical + Risk (n = 15)		Clinical + No Risk (n = 34)		Clinical + Risk (n =88)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
ERPs								
ERN (μV)	-1.94	2.78	-3.40	2.38	-3.26	3.06	-4.34	3.88
CRN (μV)	1.29	1.90	0.43	2.65	0.75	2.08	0.00	2.60
Behavior								
Accuracy (%)	86.26	7.39	83.65	12.77	87.18	7.18	84.94	8.28
RT correct (ms)	428.59	42.49	424.73	54.09	421.29	44.54	424.57	44.75
RT incorrect (ms)	386.85	69.93	366.40	61.29	357.91	64.06	355.01	53.18
PES (ms)	27.04	21.44	48.71	30.85	37.63	25.05	43.51	26.59

Note. ERPs = event-related potentials; ERN = error-related negativity (mean amplitude 0 – 100 ms at FCz); CRN = correct-response negativity (mean amplitude 0 – 100 ms at FCz); RT = response time; PES = post-error slowing. N = 156.

Table S4

ANCOVA Results for ERN and CRN across Diagnostic Groups Using Different Scoring Approaches.

	Response				Group				Response × Group			
Variable	*F*	*df*	η_p^2	*p*	*F*	*df*	η_p^2	*p*	*F*	*df*	η_p^2	*p*
Baseline -500 to -300ms												
Mean Amplitude	19.89	1, 151	0.12	**<.001**	1.56	3, 151	0.03	.201	0.95	3, 151	0.02	.418
Adaptive Mean	19.06	1, 151	0.11	**<.001**	1.51	3, 151	0.03	.214	0.63	3, 151	0.01	.598
Baseline -200 to 0ms												
Mean Amplitude	24.18	1, 151	0.14	**<.001**	1.60	3, 151	0.03	.192	0.71	3, 151	0.01	.546
Adaptive Mean	23.90	1, 151	0.14	**<.001**	1.85	3, 151	0.04	.141	0.53	3, 151	0.01	.661
Baseline Independent												
Peak-to-Peak	10.63	1, 151	0.07	**.001**	0.80	3, 151	0.02	.497	0.63	3, 151	0.01	.595

Note. ERN = error-related negativity; CRN = correct-response negativity; groups (OCD, SAD, PHOB, CON); response (correct, incorrect); mean amplitude (0 – 100 ms); adaptive mean around the peak (± 20 ms); peak-to-peak (difference between most positive pre-response and most negative post-response peak); all quantifications were based on electrode FCz; the ANCOVA included the covariate error response time, as this variable differed between the PHOB and CON group. $N = 156$.

$p < .05$ are printed in bold

Table S5

ANCOVA Results for ERN and CRN across Diagnostic Groups Including the Healthy Control Group Using Different Scoring Approaches.

	Response				Group				Response × Group			
Variable	*F*	*df*	η_p^2	*p*	*F*	*df*	η_p^2	*p*	*F*	*df*	η_p^2	*p*
Baseline -500 to -300ms												
Mean Amplitude	18.05	1, 146	0.11	**<.001**	2.49	3, 146	0.04	.062	1.03	3, 146	0.02	.379
Adaptive Mean	17.55	1, 146	0.11	**<.001**	2.55	3, 146	0.05	.058	0.79	3, 146	0.02	.504
Baseline -200 to 0ms												
Mean Amplitude	22.29	1, 146	0.13	**<.001**	2.45	3, 146	0.05	.066	0.86	3, 146	0.02	.462
Adaptive Mean	22.39	1, 146	0.13	**<.001**	3.00	3, 146	0.06	**.033**	0.76	3, 146	0.02	.520
Baseline Independent												
Peak-to-Peak	10.00	1, 146	0.06	**.002**	1.34	3, 146	0.03	.265	0.76	3, 146	0.02	.517

Note. ERN = error-related negativity; CRN = correct-response negativity; groups (OCD, SAD, PHOB, HC); response (correct, incorrect); mean amplitude (0 – 100 ms); adaptive mean around the peak (± 20 ms); peak-to-peak (difference between most positive pre-response and most negative post-response peak); all quantifications were based on electrode FCz; the ANCOVA included the covariate error response time, as this variable differed between the PHOB and HC group. $N = 151$.

$p < .05$ are printed in bold

Table S6

ANCOVA Results of the Effects of Clinical Status of an Internalizing Disorder and Family Risk for Internalizing Psychopathology on the ERN and CRN Using Different Scoring Approaches.

	F	*df*	η_p^2	*p*
Baseline -500 to -300ms				
Mean Amplitude				
Response	19.10	1, 150	0.11	**<.001**
Response × Clinical	0.69	1, 150	0.01	.406
Response × FHS Internalizing	0.54	1, 150	0.00	.466
Response × Clinical × FHS Internalizing	0.03	1, 150	0.00	.860
Clinical	3.32	1, 150	0.02	.071
Clinical × FHS Internalizing	0.12	1, 150	0.00	.731
FHS Internalizing	4.35	1, 150	0.03	**.039**
Adaptive Mean				
Response	18.37	1, 150	0.11	**<.001**
Response × Clinical	1.18	1, 150	0.01	.278
Response × FHS Internalizing	0.08	1, 150	0.00	.780
Response × Clinical × FHS Internalizing	0.02	1, 150	0.00	.882
Clinical	4.01	1, 150	0.03	**.047**
Clinical × FHS Internalizing	0.05	1, 150	0.00	.822
FHS Internalizing	2.78	1, 150	0.02	.098
Baseline -200 to 0ms				
Mean Amplitude				
Response	23.26	1, 150	0.13	**<.001**
Response × Clinical	1.03	1, 150	0.01	.313
Response × FHS Internalizing	0.13	1, 150	0.00	.719
Response × Clinical × FHS Internalizing	0.26	1, 150	0.00	.613
Clinical	6.40	1, 150	0.04	**.012**
Clinical × FHS Internalizing	0.02	1, 150	0.00	.902
FHS Internalizing	0.03	1, 150	0.00	.861
Adaptive Mean				
Response	22.95	1, 150	0.13	**<.001**
Response × Clinical	1.64	1, 150	0.01	.203
Response × FHS Internalizing	0.41	1, 150	0.00	.524
Response × Clinical × FHS Internalizing	0.21	1, 150	0.00	.651
Clinical	8.69	1, 150	0.06	**.004**
Clinical × FHS Internalizing	0.07	1, 150	0.00	.791
FHS Internalizing	0.35	1, 150	0.00	.553
Baseline Independent				
Peak-to-Peak				
Response	10.57	1, 150	0.07	**.001**
Response × Clinical	1.99	1, 150	0.01	.160
Response × FHS Internalizing	1.08	1, 150	0.01	.300
Response × Clinical × FHS Internalizing	0.92	1, 150	0.01	.340
Clinical	4.85	1, 150	0.03	**.029**
Clinical × FHS Internalizing	0.20	1, 150	0.00	.652
FHS Internalizing	2.99	1, 150	0.02	.086

Note. ERN = error-related negativity; CRN = correct-response negativity; FHS = Family History Screen (no risk, risk); groups (nonclinical, clinical); response (correct, incorrect); mean amplitude (0 – 100 ms); adaptive mean around the peak (± 20 ms); peak-to-peak (difference between most positive pre-response and most negative post-response peak); all quantifications were based on electrode FCz. Analyses were controlled for error response time and post-error slowing, as these performance variables differed between the respective groups. $N = 156$.

$p < .05$ are printed in bold

Table S7

The Role of Clinical Status, PSWQ, and MASQ-AA on the ERN and CRN within the Combined Sample across the Severity Continuum.

	ERN									CRN								
	b	$SE_{boot.}$	*β*	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*	*b*	$SE_{boot.}$	*β*	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*
Baseline -500 to -300ms																		
Mean Amplitude						0.02	1.62	9, 236	.110						0.08	3.35	9, 236	**<.001**
Gender	0.78	0.61	0.09	1.33	.198					1.08	0.34	0.18	2.85	**.002**				
Age	0.07	0.03	0.13	1.97	**.017**					0.02	0.02	0.06	0.87	.320				
Clinical	-1.07	0.59	-0.14	-1.70	.069					0.05	0.41	0.01	0.13	.907				
PSWQ	-0.04	0.05	-0.13	-0.89	.366					-0.06	0.03	-0.28	-1.90	.060				
MASQ-AA	0.03	0.06	0.07	0.48	.569					-0.01	0.04	-0.04	-0.31	.766				
Clinical × PSWQ	0.04	0.06	0.10	0.71	.476					0.08	0.04	0.27	2.12	**.032**				
Clinical × MASQ-AA	-0.09	0.08	-0.17	-1.16	.244					-0.02	0.05	-0.06	-0.42	.668				
PSWQ × MASQ-AA	-0.01	0.01	-0.20	-1.36	.294					0.00	0.00	-0.15	-1.04	.351				
Clinical × PSWQ × MASQ-AA	0.01	0.01	0.24	1.54	.194					0.00	0.00	0.01	0.05	.969				
Adaptive Mean						0.03	1.96	9, 236	**.044**						0.10	4.03	9, 236	**<.001**
Gender	0.84	0.61	0.09	1.33	.174					1.34	0.40	0.21	3.26	**.002**				
Age	0.10	0.03	0.16	2.45	**.004**					0.02	0.02	0.05	0.79	.397				
Clinical	-1.30	0.64	-0.15	-1.93	**.043**					0.22	0.43	0.04	0.51	.598				
PSWQ	-0.03	0.05	-0.10	-0.69	.491					-0.07	0.03	-0.32	-2.18	**.037**				
MASQ-AA	0.05	0.06	0.09	0.68	.421					-0.02	0.05	-0.06	-0.42	.692				
Clinical × PSWQ	0.03	0.06	0.07	0.56	.545					0.08	0.04	0.26	2.06	**.040**				
Clinical × MASQ-AA	-0.13	0.09	-0.21	-1.46	.127					-0.01	0.06	-0.03	-0.19	.864				
PSWQ × MASQ-AA	-0.01	0.01	-0.18	-1.27	.255					-0.01	0.01	-0.21	-1.52	.225				
Clinical × PSWQ × MASQ-AA	0.01	0.01	0.27	1.72	.120					0.00	0.01	0.05	0.33	.776				
Baseline -200 to 0ms																		
Mean Amplitude						0.03	1.69	9, 236	.091						0.05	2.46	9, 236	**.011**
Gender	0.42	0.45	0.06	0.96	.360					0.73	0.31	0.16	2.39	**.018**				
Age	0.05	0.02	0.13	1.96	**.017**					-0.01	0.02	-0.03	-0.46	.610				
Clinical	-1.04	0.45	-0.18	-2.25	**.020**					-0.02	0.31	-0.01	-0.06	.949				
PSWQ	-0.02	0.03	-0.10	-0.69	.481					-0.05	0.02	-0.30	-2.02	**.022**				
MASQ-AA	0.03	0.04	0.08	0.59	.452					0.02	0.03	0.10	0.72	.385				
Clinical × PSWQ	0.02	0.04	0.06	0.48	.619					0.07	0.03	0.33	2.54	**.007**				

Clinical × MASQ-AA	-0.08	0.06	-0.18	-1.29	.179					-0.04	0.04	-0.13	-0.93	.242				
PSWQ × MASQ-AA	-0.01	0.00	-0.18	-1.20	.275					0.00	0.00	-0.06	-0.39	.671				
Clinical × PSWQ × MASQ-AA	0.01	0.01	0.25	1.60	.116					0.00	0.00	-0.08	-0.48	.566				
Adaptive Mean						0.05	2.27	9, 236	**.018**						0.09	3.75	9, 236	**<.001**
Gender	0.48	0.47	0.07	1.00	.306					1.00	0.32	0.21	3.25	**.004**				
Age	0.08	0.02	0.17	2.60	**.004**					-0.01	0.02	-0.03	-0.48	.598				
Clinical	-1.27	0.52	-0.20	-2.52	**.014**					0.15	0.31	0.04	0.46	.607				
PSWQ	-0.02	0.04	-0.07	-0.44	.653					-0.06	0.02	-0.38	-2.60	**.002**				
MASQ-AA	0.04	0.05	0.12	0.86	.329					0.02	0.03	0.07	0.54	.499				
Clinical × PSWQ	0.01	0.05	0.04	0.30	.753					0.08	0.03	0.34	2.68	**.004**				
Clinical × MASQ-AA	-0.11	0.06	-0.24	-1.68	.067					-0.03	0.04	-0.09	-0.66	.434				
PSWQ × MASQ-AA	-0.01	0.01	-0.16	-1.10	.300					0.00	0.00	-0.16	-1.15	.166				
Clinical × PSWQ × MASQ-AA	0.01	0.01	0.29	1.84	.069					0.00	0.00	-0.02	-0.11	.887				
Baseline Independent																		
Peak-to-Peak						0.07	2.95	9, 236	**.002**						0.05	2.32	9, 236	**.016**
Gender	0.85	0.51	0.10	1.61	.110					0.66	0.30	0.14	2.19	**.036**				
Age	0.08	0.03	0.16	2.52	**.006**					0.00	0.02	0.00	0.01	.993				
Clinical	-1.85	0.56	-0.25	-3.27	**.003**					-0.19	0.30	-0.05	-0.60	.532				
PSWQ	0.01	0.04	0.05	0.30	.761					-0.07	0.03	-0.42	-2.81	**.020**				
MASQ-AA	0.04	0.05	0.08	0.62	.518					0.05	0.03	0.20	1.43	.107				
Clinical × PSWQ	0.00	0.05	-0.01	-0.04	.976					0.07	0.03	0.35	2.66	**.014**				
Clinical × MASQ-AA	-0.14	0.07	-0.26	-1.85	.053					-0.05	0.04	-0.18	-1.27	.127				
PSWQ × MASQ-AA	-0.01	0.01	-0.17	-1.18	.259					-0.01	0.00	-0.40	-2.73	**.010**				
Clinical × PSWQ × MASQ-AA	0.01	0.01	0.34	2.20	**.037**					0.01	0.00	0.31	1.97	**.044**				

Note. ERN = error-related negativity; CRN = correct-response negativity; Gender (0 = female, 1 = male); age in years; clinical (0 = nonclinical, 1 = clinical); PSWQ = Penn State Worry Questionnaire (mean centered); MASQ-AA = Mood and Anxiety Symptom Questionnaire (mean centered); mean amplitude (0 – 100 ms); adaptive mean around the peak (± 20 ms); peak-to-peak (difference between most positive pre-response and most negative post-response peak); all quantifications were based on electrode FCz. $N = 246$.

$p < .05$ are printed in bold

Table S8

The Role of PSWQ and MASQ-AA on the ERN and CRN Using Different Scoring Approaches.

	ERN									CRN								
	b	$SE_{boot.}$	*β*	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*	*b*	SE_{boot}	*β*	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*
Baseline -500 to -300ms																		
Mean Amplitude						0.02	1.62	9, 236	.110						0.08	3.35	9, 236	**<.001**
Gender	0.71	0.63	0.09	1.08	.262					1.18	0.41	0.21	2.65	**.007**				
Age	0.06	0.03	0.13	1.55	**.042**					0.01	0.02	0.02	0.24	.793				
PSWQ	-0.04	0.03	-0.16	-1.47	.090					-0.01	0.02	-0.03	-0.29	.749				
MASQ-AA	0.03	0.04	0.06	0.51	.494					-0.03	0.03	-0.09	-0.74	.378				
PSWQ × MASQ-AA	0.00	0.00	-0.03	-0.33	.637					0.00	0.00	-0.10	-1.01	.167				
Adaptive Mean						0.03	1.96	9, 236	**.044**						0.10	4.03	9, 236	**<.001**
Gender	0.81	0.64	0.09	1.13	.222					1.42	0.49	0.23	2.91	**.007**				
Age	0.08	0.03	0.16	1.95	**.017**					0.00	0.03	-0.01	-0.10	.916				
PSWQ	-0.05	0.03	-0.18	-1.60	.065					-0.01	0.02	-0.05	-0.49	.576				
MASQ-AA	0.04	0.04	0.10	0.82	.261					-0.03	0.03	-0.10	-0.83	.308				
PSWQ × MASQ-AA	0.00	0.00	-0.03	-0.31	.699					0.00	0.00	-0.09	-0.97	.270				
Baseline -200 to 0ms																		
Mean Amplitude						0.03	1.69	9, 236	.091						0.05	2.46	9, 236	**.011**
Gender	0.14	0.46	0.02	0.30	.752					0.73	0.41	0.15	1.78	.068				
Age	0.04	0.02	0.12	1.40	.071					-0.02	0.02	-0.07	-0.79	.362				
PSWQ	-0.04	0.02	-0.19	-1.75	.073					0.01	0.02	0.05	0.41	.644				
MASQ-AA	0.01	0.03	0.04	0.33	.702					-0.03	0.03	-0.12	-1.01	.240				
PSWQ × MASQ-AA	0.00	0.00	0.00	0.00	.997					0.00	0.00	-0.03	-0.30	.719				
Adaptive Mean						0.05	2.27	9, 236	**.018**						0.09	3.75	9, 236	**<.001**
Gender	0.24	0.49	0.04	0.45	.637					0.97	0.44	0.20	2.41	**.036**				
Age	0.06	0.02	0.16	1.97	**.015**					-0.03	0.02	-0.10	-1.19	.197				
PSWQ	-0.04	0.02	-0.21	-1.92	**.044**					0.00	0.02	0.02	0.15	.883				
MASQ-AA	0.03	0.04	0.09	0.78	.388					-0.04	0.03	-0.15	-1.21	.166				
PSWQ × MASQ-AA	0.00	0.00	0.00	0.00	.999					0.00	0.00	-0.04	-0.37	.662				
Baseline Independent																		
Peak-to-Peak						0.07	2.95	9, 236	**.002**						0.05	2.32	9, 236	**.016**
Gender	0.57	0.53	0.08	0.92	.287					0.62	0.36	0.14	1.70	.078				

Age	0.07	0.03	0.16	1.94	**.015**	-0.01	0.02	-0.05	-0.61	.486
PSWQ	-0.03	0.02	-0.11	-1.02	.248	0.00	0.01	-0.01	-0.10	.908
MASQ-AA	0.02	0.04	0.05	0.41	.674	0.00	0.03	-0.01	-0.07	.946
PSWQ × MASQ-AA	0.00	0.00	0.03	0.34	.640	0.00	0.00	-0.03	-0.33	.729

Note. ERN = error-related negativity; CRN = correct-response negativity; Gender (0 = female, 1 = male); age in years; PSWQ = Penn State Worry Questionnaire (mean centered); MASQ-AA = Mood and Anxiety Symptom Questionnaire (mean centered); mean amplitude (0 – 100 ms); adaptive mean around the peak (± 20 ms); peak-to-peak (difference between most positive pre-response and most negative post-response peak); all quantifications were based on electrode FCz. $N = 156$.

$p < .05$ are printed in bold

Table S9

The Role of Gender and PSWQ on the ERN and CRN within the Combined Sample across the Severity Continuum.

	ERN									CRN								
	b	$SE_{boot.}$	*β*	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*	*b*	SE_{boot}	*β*	*t*	$p_{boot.}$	R^2_{corr}	*F*	*df*	*p*
Baseline -500 to -300ms																		
Mean Amplitude						0.03	2.41	6, 239	**.028**						0.06	3.79	6, 239	**.001**
Gender	1.30	0.93	0.15	1.34	.155					1.76	0.63	0.30	2.78	**.002**				
Age	0.08	0.03	0.15	2.30	**.008**					0.02	0.02	0.06	0.99	.289				
Clinical	-0.67	0.62	-0.09	-1.05	.284					0.00	0.42	0.00	-0.01	.994				
PSWQ	-0.04	0.02	-0.12	-1.46	.135					-0.04	0.02	-0.18	-2.22	**.010**				
Gender × Clinical	-0.40	1.18	-0.03	-0.30	.715					-0.57	0.87	-0.07	-0.66	.461				
Gender × PSWQ	0.09	0.05	0.16	1.74	**.048**					0.07	0.03	0.20	2.19	**.003**				
Adaptive Mean						0.05	2.98	6, 239	**.008**						0.08	4.40	6, 239	**<.001**
Gender	1.26	1.07	0.13	1.21	.223					2.11	0.68	0.33	3.05	**<.001**				
Age	0.11	0.03	0.19	2.88	**<.001**					0.02	0.03	0.05	0.81	.397				
Clinical	-0.87	0.63	-0.10	-1.26	.170					0.24	0.47	0.04	0.53	.626				
PSWQ	-0.04	0.02	-0.12	-1.38	.122					-0.05	0.02	-0.21	-2.56	**.016**				
Gender × Clinical	-0.15	1.32	-0.01	-0.11	.894					-0.75	0.91	-0.09	-0.78	.397				
Gender × PSWQ	0.11	0.05	0.18	1.92	**.039**					0.07	0.03	0.18	2.00	**.019**				
Baseline -200 to 0ms																		
Mean Amplitude						0.05	3.05	6, 239	**.007**						0.04	2.78	6, 239	**.013**
Gender	1.14	0.73	0.17	1.60	.116					1.56	0.43	0.33	3.04	**<.001**				
Age	0.07	0.02	0.16	2.51	**.003**					-0.01	0.02	-0.02	-0.34	.709				
Clinical	-0.62	0.44	-0.11	-1.31	.157					-0.04	0.31	-0.01	-0.12	.910				
PSWQ	-0.03	0.02	-0.15	-1.75	.058					-0.02	0.01	-0.12	-1.44	.129				
Gender × Clinical	-0.83	0.91	-0.10	-0.85	.347					-0.96	0.63	-0.15	-1.35	.110				
Gender × PSWQ	0.10	0.03	0.24	2.53	**.003**					0.06	0.02	0.22	2.30	**.007**				
Adaptive Mean						0.07	3.83	6, 239	**.001**						0.07	4.15	6, 239	**<.001**
Gender	1.10	0.87	0.15	1.41	.192					1.91	0.45	0.40	3.70	**<.001**				
Age	0.09	0.02	0.21	3.27	**<.001**					-0.01	0.02	-0.03	-0.46	.588				
Clinical	-0.82	0.45	-0.13	-1.58	.065					0.21	0.32	0.05	0.60	.536				
PSWQ	-0.03	0.02	-0.13	-1.62	.077					-0.03	0.01	-0.18	-2.15	**.046**				
Gender × Clinical	-0.58	1.04	-0.06	-0.54	.545					-1.13	0.66	-0.18	-1.59	.087				

Gender × PSWQ	0.11	0.04	0.25	2.71	**.006**					0.06	0.03	0.21	2.28	**.015**	
Baseline Independent															
Peak-to-Peak						0.07	4.13	6, 239	**<.001**						0.03	2.24	6, 239	**.040**
Gender	1.21	0.87	0.15	1.38	.167					1.37	0.42	0.30	2.72	**.005**	
Age	0.10	0.03	0.20	3.20	**<.001**					0.00	0.02	0.00	0.07	.932	
Clinical	-1.32	0.51	-0.18	-2.27	**.015**					0.09	0.36	0.02	0.26	.818	
PSWQ	-0.02	0.02	-0.07	-0.85	.328					-0.02	0.01	-0.14	-1.64	.136	
Gender × Clinical	-0.10	1.06	-0.01	-0.08	.922					-0.86	0.62	-0.14	-1.25	.160	
Gender × PSWQ	0.11	0.04	0.22	2.42	**.006**					0.06	0.02	0.22	2.32	**.007**	

Note. ERN = error-related negativity; CRN = correct-response negativity; Gender (0 = female, 1 = male); age in years; clinical (0 = nonclinical, 1 = clinical); PSWQ = Penn State Worry Questionnaire (mean centered); adaptive mean around the peak (± 20 ms); peak-to-peak (difference between most positive pre-response and most negative post-response peak); all quantifications were based on electrode FCz. $N = 246$.

$p < .05$ are printed in bold

Study 3

In the Face of Potential Harm: The Predictive Validity of Neural Correlates of Performance Monitoring for Perceived Risk, Stress, and Internalizing Psychopathology During the COVID-19 Pandemic

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Archival Report

In the Face of Potential Harm: The Predictive Validity of Neural Correlates of Performance Monitoring for Perceived Risk, Stress, and Internalizing Psychopathology During the COVID-19 Pandemic

Anja Riesel, Kai Härpfer, Norbert Kathmann, and Julia Klawohn

ABSTRACT

BACKGROUND: The COVID-19 pandemic is a major life stressor posing serious threats not only to physical but also to mental health. To better understand mechanisms of vulnerability and identify individuals at risk for psychopathological symptoms in response to stressors is critical for prevention and intervention. The error-related negativity (ERN) has been discussed as a neural risk marker for psychopathology, and this study examined its predictive validity for perceived risk, stress, and psychopathological symptoms during the COVID-19 pandemic.

METHODS: A total of 113 individuals who had participated as healthy control participants in previous electroencephalography studies (2014–2019) completed a follow-up online survey during the first COVID-19 wave in Germany. Associations of pre-pandemic ERN and correct-response negativity (CRN) with perceived risk regarding COVID-19 infection, stress, and internalizing symptoms during the pandemic were examined using mediation models.

RESULTS: Pre-pandemic ERN and CRN were associated with increased perceived risk regarding a COVID-19 infection. Via this perceived risk, the ERN and CRN were associated with increased stress during the pandemic. Furthermore, risk perception and stress mediated indirect effects of ERN and CRN on internalizing psychopathology, including anxiety, depression, and obsessive-compulsive symptoms, while controlling for the effects of pre-pandemic symptom levels.

CONCLUSIONS: In summary, heightened pre-pandemic performance monitoring showed indirect associations with increases in psychopathological symptoms during the first COVID-19 wave via effects on perceived COVID-19 risk and stress. These results further strengthen the notion of performance monitoring event-related potentials as transdiagnostic neural risk markers and highlight the relevance of stress as a catalyst for symptom development.

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In March 2020, the World Health Organization declared the outbreak of COVID-19 as a worldwide pandemic. Since then, this pandemic has had a profound impact on life, requiring individuals to adapt to changing circumstances and new hardships. A growing number of reports suggest that the COVID-19 pandemic has led to increased levels of anxiety and distress (1–6). At the same time, psychopathological effects of the pandemic differ across individuals, and our understanding of the pathways to anxiety and depression and the identification of individuals at risk for mental illness is critical for the development of targeted intervention and prevention.

Neuroscience methods, including event-related potentials (ERPs), are gaining importance for studying mechanisms that lead to mental disorders. This line of research strives to identify biomarkers informing models of pathomechanisms and predictions of future psychopathology (7), particularly under conditions of stress (8–12). The error-related negativity (ERN) (13) is of particular interest in this regard and has been highlighted as a promising transdiagnostic risk marker (14). The ERN is a well-validated neural marker of error processing with good psychometric properties (15), observable as a sharp negativity over frontocentral brain regions after errors. The corresponding ERP on correct responses, the correct-response negativity (CRN), has been studied to a lesser extent. Existing findings point toward the CRN representing broader general performance monitoring functions shared between both correct and incorrect actions, whereas an additional error-specific process is present only after incorrect responses (16,17). Both ERPs are assumed to be generated by activity in the midcingulate cortex, specifically the anterior cingulate cortex (18,19), and are thought to prompt adaptive responses with the aim to avoid harm (20,21). Variations in ERN and CRN are assumed to be trait like, shaped by genes (22), learning history (23–25), and situational demands

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(13,26–28). Furthermore, variations in ERN magnitude have been linked to individual differences in harm avoidance (14), anxious apprehension (29), or threat sensitivity (30). Similarly, increased ERN amplitudes are shared by disorders such as obsessive-compulsive disorder (OCD) and various anxiety disorders, including generalized and social anxiety disorder [e.g., (31,32)]. In contrast to ERN, CRN variations have been less frequently studied in relation to traits and psychopathology, and alterations have been reported less consistently. However, one exception is an association between elevated CRN and OCD, which has been shown repeatedly (33), suggesting changes not only in error-specific but also in general performance monitoring processes (16) and a possible association with increased concern about the correctness of actions even in the absence of errors.

Beyond this observed cross-sectional association of the ERN with psychopathology, a growing number of studies support that alterations in ERN magnitude represent a neural vulnerability marker preceding symptom development. Alterations in ERN are present in healthy individuals with increased familial risk for OCD or anxiety (14,34,35), and increased ERN amplitudes persist after symptom improvement by psychotherapy in OCD and anxiety (36–39), suggesting that they are not a consequence of symptoms. Moreover, the ERN can be used to predict symptom onset across different disorders (9,11,40). In concert, these findings underscore that ERN alterations can lead to maladaptive behaviors and mental disorders, further highlighting its critical role in mental health. However, the relationship between alterations in performance monitoring and the development of symptoms is complex, and its role in psychopathological trajectories is still poorly understood. Existing findings highlight the ERN as a potential endophenotype for internalizing psychopathology [e.g., (14)], suggesting that it may play an important mediating role on the pathways from genetic risk to psychopathological phenotypes (41,42). More recently, the role of stress as a catalyst in this relationship has been emphasized (11,43). Few studies to date have used ERPs to investigate reactions to real-life stressors. One study in children showed that the ERN prospectively predicted response to a natural disaster in that youths with initially higher ERN amplitudes showed stronger increases in anxiety after the event (8). Similarly, an increased ERN has been shown to render individuals more susceptible to the adverse effects of interpersonal stress during transition to university, thereby increasing risk for heightened anxiety (8). This growing evidence supports an involvement of the ERN in the emergence of stress susceptibility (43). Nevertheless, the mechanisms by which performance monitoring ERPs influence stress susceptibility and symptom development remain poorly understood, a research gap that this study aims to address.

The COVID-19 pandemic, as a major real-life stressor, provides a unique research environment to investigate the hypothesis that overactive performance monitoring (i.e., ERN and CRN) translates into psychopathology through heightened risk perception and its effects on stress. We explored the association of pre-pandemic ERN and CRN in previously healthy participants (2014–2019) (14,44,45) with self-reported perceived risk, stress, and psychopathological symptom dimensions, such as anxiety, depression, and obsessive-compulsive symptoms during the first COVID-19 wave in Germany.

METHODS AND MATERIALS

Participants

We invited 317 adults who had participated as healthy comparison participants in three previous electroencephalography studies conducted at Humboldt-Universität zu Berlin (14,44,45) to an online survey and clinical phone interview. The invitation was accepted by 140 participants (44.2%), but only 123 (38.8%) completed all questionnaires and 121 (38.2%) could be interviewed on the phone. Two participants reported that they had tested positive for COVID-19 at that time and were omitted from data analysis because asking for the perceived risk of an infection or severe course became obsolete. In addition, 8 participants were excluded because <6 error segments were available for ERN assessment (46). The final sample consisted of 113 participants (62.8% female) aged 20–63 years (mean = 33.47, SD = 10.35). These participants did not differ from the full participant pool in age ($t_{315} = -0.699$, $p = .485$), gender ($\chi^2_{3,317} = 3.058$, $p = .080$), trait anxiety (Spielberger State-Trait Anxiety Inventory [STAI-T]) ($t_{315} = 0.034$, $p = .973$), depressive symptoms (Beck Depression Inventory-II [BDI-II]) ($t_{315} = 0.227$, $p = .820$), obsessive-compulsive symptoms (Obsessive-Compulsive Inventory-Revised [OCI-R]) ($t_{315} = 0.266$, $p = .790$), CRN ($t_{311} = -0.32$, $p = .751$), and ERN ($t_{296} = -0.07$, $p = .947$).

Procedure

Baseline assessments were conducted during study participation in previous projects, between 0.83 and 5.38 years (mean = 2.87, SD = 1.51) prior to this investigation. Electrophysiological data of all participants had been recorded using a flanker task, and detailed study information and cross-sectional results have been reported elsewhere (14,44,45). However, raw data were reprocessed for this study to ensure identical procedures (details below). Participants who had agreed to be recontacted for future studies during initial study participation received information of the objectives and methods of this investigation. Those who agreed to participate then completed several online questionnaires and were interviewed via phone based on the Structured Clinical Interview for DSM-IV (47). Clinical data were collected between February 11 and May 19, 2020, while COVID-19 infection rates and media reports were rising across Europe and in Germany. For reference, the first case of COVID-19 infection in Germany was confirmed on January 29, 2020, and federal contact restrictions were implemented on March 23, 2020. The study procedures were in accordance with the Declaration of Helsinki as approved by the local ethics committee. Participants gave informed consent prior to the baseline and follow-up assessment and received a monetary compensation of €15 for the follow-up assessment.

Measures

The follow-up online survey consisted of several questionnaires: depressive symptoms were assessed with BDI-II (21

items; 4-point Likert scale 0–3; Cronbach's α = 0.90) (48,49), obsessive-compulsive symptoms with OCI-R (18 items; 5-point Likert scale 0–4; α = 0.88) (50,51), and trait anxiety using the trait subscale of STAI-T (4-point Likert scale 1–4; α = 0.92) (52,53). Stress was assessed using the Stress and Coping Inventory (SCI) (21 items; 5-point Likert scale 1–7; α = 0.88) (54), including the subscales strain, stress symptoms, and coping strategies. Perceived COVID-19 risk was measured by the average of 2 items (Likert scale 0–100; α = 0.69) asking participants to rate "How likely do you think you will become infected with COVID-19 within the next month?" and "How likely do you think you would experience a severe course of COVID-19, if you were infected?" As a measure of objective risk regarding COVID-19, participants were asked to self-categorize whether or not they have an increased risk of infection, including occupation (e.g., employment in health care, retail, or public transportation) and risk of a more severe course due to known medical risk factors (e.g., age over 60 years, overweight, hypertension).

Task

At the initial assessment, participants performed a modified arrow version of the flanker task (36) presented on a 19-inch liquid crystal display monitor in a dimly lit, electrically shielded cabin. Sets of 5 vertically aligned arrows, including 1 target and 4 flanker arrows (set size approximately 2.5° × 2.5°), were presented using Presentation Software (Neurobehavioral Systems, Inc.) with a fixation phase (200–1200 ms), stimulus presentation (100 ms), and a response window (maximum 1000 ms). Half of the stimuli were incongruent (i.e., target arrow pointed in the opposite direction); 480 stimuli were presented pseudorandomly. Participants were instructed to indicate direction of the target arrow as fast and accurately as possible. One study (44) provided performance feedback to the participants in between blocks, whereas the other two studies repeated the instruction irrespective of performance. Elicited ERPs did not differ between projects, neither for ERN ($F_{2,110}$ = 1.02, p = .365) nor for CRN ($F_{2,110}$ = 2.30, p = .106).

Electrophysiological Recording and Processing

Electrophysiological activity was recorded using 64 Ag/AgCl electrodes and two 32-channel BrainAmp amplifiers (Brain Products GmbH). Electrodes were mounted on a cap with equidistant layout (Easycap). Additional electrodes were placed at nasion, neck, and left infraorbital site and a ground electrode on the right cheek; Cz served as recording reference. Impedances were kept below 5 kΩ. The continuous signal was recorded with a low-cutoff time constant of 10 seconds and a high-cutoff frequency of 250 Hz. Sampling rate was 1000 Hz.

Data were processed with Brain Vision Analyzer 2.2 (Brain Products GmbH). The electroencephalogram was filtered by zero phase shift Butterworth bandpass filters from 0.1 to 30 Hz (24 dB/octave roll-off) and a 50-Hz notch filter. Ocular artifacts were removed using independent component analysis (55); components were semiautomatically identified by visual inspection. After re-referencing electroencephalography data to the common average of all scalp electrodes, response-locked segments were epoched from −500 to 1000 ms and baseline corrected using the −500 to −300-ms interval (15). Segments with artifacts were automatically removed if there was a voltage step >50 μV between data points, the absolute voltage range exceeded ±200 μV, or the voltage was <0.5 μV within 100-ms intervals. Average data loss due to artifact rejection was small (mean = 0.57%, SD = 1.22), and no participant had >25% excluded segments. Segments were discarded (mean = 12.24%, SD = 13.68) if response times were <100 or >800 ms; remaining segments were averaged separately for correct and erroneous responses. The ERN and CRN were scored as the mean activity from 0 to 100 ms after response at electrode FCz, where signals were maximal. Both ERPs had excellent psychometric properties as reflected by the Spearman-Brown–corrected split-half reliability of odd and even trials (r_{ERN} = 0.87, r_{CRN} = 0.99).

Data Analysis

Statistical analyses were conducted with SPSS version 25.0 (IBM Corp.) using a two-tailed α = 0.05. Pearson correlations were conducted to determine associations between variables at baseline and follow-up.

Exploratively, a series of mediation models was tested to examine effects of baseline ERPs on risk, stress, and symptoms during the pandemic. Mediation analyses were conducted using the PROCESS Macro for SPSS, version 3.5 (56), applying model 4 for simple mediation and model 6 for the serial mediation models to calculate 95% confidence intervals (CIs) around the indirect effect with 5000 bootstrap resamples. Because time between baseline and follow-up varied between participants, this was included as covariate in all mediation models. We used separate mediation models using the ERN and CRN, respectively, as predictors and self-reported experienced stress during the pandemic as outcome, with perceived COVID-19 risk as the mediator. In addition, specificity was examined by computing similar models with objective risk as the mediator and other ERPs as predictors.

Because experienced stress was closely related to symptoms during the pandemic (Table 1), we additionally tested indirect effects of ERN and CRN via perceived risk and stress on these symptoms in another set of models including two serial mediators. Baseline ERN or CRN, respectively, were used as predictors, while follow-up perceived COVID-19 risk and self-reported stress were included as serial mediators in the prediction of symptoms. Separate models were applied to predict symptoms of anxiety, OCD, and depression at follow-up, while controlling for the respective symptoms at baseline as covariates.

RESULTS

Table 1 shows demographic and clinical characteristics; Table S1 presents frequencies of new-onset diagnoses. Pearson correlation coefficients for associations between ERPs at baseline and symptoms measured at baseline and follow-up are presented in Table 2. A depiction of individual symptom changes between time points is shown in the Supplement (Figure S1). Neither the ERN nor CRN assessed at baseline was directly related to stress (SCI) or symptoms (BDI-II, STAI-T, OCI-R). However, significant negative correlations between ERN and CRN and perceived COVID-19 risk at follow-up were present, indicating that larger (i.e., more negative) ERN

Table 1. Demographic, Self-report/Clinical, and ERP Data in the Baseline and Follow-up Sample

Variable	Baseline	Follow-up
Demographic Data		
Gender, female/male, *n*	71/42	71/42
Age, years	30.84 (10.17)	33.47 (10.35)
Clinical Data		
BDI-II	5.07 (5.97)	6.05 (6.56)
OCI-R	9.07 (8.31)	9.10 (8.64)
STAI-T	36.96 (9.08)	36.89 (9.27)
SCI	–	43.25 (16.71)
COVID-19 perceived risk[a]	–	23.01 (20.67)
COVID 19 objective risk, risk/no risk[b]	–	38/75
ERP Data		
CRN at FCz, μV	0.62 (2.70)	–
ERN at FCz, μV	−4.55 (4.06)	–

Values are presented as mean (SD) unless otherwise indicated. $N = 113$.

BDI-II, Beck Depression Inventory-II; CRN, correct-response negativity; ERN, error-related negativity; ERP, event-related potential; OCI-R, Obsessive-Compulsive Inventory-Revised; SCI, Stress and Coping Inventory; STAI-T, Spielberger State-Trait Anxiety Inventory–Trait subscale.

[a]Averaged perceived risk of infection and severe course of disease.

[b]Dichotomous self-categorization of personal risk for COVID-19 based on occupation and medical factors.

and CRN amplitudes were associated with higher perceived COVID-19 risk estimates. The scatterplots for the associations between pre-pandemic ERN and CRN and perceived COVID-19 risk, as well as grand averages incorporating a median split regarding risk estimates to further visualize these associations, are shown in Figure 1. Perceived risk showed a significant positive correlation with objective risk. However, objective risk was not related to the ERN and CRN. Furthermore, perceived stress (SCI) during the pandemic was significantly correlated with depressive (BDI-II), anxious (STAI-T), and obsessive-compulsive (OCD-R) symptoms, whereas perceived COVID-19 risk was only correlated with BDI-II scores. Among the three symptom dimensions, STAI-T and BDI-II were strongly correlated with each other, while both were only moderately correlated with OCI-R.

A mediation model testing perceived risk as mediator between pre-pandemic ERN and stress at follow-up indicated a significant total model ($F_{3,109} = 4.591$, $p = .005$, $R^2 = 0.112$) (see Figure 2 for all coefficients). Importantly, while the direct effect of baseline ERN and stress at follow-up was not significant ($c' = 0.605$, SE = 0.380, $t = 1.591$, $p = .115$, 95% CI −0.149 to 1.358), the indirect effect of baseline ERN and stress during the pandemic via the perceived COVID-19 was significant (ab = −0.240, SE = 0.120, 95% CI −0.497 to −0.032). The analogous mediation model with baseline CRN as predictor of stress at follow-up was also significant ($F_{3,109} = 3.665$, $p = .015$, $R^2 = 0.092$). Again, there was no significant direct effect on stress ($c' = -0.049$, SE = 0.594, $t = -0.083$, $p = .934$, 95% CI −1.226 to 1.128), but the indirect effect via perceived COVID-19 risk was significant (ab = −0.437, SE = 0.260, 95% CI −1.034 to −0.036).

To examine potential distinct contributions of the ERN and CRN, we used the residualized ERN (residuals from a regression of CRN on ERN) as predictor of stress via risk perception. This model was not significant (Figure S2), suggesting that these results are rather driven by the overlap of both components than an error-specific process. Accordingly, a mediation model using the arithmetic mean of the ERN and CRN as predictor indicated a significant indirect effect on stress via

Table 2. Bivariate Correlations of ERPs With COVID-19 Risk, Stress, and Symptom Measures

Variable	1	2	3	4	5	6	7	8	9	10	11
Baseline											
1 ERN	0.87	–	–	–	–	–	–	–	–	–	–
2 CRN	0.62[a]	0.99	–	–	–	–	–	–	–	–	–
3 BDI-II	0.11	−0.03	0.90	–	–	–	–	–	–	–	–
4 OCI-R	0.14	0.01	0.35[a]	0.86	–	–	–	–	–	–	–
5 STAI-T	0.13	0.02	0.72[a]	0.44[a]	0.92	–	–	–	–	–	–
Follow-up											
6 COVID-19 objective risk	0.11	0.04	0.17	0.12	0.24[b]	0.03	–	–	–	–	–
7 COVID-19 perceived risk	−0.19[b]	−0.25[c]	0.01	0.06	0.05	0.33[a]	0.69	–	–	–	–
8 SCI	0.08	−0.10	0.39[a]	0.36[a]	0.47[a]	0.30[c]	0.27[c]	0.88	–	–	–
9 BDI-II	0.07	−0.14	0.59[a]	0.40[a]	0.52[a]	0.26[c]	0.24[b]	0.66[a]	0.90	–	–
10 OCI-R	0.04	−0.04	0.25[c]	0.63[a]	0.34[a]	0.12	0.08	0.47[a]	0.40[a]	0.88	–
11 STAI-T	0.13	−0.01	0.65[a]	0.43[a]	0.74[a]	0.22[b]	0.15	0.57[a]	0.78[a]	0.43[a]	0.92

Baseline refers to pre-pandemic assessment. Follow-up refers to assessment during pandemic. COVID-19 objective risk refers to averaged objective risk for COVID-19 based on occupation and medical factors, and COVID-19 perceived risk refers to averaged perceived risk of infection and severe course of disease. Correlations are displayed as Pearson's *r*; psychometric properties depicted in the diagonal with Spearman-Brown–corrected split-half reliability for ERP data and Cronbach's alpha for questionnaire data. $N = 113$.

BDI-II, Beck Depression Inventory-II; CRN, correct-response negativity; ERN, error-related negativity; OCI-R, Obsessive-Compulsive Inventory-Revised; SCI, Stress and Coping Inventory; STAI-T, Spielberger State-Trait Anxiety Inventory–Trait subscale.

[a]$p < .001$.

[b]$p < .05$.

[c]$p < .01$.

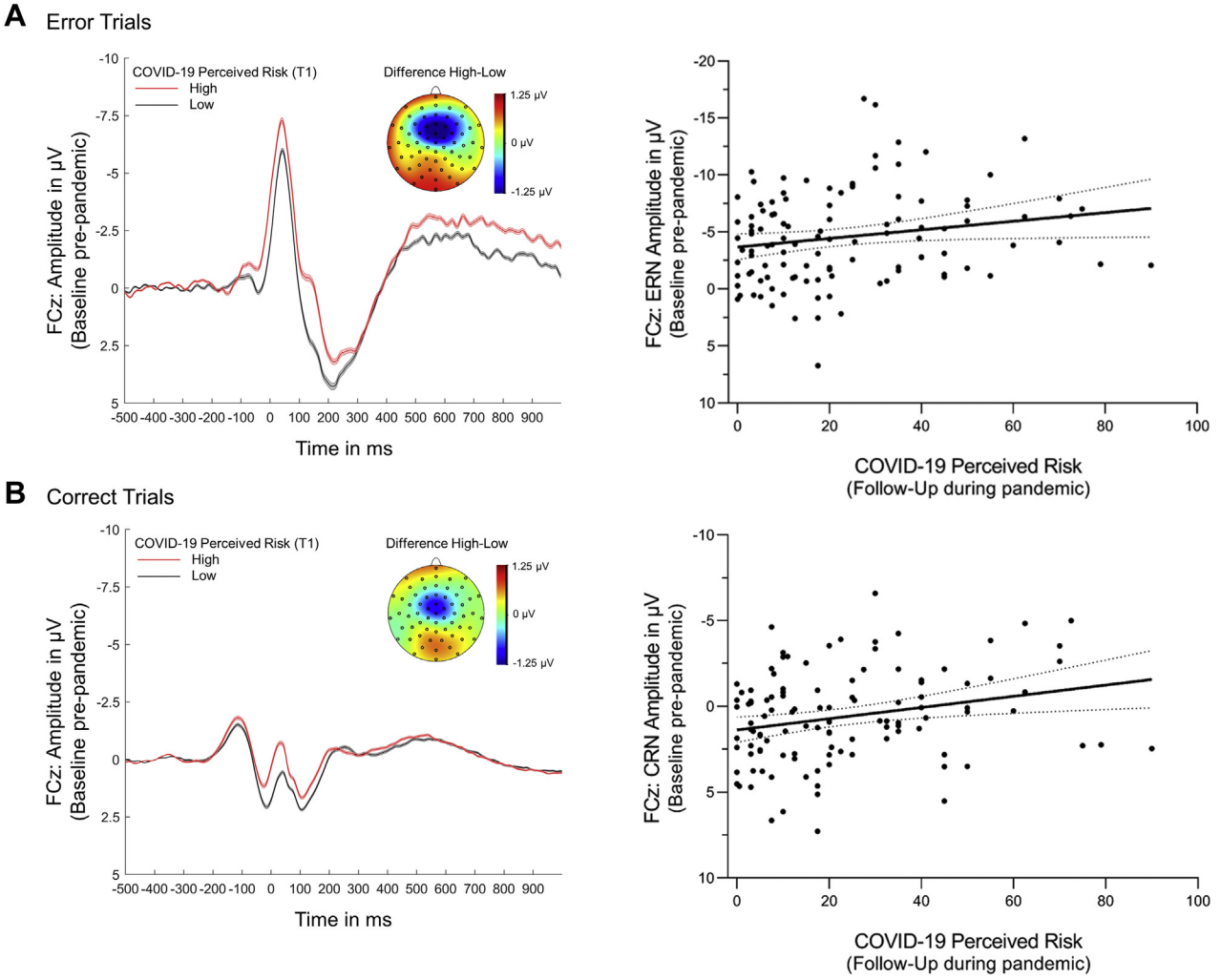

Figure 1. Grand average waveforms for error and correct trials as a function of COVID-19 risk (median split in high and low) and scatterplots for the error-related negativity (ERN) and the correct-response negativity (CRN) with COVID-19 risk. **(A)** The upper left panel shows grand average waveforms for error trials measured before the pandemic, split by individuals indicating high (i.e., above median) and low (i.e., below median) perceived COVID-19 risk, and scalp distribution of the high vs. low-risk difference (0–100 ms). Shading around waveforms indicates standard errors at the respective time point. The upper right panel displays the scatterplot for the association of the ERN at the pre-pandemic baseline and perceived COVID-19 risk at follow-up. **(B)** The lower left panel shows grand average waveforms and scalp distribution for correct trials; the lower right panel displays the respective scatterplot for the CRN and perceived COVID-19 risk. T1, follow-up during pandemic.

perceived risk (Figure S3). Furthermore, mediation models examining whether objective risk mediated an association between pre-pandemic ERN or CRN and stress at follow-up did not reach significance, supporting a specific role of perceived risk (Figures S4 and S5). To further probe specificity for the ERN and CRN, Pe on error trials and stimulus-locked N2 for correct trials were tested as predictors. None of these models reached significance (Tables S5 and S6).

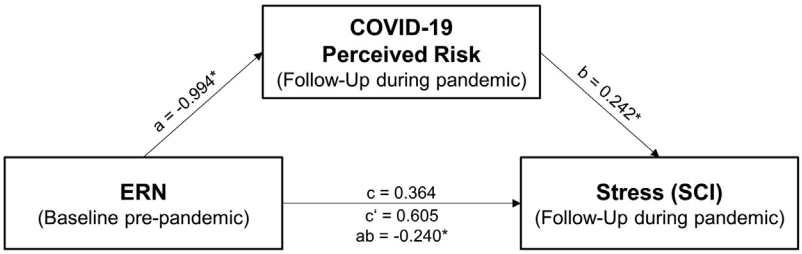

Figure 2. Mediation model with pre-pandemic error-related negativity (ERN) as predictor of stress at follow-up and COVID-19 risk as mediator. Time between baseline and follow-up is controlled for by implementation as covariate in the model. *$p < .05$. SCI, Stress and Coping Inventory.

Using serial mediation models, we examined the indirect effects of ERN and CRN on psychopathology while correcting for the respective baseline symptoms. For the three models using STAI-T, BDI-II, and OCI-R at follow-up as outcome variables, an indirect effect of ERN on symptoms, mediated via perceived risk and stress, was observed (Table 3 for all coefficients and Figure 3 for the exemplary model). Similarly, the serial mediation models to predict symptoms from the CRN at baseline indicated indirect effects through perceived COVID-19 risk and stress (Table 4).

DISCUSSION

To our knowledge, this study is among the first longitudinal studies to investigate the utility of neural correlates to predict behavioral and clinical outcomes in the context of the COVID-19 pandemic. Previous research has identified overactive performance monitoring ERPs (i.e., ERN and CRN) as risk markers for internalizing disorders (9,14,31,38), but the underlying pathways and the role of stress are still largely unclear (11,43). This study revealed the following key findings. Both the ERN and CRN were prospectively associated with risk perception regarding COVID-19. Moreover, this perceived COVID-19 risk functioned as mediator for indirect effects of pre-pandemic ERN and CRN on stress during the pandemic. Finally, indirect effects of ERN and CRN on psychopathological symptoms during the first COVID-19 wave were observed, mediated by effects on perceived risk and stress. Notably, these associations were independent of pre-pandemic symptom levels, indicating a prediction of stress-related changes in symptoms.

Individuals with higher (i.e., more negative) pre-pandemic ERN or CRN amplitudes reported an increased perceived risk for an infection and a severe course of COVID-19 disease during the pandemic. This association with risk estimation complements previous knowledge, specifically studies linking reduced ERN amplitudes or reduced activity of the anterior cingulate cortex, i.e., the main generator of the ERN, to heightened risk taking (57,58) and increased anterior cingulate cortex activity to risk aversion during decision making (58). Furthermore, an association between ERN and risk perception corresponds with findings that conceptualize an elevated ERN as a low-threshold alarm signal in line with a better safe than sorry logic (20,59,60) and suggest relationships to harm avoidance (14) and threat sensitivity (30). Moreover, alterations in the ERN, and less consistently the CRN, have been observed across disorders such as OCD and generalized anxiety disorder [e.g., (32)], known to be characterized by cognitive biases such as overestimation of threat and risk aversion (61–63). In addition, interventions aimed at reducing attentional bias to threatening information have been shown to decrease the ERN (45,64). Collectively, these findings support an association of heightened ERN/CRN with alterations in risk perception. The risk measure used here was tailored specifically to the COVID-19 pandemic, but the fit with previous findings suggests that similar associations might apply to risk estimation of other life stressors as well.

The ERN and CRN also predicted individual stress levels during the pandemic, mediated via effects on risk perception. With regard to potential underlying mechanisms, this seems to suggest that individual differences in performance monitoring might influence how individuals appraise risk for harm when confronted with real-life stressors, which then determines resulting stress levels. In line with previous reports, this may suggest an effect of the ERN on susceptibility to stress (11). However, it may also indicate that ERPs of performance monitoring influence which individuals are more likely to experience stress, i.e., in the sense of dependent stressors (i.e., stressors to which an individual contributes). Finally, experiencing stress may in turn affect the function of the performance monitoring system [e.g., (23–25)]. Future research is needed to improve our understanding of the complex relationship between increased performance monitoring and stress, possibly incorporating more objective stress measures, e.g., cortisol responses.

Table 3. Coefficients for the Mediation Models With the ERN as Predictor, Perceived COVID-19 Risk and Stress as Serial Mediators, and Symptoms (STAI-T, OCI-R, or BDI-II) as Outcomes, Controlling for Respective Symptoms at Baseline and Time Between Baseline and Follow-up as Covariates

	STAI-T: Coefficient (95% CI)	OCI-R: Coefficient (95% CI)	BDI-II: Coefficient (95% CI)
Paths			
a_1	−1.073[a] (−2.031 to −0.114)	−1.075[a] (−2.034 to −0.117)	−1.022[a] (−1.979 to −0.064)
a_2	0.294 (−0.395 to 0.983)	0.381 (−0.349 to 1.111)	0.412 (−0.299 to 1.123)
b_1	0.023 (−0.034 to 0.076)	−0.017 (−0.079 to 0.046)	0.034 (−0.009 to 0.076)
b_2	0.145[a] (0.068 to 0.223)	0.151[a] (0.070 to 0.232)	0.187[a] (0.131 to 0.243)
d_{21}	0.208[a] (0.074 to 0.341)	0.217[a] (0.076 to 0.358)	0.230[a] (0.091 to 0.367)
Effects			
c	0.070 (−0.223 to 0.364)	−0.091 (−0.411 to 0.218)	0.007 (−0.244 to 0.258)
c′	0.084 (−0.196 to 0.366)	−0.131 (−0.443 to 0.180)	0.008 (−0.202 to 0.218)
a_1b_1	−0.024 (−0.110 to 0.065)	0.018 (−0.044 to 0.098)	−0.036 (−0.115 to 0.018)
a_2b_2	0.043 (−0.063 to 0.142)	0.058 (−0.054 to 0.157)	0.077 (−0.046 to 0.201)
$a_1d_{21}b_2$	−0.032[a] (−0.079 to −0.002)	−0.035[a] (−0.077 to −0.004)	−0.044[a] (−0.106 to −0.004)

$N = 113$.

BDI-II, Beck Depression Inventory-II; OCI-R, Obsessive-Compulsive Inventory-Revised; ERN, error-related negativity; STAI-T, Spielberger State-Trait Anxiety Inventory–Trait subscale.

[a]Significant at $p < .05$.

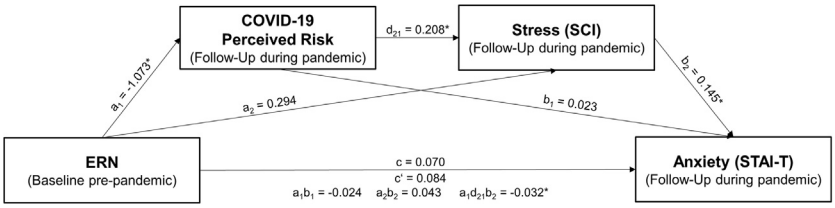

Figure 3. Exemplary depiction of mediation model to examine the error-related negativity (ERN) at baseline as a predictor of anxiety symptoms at follow-up, with perceived COVID-19 risk and stress as mediators. Baseline Spielberger State-Trait Anxiety Inventory (STAI-T) (trait anxiety) and time between baseline and follow-up is controlled for by implementation as covariates in the model. *$p < .05$. SCI, Stress and Coping Inventory.

Consistent with vulnerability stress models (65), self-reported stress further closely related to multiple internalizing symptoms, including anxiety, depression, and OC symptoms. By this, the performance monitoring ERPs could be used to indirectly predict the development of internalizing symptoms via their effect on perceived risk and stress. This holds even when controlling for the effects of initial symptoms, supporting a predictive utility of performance monitoring ERPs for psychopathological symptoms above and beyond preexisting symptoms. It should be noted that in this study, no direct association was observed between variations in ERN and CRN and internalizing symptoms at follow-up, which is at odds with some previous reports of direct predictions [e.g., (66)] but in line with the notion that the association might be stronger or even limited to clinical groups (67). Instead, the connections observed in this study appear to be more complex, and mediating factors, such as perceived risk and stress, need to be considered. Notably, in this complex mechanistic model encompassing risk perception and stress as mediators, indirect prediction of psychopathology includes increases in depressive symptoms, whereas findings regarding direct associations of ERN/CRN alterations with depression are still rather mixed (68–70). However, the rather unspecific predictive effects across different symptoms can be explained by the indirect effects of ERN and CRN on psychopathology acting through a common mechanism in which stress plays a key role. Such a model also aligns with findings that suggest the ERN as a transdiagnostic risk marker (31,32) preceding psychopathological symptoms (9,11,66,71).

These results showed that both ERN and CRN variations were associated with risk perception and indirectly predicted symptom development, pointing to an association with increases in performance monitoring after erroneous and correct responses. In line with this, the arithmetic mean of both ERPs also acted as a significant predictor. These findings support the interpretation of alterations in a general performance monitoring process shared across both ERPs (16) being implicated in risk perception, stress reactivity, and ultimately psychopathology. In future studies on stress reactivity, the role of the CRN, which is often given little attention, should thus be considered alongside the ERN. Collectively, the results suggest that ERPs of performance monitoring represent a promising target for interventions aimed to improve symptoms or prevent psychopathology (45,72) that can be applied transdiagnostically.

This investigation has several limitations. Most importantly, the analyses are primarily exploratory, and while the results provide insight into the complex relationship between performance monitoring and psychopathology, consolidation in future studies is needed. Because the follow-up measures (i.e.,

Table 4. Coefficients for the Mediation Models With the CRN as Predictor, Perceived COVID-19 Risk and Stress as Serial Mediators, and Symptoms (STAI-T, OCI-R, or BDI-II) as Outcomes, Controlling for Respective Symptoms at Baseline and Time Between Baseline and Follow-up as Covariates

	STAI-T: Coefficient (95% CI)	OCI-R: Coefficient (95% CI)	BDI-II: Coefficient (95% CI)
Paths			
a_1	−2.064[a] (−3.487 to −0.640)	−2.041[a] (−3.462 to −0.620)	−2.012[a] (−3.436 to −0.587)
a_2	−0.335 (−1.390 to 0.721)	−0.191 (−1.308 to 0.926)	−0.069 (−1.164 to 1.026)
b_1	0.019 (−0.038 to 0.076)	−0.013 (−0.076 to 0.051)	0.028 (−0.014 to 0.071)
b_2	0.147[a] (0.070 to 0.225)	0.147[a] (0.067 to 0.228)	0.187[a] (0.131 to 0.242)
d_{21}	0.185[a] (0.049 to 0.320)	0.195[a] (0.051 to 0.339)	0.211[a] (0.070 to 0.352)
Effects			
c	−0.141 (−0.583 to 0.301)	−0.124 (−0.604 to 0.357)	−0.311 (−0.686 to 0.063)
c′	0.003 (−0.428 to 0.434)	−0.062 (−0.536 to 0.412)	−0.162 (−0.480 to 0.156)
a_1b_1	−0.039 (−0.218 to 0.130)	0.025 (−0.095 to 0.170)	−0.056 (−0.214 to 0.050)
a_2b_2	−0.049 (−0.247 to 0.101)	−0.028 (−0.216 to 0.127)	−0.013 (−0.241 to 0.161)
$a_1d_{21}b_2$	−0.056[a] (−0.154 to −0.002)	−0.059[a] (−0.142 to −0.003)	−0.079[a] (−0.211 to −0.006)

$N = 113$.

BDI-II, Beck Depression Inventory-II; CRN, correct-response negativity; OCI-R, Obsessive-Compulsive Inventory-Revised; STAI-T, Spielberger State-Trait Anxiety Inventory–Trait subscale.

[a]Significant at $p < .05$.

COVID-19 risk, stress, and symptoms) were assessed at the same time, direction of the associations cannot be discerned and the indirect paths, albeit significant and plausible, can only suggest potential mechanisms pending further replication in fully prospective investigations. In addition, the COVID-19–related risk measures used here have not been validated yet, and the observed associations need replication. Moreover, we examined psychopathology dimensionally, and it needs to be determined whether similar results can be seen with regard to clinical outcomes. Nonetheless, this approach allows for the prospective investigation of symptom development among previously healthy individuals. Moreover, although there was variability in symptoms at both assessments, no overall increase in symptoms during the pandemic was apparent in this sample. This might indicate that despite being a major life stressor, the COVID-19 pandemic did not affect all participants uniformly with regard to increases in stress and psychopathology symptoms. Finally, our results showed rather small effects, which is typical for multifactorial, complex processes such as the development of psychopathology, but again, replication in well-powered samples will be essential.

Conclusions

The COVID-19 pandemic constitutes a global health crisis with profound impact on mental health. With this study, we adopted a longitudinal approach to study mechanisms and neural predictors of such effects. Results suggest that pre-pandemic ERPs of performance monitoring (i.e., ERN and CRN) may contribute to predict risk perception, stress, and exacerbation of internalizing symptoms during such a real-life stressor. Specifically, individuals with increased neural sensitivity to errors and correct responses experienced heightened risk perception, which was further connected to elevated stress levels during the first COVID-19 wave. Through these potential mediators, the ERN and CRN were also related to increases in internalizing symptoms (anxiety, depression, and OC symptoms). These findings bear clinical relevance because they demonstrate predictive utility of performance monitoring ERPs for identification of individuals at risk for mental health issues under real-life stressors, and the mechanisms elucidated here can offer vantage points for targeted prevention efforts.

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ARTICLE INFORMATION

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REFERENCES

1. Lakhan R, Agrawal A, Sharma M (2020): Prevalence of depression, anxiety, and stress during COVID-19 pandemic. J Neurosci Rural Pract 11:519–525.
2. Liu S, Heinzel S, Haucke MN, Heinz A (2021): Increased psychological distress, loneliness, and unemployment in the spread of COVID-19 over 6 months in Germany. Medicina (Kaunas) 57:53.
3. Wang C, Pan R, Wan X, Tan Y, Xu L, McIntyre RS, *et al.* (2020): A longitudinal study on the mental health of general population during the COVID-19 epidemic in China. Brain Behav Immun 87:40–48.
4. Hawes MT, Szenczy AK, Klein DN, Hajcak G, Nelson BD (2021): Increases in depression and anxiety symptoms in adolescents and young adults during the COVID-19 pandemic [published online ahead of print Jan 13]. Psychol Med.
5. Hyland P, Shevlin M, McBride O, Murphy J, Karatzias T, Bentall RP, *et al.* (2020): Anxiety and depression in the Republic of Ireland during the COVID-19 pandemic. Acta Psychiatr Scand 142:249–256.
6. Kujawa A, Green H, Compas BE, Dickey L, Pegg S (2020): Exposure to COVID-19 pandemic stress: Associations with depression and anxiety in emerging adults in the United States. Depress Anxiety 37:1280–1288.
7. Hajcak G, Klawohn J, Meyer A (2019): The utility of event-related potentials in clinical psychology. Annu Rev Clin Psychol 15:71–95.
8. Kujawa A, Burkhouse KL (2017): Vulnerability to depression in youth: Advances from affective neuroscience. Biol Psychiatry Cogn Neurosci Neuroimaging 2:28–37.
9. Meyer A, Danielson CK, Danzig AP, Bhatia V, Black SR, Bromet E, *et al.* (2017): Neural biomarker and early temperament predict increased internalizing symptoms after a natural disaster. J Am Acad Child Adolesc Psychiatry 56:410–416.
10. Dickey L, West M, Pegg S, Green H, Kujawa A (2021): Neurophysiological responses to interpersonal emotional images prospectively predict the impact of COVID-19 pandemic-related stress on internalizing symptoms. Biol Psychiatry Cogn Neurosci Neuroimaging 6:887–897.
11. Banica I, Sandre A, Shields GS, Slavich GM, Weinberg A (2020): The error-related negativity (ERN) moderates the association between interpersonal stress and anxiety symptoms six months later. Int J Psychophysiol 153:27–36.
12. Burani K, Klawohn J, Levinson AR, Klein DN, Nelson BD, Hajcak G (2021): Neural response to rewards, stress and sleep interact to prospectively predict depressive symptoms in adolescent girls. J Clin Child Adolesc Psychol 50:131–140.
13. Gehring WJ, Goss B, Coles MGH, Meyer DE, Donchin E (1993): A neural system for error detection and compensation. Psychol Sci 4:385–390.
14. Riesel A, Klawohn J, Grützmann R, Kaufmann C, Heinzel S, Bey K, *et al.* (2019): Error-related brain activity as a transdiagnostic endophenotype for obsessive-compulsive disorder, anxiety and substance use disorder. Psychol Med 49:1207–1217.
15. Klawohn J, Meyer A, Weinberg A, Hajcak G (2020): Methodological choices in event-related potential (ERP) research and their impact on internal consistency reliability and individual differences: An examination of the error-related negativity (ERN) and anxiety. J Abnorm Psychol 129:29–37.
16. Klawohn J, Riesel A, Grützmann R, Kathmann N, Endrass T (2014): Performance monitoring in obsessive-compulsive disorder: A temporo-spatial principal component analysis. Cogn Affect Behav Neurosci 14:983–995.
17. Endrass T, Klawohn J, Gruetzmann R, Ischebeck M, Kathmann N (2012): Response-related negativities following correct and incorrect responses: Evidence from a temporospatial principal component analysis. Psychophysiology 49:733–743.
18. Debener S, Ullsperger M, Siegel M, Fiehler K, von Cramon DY, Engel AK (2005): Trial-by-trial coupling of concurrent electroencephalogram and functional magnetic resonance imaging identifies the dynamics of performance monitoring. J Neurosci 25:11730–11737.
19. Ridderinkhof KR, Ullsperger M, Crone EA, Nieuwenhuis S (2004): The role of the medial frontal cortex in cognitive control. Science 306:443–447.

20. Proudfit GH, Inzlicht M, Mennin DS (2013): Anxiety and error monitoring: The importance of motivation and emotion. Front Hum Neurosci 7:636.
21. Shackman AJ, Salomons TV, Slagter HA, Fox AS, Winter JJ, Davidson RJ (2011): The integration of negative affect, pain and cognitive control in the cingulate cortex. Nat Rev Neurosci 12:154–167.
22. Anokhin AP, Golosheykin S, Heath AC (2008): Heritability of frontal brain function related to action monitoring. Psychophysiology 45:524–534.
23. Banica I, Sandre A, Weinberg A (2019): Overprotective/authoritarian maternal parenting is associated with an enhanced error-related negativity (ERN) in emerging adult females. Int J Psychophysiol 137:12–20.
24. Riesel A, Kathmann N, Wüllhorst V, Banica I, Weinberg A (2019): Punishment has a persistent effect on error-related brain activity in highly anxious individuals twenty-four hours after conditioning. Int J Psychophysiol 146:63–72.
25. Meyer A, Proudfit GH, Bufferd SJ, Kujawa AJ, Laptook RS, Torpey DC, Klein DN (2015): Self-reported and observed punitive parenting prospectively predicts increased error-related brain activity in six-year-old children. J Abnorm Child Psychol 43:821–829.
26. Klawohn J, Endrass T, Preuss J, Riesel A, Kathmann N (2016): Modulation of hyperactive error signals in obsessive-compulsive disorder by dual-task demands. J Abnorm Psychol 125:292–298.
27. Riesel A, Kathmann N, Klawohn J (2019): Flexibility of error-monitoring in obsessive-compulsive disorder under speed and accuracy instructions. J Abnorm Psychol 128:671–677.
28. Hajcak G, Moser JS, Yeung N, Simons RF (2005): On the ERN and the significance of errors. Psychophysiology 42:151–160.
29. Moser JS, Moran TP, Schroder HS, Donnellan MB, Yeung N (2013): On the relationship between anxiety and error monitoring: A meta-analysis and conceptual framework. Front Hum Neurosci 7:466.
30. Weinberg A, Riesel A, Hajcak G (2012): Integrating multiple perspectives on error-related brain activity: The ERN as a neural indicator of trait defensive reactivity. Motiv Emot 36:84–100.
31. Riesel A (2019): The erring brain: Error-related negativity as an endophenotype for OCD-A review and meta-analysis. Psychophysiology 56:e13348.
32. Weinberg A, Dieterich R, Riesel A (2015): Error-related brain activity in the age of RDoC: A review of the literature. Int J Psychophysiol 98:276–299.
33. Endrass T, Ullsperger M (2014): Specificity of performance monitoring changes in obsessive-compulsive disorder. Neurosci Biobehav Rev 46:124–138.
34. Carrasco M, Harbin SM, Nienhuis JK, Fitzgerald KD, Gehring WJ, Hanna GL (2013): Increased error-related brain activity in youth with obsessive-compulsive disorder and unaffected siblings. Depress Anxiety 30:39–46.
35. Riesel A, Endrass T, Kaufmann C, Kathmann N (2011): Overactive error-related brain activity as a candidate endophenotype for obsessive-compulsive disorder: Evidence from unaffected first-degree relatives. Am J Psychiatry 168:317–324.
36. Huyser C, Veltman DJ, Wolters LH, de Haan E, Boer F (2011): Developmental aspects of error and high-conflict-related brain activity in pediatric obsessive-compulsive disorder: A fMRI study with a Flanker task before and after CBT. J Child Psychol Psychiatry 52:1251–1260.
37. Riesel A, Endrass T, Auerbach LA, Kathmann N (2015): Overactive performance monitoring as an endophenotype for obsessive-compulsive disorder: Evidence from a treatment study. Am J Psychiatry 172:665–673.
38. Kujawa A, Weinberg A, Bunford N, Fitzgerald KD, Hanna GL, Monk CS, *et al.* (2016): Error-related brain activity in youth and young adults before and after treatment for generalized or social anxiety disorder. Prog Neuropsychopharmacol Biol Psychiatry 71:162–168.
39. Hajcak G, Franklin ME, Foa EB, Simons RF (2008): Increased error-related brain activity in pediatric obsessive-compulsive disorder before and after treatment. Am J Psychiatry 165:116–123.
40. Marhe R, van de Wetering BJ, Franken IH (2013): Error-related brain activity predicts cocaine use after treatment at 3-month follow-up. Biol Psychiatry 73:782–788.
41. Insel TR, Cuthbert BN (2009): Endophenotypes: Bridging genomic complexity and disorder heterogeneity. Biol Psychiatry 66:988–989.
42. Kendler KS, Neale MC (2010): Endophenotype: A conceptual analysis. Mol Psychiatry 15:789–797.
43. Weinberg A, Kujawa A, Riesel A (in press): Understanding trajectories to anxiety and depression: Neural responses to errors and reward as indices of stress susceptibility. Curr Dir Psychol Sci.
44. Härpfer K, Carsten HP, Spychalski D, Kathmann N, Riesel A (2020): Were we erring? The impact of worry and arousal on error-related negativity in a non-clinical sample. Psychophysiology 57:e13661.
45. Klawohn J, Hajcak G, Amir N, Kathmann N, Riesel A (2020): Application of attentional bias modification training to modulate hyperactive error-monitoring in OCD. Int J Psychophysiol 156:79–86.
46. Olvet DM, Hajcak G (2009): The stability of error-related brain activity with increasing trials. Psychophysiology 46:957–961.
47. Wittchen HU, Zaudig M, Fydrich T (1997): Strukturiertes Klinisches Interview für DSM-IV. Göttingen: Hogrefe.
48. Beck AT, Steer RA, Brown GK (1996): Manual for the Beck Depression Inventory-II. San Antonio: Psychological Corporation.
49. Kühner C, Bürger C, Keller F, Hautzinger M (2007): Reliabilität und Validität des revidierten Beck-Depressionsinventars (BDI-II) [Reliability and validity of the Revised Beck Depression Inventory (BDI-II). Results from German samples.]. Nervenarzt 78:651–656.
50. Foa EB, Huppert JD, Leiberg S, Langner R, Kichic R, Hajcak G, Salkovskis PM (2002): The Obsessive-Compulsive Inventory: Development and validation of a short version. Psychol Assess 14:485–496.
51. Gönner S, Leonhart R, Ecker W (2007): [The German version of the obsessive-compulsive inventory-revised: A brief self-report measure for the multidimensional assessment of obsessive-compulsive symptoms]. Psychother Psychosom Med Psychol 57:395–404.
52. Spielberger CD, Gorsuch RL, Lushene RE (1970): Manual for the State-Trait Anxiety Inventory. Palo Alto: Consulting Psychologists Press.
53. Laux L, Glanzmann P, Schaffner P, Spielberger CD (1981): Das State-Trait-Angstinventar (STAI). (The State-Trait Anxiety Inventory (STAI).). Weinheim: Beltz.
54. Satow L (2012): Stress- und Coping-Inventar (SCI). In: Testarchiv E, editor. Leibniz-Zentrum für Psychologische Information und Dokumentation. Trier: ZPID.
55. Jung TP, Makeig S, Humphries C, Lee TW, McKeown MJ, Iragui V, Sejnowski TJ (2000): Removing electroencephalographic artifacts by blind source separation. Psychophysiology 37:163–178.
56. Hayes AF, Rockwood NJ (2017): Regression-based statistical mediation and moderation analysis in clinical research: Observations, recommendations, and implementation. Behav Res Ther 98:39–57.
57. Hewig J, Trippe R, Hecht H, Coles MG, Holroyd CB, Miltner WH (2007): Decision-making in Blackjack: An electrophysiological analysis. Cereb Cortex 17:865–877.
58. Paulus MP, Frank LR (2006): Anterior cingulate activity modulates nonlinear decision weight function of uncertain prospects. Neuroimage 30:668–677.
59. Weinberg A, Meyer A, Hale-Rude E, Perlman G, Kotov R, Klein DN, Hajcak G (2016): Error-related negativity (ERN) and sustained threat: Conceptual framework and empirical evaluation in an adolescent sample. Psychophysiology 53:372–385.
60. Van den Bergh O, Brosschot J, Critchley H, Thayer JF, Ottaviani C (2021): Better safe than sorry: A common signature of general vulnerability for psychopathology. Perspect Psychol Sci 16:225–246.
61. Thimm JC, Holte A, Brennen T, Wang CE (2013): Hope and expectancies for future events in depression. Front Psychol 4:470.
62. Moritz S, Jelinek L (2009): Inversion of the "unrealistic optimism" bias contributes to overestimation of threat in obsessive-compulsive disorder. Behav Cogn Psychother 37:179–193.
63. Strunk DR, Lopez H, DeRubeis RJ (2006): Depressive symptoms are associated with unrealistic negative predictions of future life events. Behav Res Ther 44:861–882.
64. Nelson BD, Jackson F, Amir N, Hajcak G (2017): Attention bias modification reduces neural correlates of response monitoring. Biol Psychol 129:103–110.

65. Hankin BL, Abela J, editors. (2005). Development of Psychopathology: A Vulnerability-Stress Perspective Vulnerability-Stress Models. Thousand Oaks: Sage Publications.
66. Meyer A, Hajcak G, Torpey-Newman DC, Kujawa A, Klein DN (2015): Enhanced error-related brain activity in children predicts the onset of anxiety disorders between the ages of 6 and 9. J Abnorm Psychol 124:266–274.
67. Saunders B, Inzlicht M (2020): Assessing and adjusting for publication bias in the relationship between anxiety and the error-related negativity. Int J Psychophysiol 155:87–98.
68. Klawohn J, Santopetro NJ, Meyer A, Hajcak G (2020): Reduced P300 in depression: Evidence from a flanker task and impact on ERN, CRN, and Pe. Psychophysiology 57:e13520.
69. Gorka SM, Burkhouse KL, Afshar K, Phan KL (2017): Error-related brain activity and internalizing disorder symptom dimensions in depression and anxiety. Depress Anxiety 34:985–995.
70. Weinberg A, Liu H, Shankman SA (2016): Blunted neural response to errors as a trait marker of melancholic depression. Biol Psychol 113:100–107.
71. Meyer A, Nelson B, Perlman G, Klein DN, Kotov R (2018): A neural biomarker, the error-related negativity, predicts the first onset of generalized anxiety disorder in a large sample of adolescent females. J Child Psychol Psychiatry 59:1162–1170.
72. Meyer A, Gibby B, Wissemann K, Klawohn J, Hajcak G, Schmidt NB (2020): A brief, computerized intervention targeting error sensitivity reduces the error-related negativity. Cogn Affect Behav Neurosci 20:172–180.

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SUPPLEMENTARY INFORMATION

Contents:

Table S1. Frequency of Clinical Diagnoses With First Onset After Baseline as Determined by SCID-Interview Conducted via Phone

	n	%
Agoraphobia	3	2.65
Depression	9	7.96
Panic	1	0.88
PTSD	2	1.77

Note. PTSD = Posttraumatic Stress Disorder

Figure S1. Figure displaying variability in symptoms at baseline and follow-up assessments

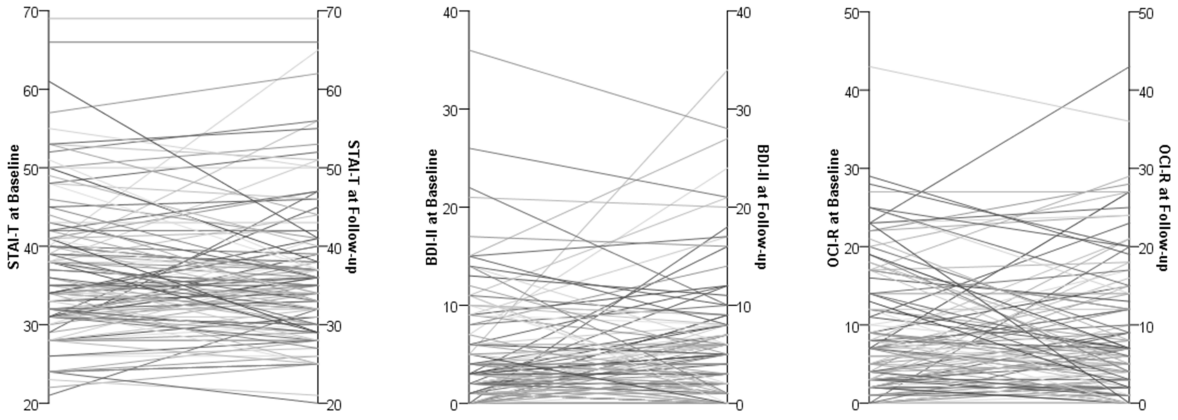


Figure S2. Results of mediation model using residualized ERN as predictor

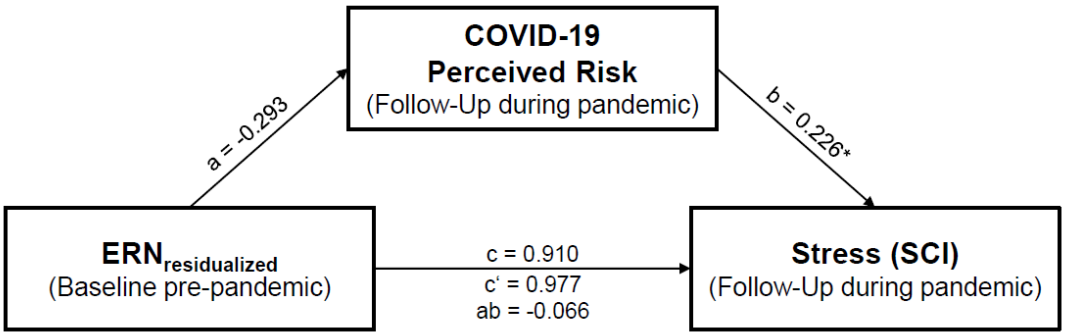


Figure S3. Results of mediation model using the arithmetic mean of ERN and CRN as predictor

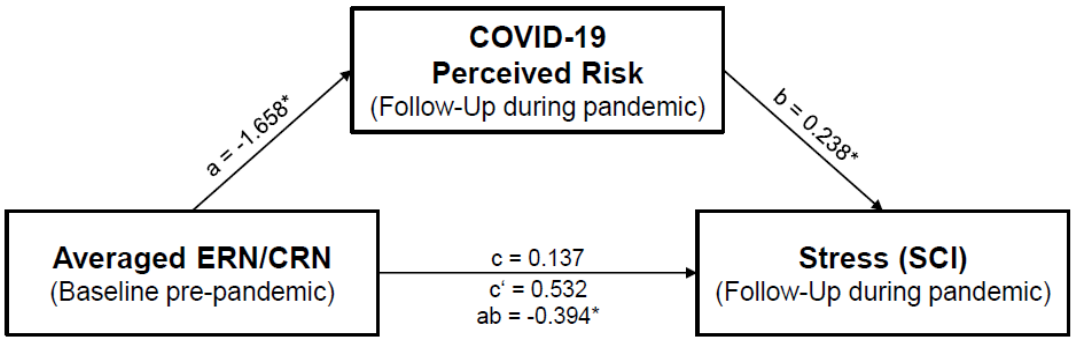


Figure S4. Results of mediation model using ERN as predictor and objective risk as mediator

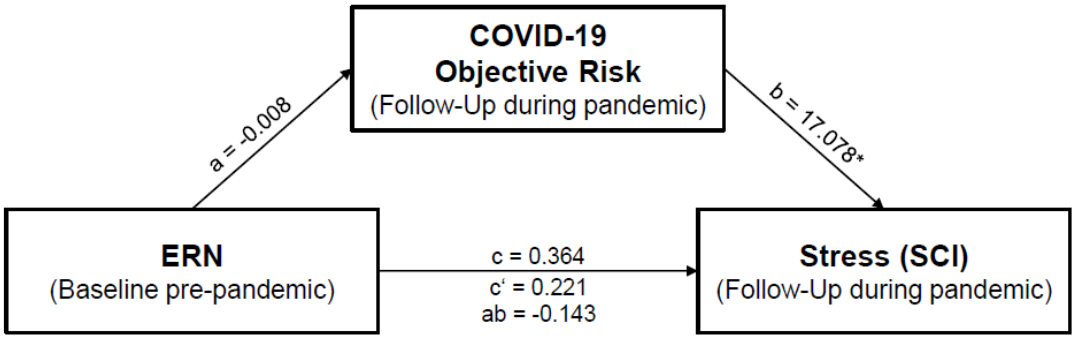


Figure S5. Results of mediation model using CRN as predictor and objective risk as mediator

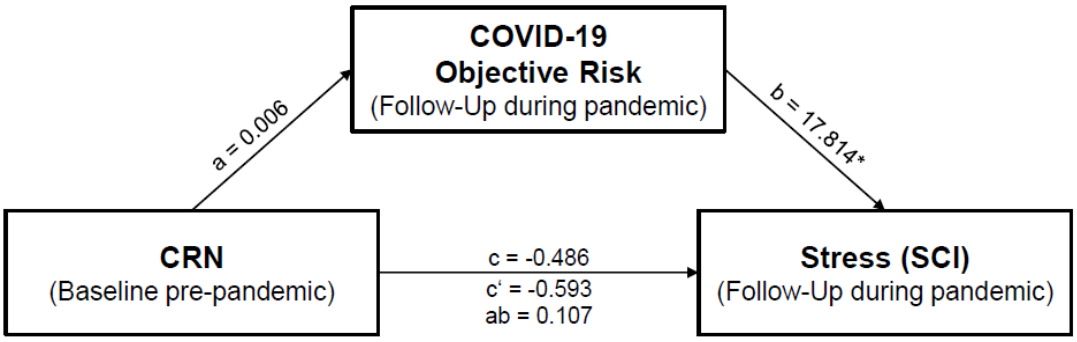
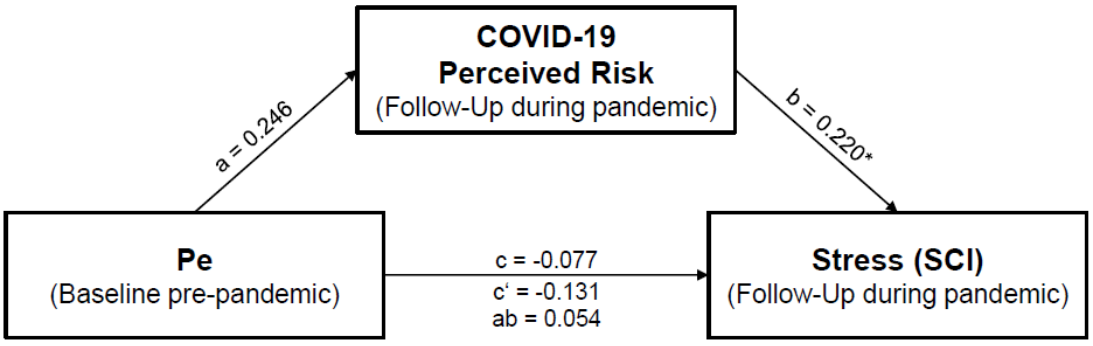
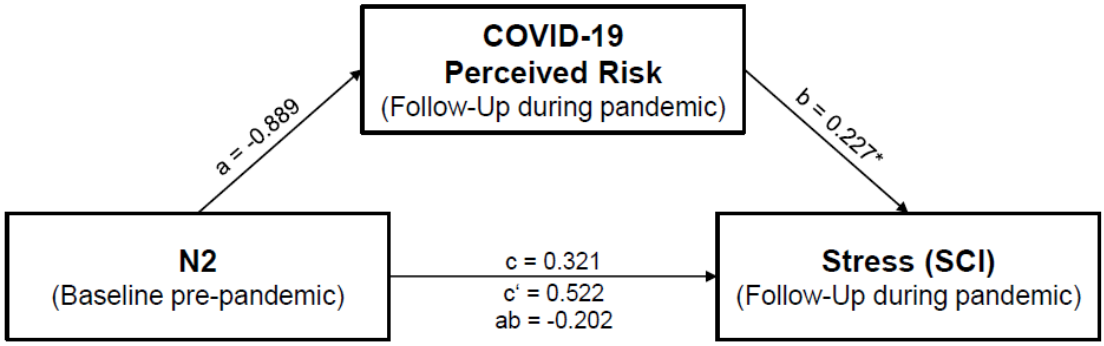


Figure S6. Results of mediation model using the Pe as predictor



Pe measured at Pz for error trials, area 200 - 400 ms, Baseline correction -500 to -300 pre response

Figure S7. Results of mediation model using the N2 as predictor



N2 measured at FCz for correct trials, area 220 - 320 ms, Baseline correction -200 to 0 pre stimulus

Declarations

Erklärung gemäß § 5 (4d) der Promotionsordnung des Instituts für Psychologie der Universität Hamburg vom 20.08.2003

Eidesstattliche Erklärung nach § 9 (1c und 1d) der Promotionsordnung des Instituts für Psychologie der Universität Hamburg vom 20.08.2003

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BEWEGUNGSWISSENSCHAFT
Institut für Bewegungswissenschaft
Institut für Psychologie

Erklärung gemäß *(bitte Zutreffendes ankreuzen)*

☐ **§ 4 (1c) der Promotionsordnung des Instituts für Bewegungswissenschaft der Universität Hamburg vom 18.08.2010**

☐ **§ 5 (4d) der Promotionsordnung des Instituts für Psychologie der Universität Hamburg vom 20.08.2003**

Hiermit erkläre ich,

_____ (Vorname, Nachname),

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Institut für Bewegungswissenschaft
Institut für Psychologie

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☐ **§ 7 (4) der Promotionsordnung des Instituts für Bewegungswissenschaft der Universität Hamburg vom 18.08.2010**

☐ **§ 9 (1c und 1d) der Promotionsordnung des Instituts für Psychologie der Universität Hamburg vom 20.08.2003**

Hiermit erkläre ich an Eides statt,

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