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EEG correlates of attentional control in anxiety disorders: A systematic review of error-related negativity and correct-response negativity findings

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ABSTRACT

Background: Anxiety disorders are highly prevalent and cause substantial personal, social and economic burden. Altered attentional control has been shown to be present across anxiety disorders and is associated with specific changes in brain activity which can be recorded by electroencephalogram (EEG). These include changes in the EEG markers of error-related negativity (ERN) and correct-response negativity (CRN), both believed to reflect response monitoring and attentional control pathophysiology in anxiety. The aim of this review was to systematically assess the research on ERN and CRN in attentional control in individuals with clinical anxiety and healthy controls, across emotional and non-emotional attentional control.

Methods: A comprehensive literature search was conducted for studies published prior to October 22nd, 2020. Details of the protocol for this systematic review were registered on PROSPERO (CRD42019144885).

Results: 66 studies had their data extracted. All 66 studies measured ERN, with 85% finding significantly increased ERN amplitudes associated with clinical anxiety. Only 44 of the extracted studies analysed CRN and only ~20% of these found significant changes in CRN amplitude associated with individuals with clinical anxiety.

Limitations: There were several anxiety disorders that had either limited literature (i.e. specific phobia, separation anxiety disorder or agoraphobia) or nil literature (i.e. selective mutism) available. No extracted studies included samples of older adults (i.e. aged 60+ years), and only six extracted studies included measures of emotional attentional control.

Conclusions: Findings indicate the promising utility of ERN of attentional control as a robust, transdiagnostic trait marker of clinical anxiety.

1. Introduction

Anxiety disorders are a class of mental health disorders primarily characterised by worry, anxiety and/or fear. They include generalised anxiety disorder (GAD), social anxiety disorder (SAD), panic disorder, phobias, obsessive compulsive disorder (OCD) and post-traumatic stress disorder (PTSD) as well as separation anxiety disorder and selective mutism (World Health Organization, 2017). Anxiety disorders are linked to substantial economic (Smit et al., 2006) and personal burden including impairments in health-related quality of life and psychosocial functioning (Revicki et al., 2012). Up to a third of the population will have a diagnosis of an anxiety disorder in their lifetime (Bandelow and Michaelis, 2015). In addition, a substantial proportion of people will be diagnosed with more than one type of anxiety disorder (Kroenke et al., 2007). This has led to the development of transdiagnostic models whereby more focus is given to commonalities across disorders as opposed to their differences (Norton and Paulus, 2017). An advantage of such models includes that they provide a framework to study the underlying mechanisms, rather than the symptoms of disorders, which in turn may lead to more effective preventative and treatment approaches

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(Cuthbert, 2014; Frank et al., 2017).

One potential transdiagnostic symptom in anxiety disorders is altered attentional control, which has been shown to occur across diagnoses and to be related to the onset and maintenance of anxiety (Sharp et al., 2015). Attentional control refers to an individual's ability to pay attention to a wanted or required stimulus whilst ignoring all other stimuli (Astle and Scerif, 2009). Poor attentional control is believed to lead to the phenomenon of hyperactive response monitoring (Simons, 2010) which has been implicated across clinical anxiety symptomology, namely excessive worry and hyperarousal to mild stressors (Bar-Haim et al., 2007; Sharp et al., 2015). A further distinction can be made between non-emotional attentional control (i.e. based on emotionally neutral information) and emotional attentional control (i.e. based on emotionally salient information) (Robinson et al., 2013). Emotional attentional control is particularly pertinent to investigating in clinical anxiety as it encompasses the processes inherent in attentional bias i.e. the tendency to focus on negative, threat-related stimuli (MacLeod et al., 1986). This attentional bias, is particularly overactive in individuals with clinical anxiety, and presents clinically as the aforementioned hyperarousal to mild stressors, which is closely tied to core worry symptomology (Bar-Haim et al., 2007; Sharp et al., 2015). Past studies looking at attentional control and anxiety tend to not make the distinction between non-emotional attentional control and emotional attentional control (Robinson et al., 2013), which may be potentially limiting our understanding of attentional control and its influence in clinical anxiety. This is an issue which has not been previously investigated systematically.

Attentional control is believed to originate from the dorsolateral prefrontal cortex, with regulation by the anterior cingulate cortex which is the source of response or conflict monitoring (Silton et al., 2010). Crucially, the specific brain activity thought to mediate attentional control can be recorded by electroencephalogram (EEG) (Simons, 2010). Event related potentials are an electrophysiological response to a stimulus which can be recorded by EEG (Woodman, 2010). These include error-related negativity (ERN) and correct-response negativity (CRN) (Hochman et al., 2014; Simons, 2010). ERN is a type of event-related potential reflecting a response-locked negative deflection which occurs when a behavioural error is made (Weinberg et al., 2012a). Similarly, CRN, is a smaller 'ERN-like response' that occurs in the presence of a correct response, or in the absence of an error (Simons, 2010). However, whilst the ERN and CRN are considered markers of attentional control, it important to acknowledge that the ERN is also related to other cognitive processes including working memory (Miller et al., 2012), and has been associated with sensitivity to evaluative threat (Weinberg et al., 2016). In the current review, we focus on the role of the ERN specifically in relation to anxiety and attentional control (Davies et al., 2001; Eysenck et al., 2007). By examining markers of attentional control, such as the ERN and CRN, as they relate to clinical anxiety, we may gain a better understanding of the pathophysiology underlying anxiety. Whilst past reviews have explored the relationship between clinical anxiety and the ERN in children and adolescents (Meyer, 2017), or adult populations (Klawohn et al., 2020a) no systematic review to date has looked at the ERN and CRN in clinical anxiety populations *across the lifespan.*

Another commonly related measure is delta ERN (i.e. ΔERN or dERN) which refers to the ERN minus the CRN amplitude, used to define activity unique to error processing (i.e. ERN) by eliminating shared neural activity found in both error and correct responses (Simons, 2010). However, recent work has shown that ERN and CRN tend to be highly correlated, which means the interpretation of the ΔERN as measuring activity unique to error processing is likely problematic (Meyer et al., 2017a). Therefore, to account for this issue an alternative to the ΔERN was suggested- the residualised ERN. The residualised ERN was calculated through a standard regression, with the ERN as the predictor variable and CRN as the outcome variable, by saving the leftover variance. The same process was used vice-versa to calculate the residualised CRN. In a GAD sample, it was shown that residualised ERN only correlated with ERN (not the CRN), and residualised CRN only correlated with CRN (not the ERN), therefore truly defining the unique neural activity of both the ERN and CRN. To date, no previous systematic review has investigated residualised ERN and CRN findings as separate from their ERN and CRN counterparts in the context of anxiety.

There are a number of implications for identifying a neurophysiological marker for attentional control, a transdiagnostic symptom of clinical anxiety. Such markers could act as objective diagnostic measures which would not only help identify individuals with anxiety disorders but may help redefine existing anxiety disorder classifications (Califf, 2018). Additionally, these markers could act as an indicator of the susceptibility of a healthy individual to develop an anxiety disorder (Califf, 2018). Finally, neurophysiological markers of attentional control symptoms may aid in the development of future treatments, by serving as biological markers of improvement. i.e. as a marker of intervention response (Califf, 2018).

Notably, past research investigating neurophysiological markers of anxiety has often looked at individual anxiety disorder symptomology in isolation (e.g. GAD (Abdallah et al., 2013; Qiao et al., 2017), OCD (Gnanavel et al., 2014; Peng et al., 2014), SAD (Gelernter et al., 2004; Phan et al., 2006)) which can limit applicability given the high co-morbidity of anxiety disorders with other anxiety disorders (Kroenke et al., 2007). As such, establishing neurophysiological markers for transdiagnostic symptoms, such as attentional control, could have real-world implications for the diagnosis, management and treatment development of clinical anxiety.

Finally, it is also important to consider for neurophysiological markers are measured in the context of the tasks utilised. For example, in the commonly used Eriksen Flanker Task, Riesel et al., (2019a), found ERN amplitude differences between OCD and healthy control groups only under a condition in which performance feedback emphasised speed, but not in a condition where accuracy was emphasised. Participants in both conditions were given a 2 € bonus for being more accurate or faster as per their experimental condition. Exploring the impact of task-related factors, such as performance feedback and monetary incentive could help to ensure that any neurophysiological markers for transdiagnostic symptoms, are truly representative of said symptoms, and not an artefact of task factors.

Therefore, the aim of this systematic review was to examine attentional control studies comparing differences in ERN and CRN in individuals with anxiety disorders to healthy controls. We additionally explored the emotional versus non-emotional attentional control distinction across these studies and considered task differences related to common attentional control tasks including the use of performance feedback to influence speed and accuracy, including monetary incentive . To the best of our knowledge, this is the first systematic review to: a) explore ERN and CRN of attentional control across the lifespan, b) explore the distinction between non-emotional and emotional attentional control across this population and, c) investigate any potential differences in findings between new and improved measures (i.e. residualised ERN and residualised CRN), compared to older approaches (i.e. ΔERN).

2. Methods

The systematic review was conducted according to PRISMA guidelines (Moher, 2009).

2.1. Search strategy

Details of the protocol for this systematic review were registered on PROSPERO and can be accessed at www.crd.york.ac.uk/PROSPERO/display_record.php?ID=CRD42019144885. A computerised search was conducted up to October 22nd, 2020 using the databases PubMed, PsycINFO, Embase and Scopus. The following search terms were used for all default search fields as per the given database (where * =

truncation): ((((((Attention* control OR Attention* bias OR Cognitive Control OR Endogenous Attention* OR Executive Attention* OR Facilitated Attention* OR Information Process*) AND electroencephalogram) OR EEG OR electroencephalography) AND error related negativity) OR error-related negativity OR ERN OR correct response negativity OR correct-response negativity OR correct response related negativity OR correct response-related negativity OR CRN) AND (Clinical Anxi* OR General* Anxiety OR Social Anxiety OR Social phobia OR Panic OR *Phobia OR Agoraphobia OR Separation Anxiety Disorder OR Selective Mutism OR Obsessive Compulsive Disorder OR OCD OR Post Traumatic Stress Disorder OR PTSD)).

2.2. Study selection

Specifically, studies were included if they matched all three a priori criteria, if: (1) attentional control was measured using one or both of the EEG correlates: ERN and CRN (2) individuals with clinical anxiety were part of the study sample (e.g. generalised anxiety disorder, social anxiety disorder, panic disorder, specific phobia, agoraphobia, separation anxiety disorder, selective mutism, obsessive compulsive disorder, post-traumatic stress disorder); and (3) they were original research published in peer-reviewed journals and written in English. Reviews, systematic reviews and meta-analyses were excluded.

Two reviewers (J.M) and (M.W) independently assessed if studies met the three a priori criteria during the first and second screening phrases. After independent assessment, any differences in study selection were discussed until a consensus was achieved. After the first screening phase based on titles and abstracts 303 papers were excluded. During the second screening phrase articles were screened based on the full-text using the inclusion-exclusion criteria. In this phase if full texts could not be found or if articles were conference abstracts then these papers were also excluded. Figure 1 indicates the process of study selection according to PRISMA guidelines (Moher, 2009). Reasons for exclusion of studies based on the second screening phase included; no clinical anxiety sample was assessed ($n = 47$), conference abstract ($n = 20$), full-text article could not be found ($n = 5$), not published in a peer-reviewed journal ($n = 5$), no attentional control task ($n = 4$), clinical anxiety sample previously published and already included in this systematic review ($n = 7$), erratum/correction only ($n = 2$), no EEG measure of ERN or CRN was used ($n = 2$), full-text not available in English language ($n = 1$) and the article was only available as a pre-print, where not enough information was provided to determine if criteria was met ($n = 1$).

2.3. Data extraction

Data extraction included: author, year, study design, mean (SD) age, sample size, percentage of females, type(s) of clinical anxiety in sample, type of EEG correlate measure(s) used, attentional control task(s) used (and whether they primarily measure emotional or non-emotional attentional control), main findings and conclusions. Data was only extracted from eligible studies.

2.4. Assessment of risk of bias

Two reviewers (J.M) and (M.W) independently assessed the methodological quality of eligible studies using the appropriate SIGN methodological checklist, based on the study design (Healthcare Improvement Scotland, SIGN, 2012). After independent assessment, any differences in ratings were discussed until a consensus was achieved. When a checklist was not available for a study design i.e. for correlational studies, the reviewers individually assessed the studies based on the general criteria found throughout the differing checklists, then discussed their ratings until a consensus was achieved. The SIGN methodological checklists consists of 15 or more items concerning a study's internal validity and overall assessment where items are rated as: 'yes', 'no' and for some items also 'Can't say' and/or 'Unsure'. The overall methodological quality of a study was rated as either: "High quality (++): Majority of criteria met. Little or no risk of bias. Results unlikely to be changed by further research OR Acceptable (+): Most criteria met.

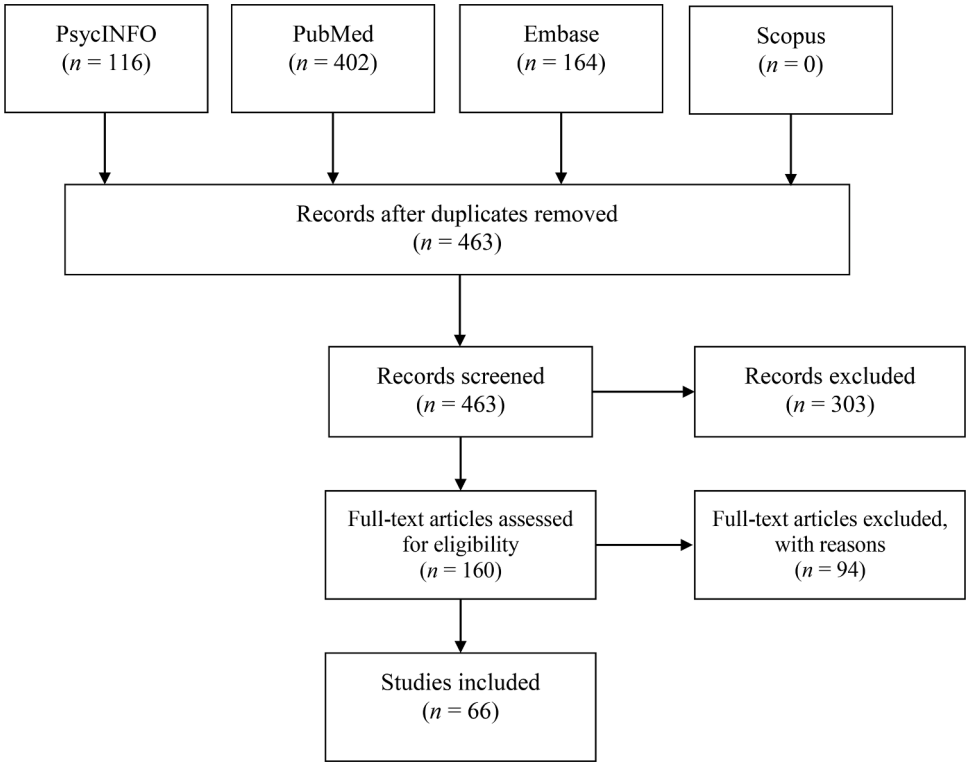

Fig. 1. Systematic study selection highlighted in a PRISMA flowchart.

Some flaws in the study with an associated risk of bias, Conclusions may change in the light of further studies. OR Low quality (0): Either most criteria not met, or significant flaws relating to key aspects of study design. Conclusions likely to change in the light of further studies." (Healthcare Improvement Scotland, SIGN, 2012). In SIGN guidelines it is also noted that any study that does not address the possibility of confounding variables should be rejected (Healthcare Improvement Scotland, SIGN, 2012).

3. Results

3.1. Study selection

A total of 682 articles were obtained after the searching across the four databases. After removing duplicates 463 unique papers remained. After the two aforementioned screening phrases, 66 papers remained and had their data extracted.

3.2. Study characteristics

Of the 66 extracted studies, the sample sizes ranged from 7 – 117 for the clinical anxiety samples, and from 9 – 319 for healthy controls, with ~2500 confirmed participants with clinical anxiety and ~1600 healthy controls. Note that these are approximate numbers as: a) not all comparison groups were 'true' healthy controls i.e. studies did not confirm that they had no psychiatric diagnoses and may just confirm that they do not have the disorder of interest e.g. a non-PTSD group; and, b) clinical anxiety samples can be part of general psychopathology samples. Where possible, exact numbers of clinical anxiety sample size have been included, although this data was not always available or was not clear. For example, do 10 individual people have an anxiety diagnosis or are there 10 comorbid anxiety disorder diagnoses throughout a sample? The latter could mean that there were less than 10 participants with an anxiety disorder if there were individuals with two or more anxiety disorder diagnoses (which is not uncommon).

The majority of the studies, (i.e. 51 of 66) used a case-control design. Of the total remaining studies; five used a cohort study design, three used a controlled trial design and seven used a correlational design. Table 1 shows the main findings extracted from included studies and risk of bias ratings for included studies. See Supplementary Table 1 for the data extracted from included studies related to study characteristics i.e. study design, mean (SD) age, sample size, percentage of females, type(s) of clinical anxiety in sample, type of EEG correlate measure(s) used and attentional control task(s) used (and whether they primarily measure emotional or non-emotional attentional control).

3.3. Age

The mean age ranged from 6.11 – 46.5 years for clinical anxiety samples and 6.06 – 45.4 years for healthy controls. Two studies did not report mean age of their samples, one study did not report the mean age of their healthy control group and five studies reported the mean age of the total sample, but not the proportion of the sample with clinical anxiety (Supplementary Table 1).

3.4. Gender

Most studies contained both male and female participants with only three studies with males only and three studies with females only (Supplementary Table 1). Five studies did not report the gender of their samples, and five studies reported the gender of the total sample, but not the proportion of the sample with clinical anxiety (Supplementary Table 1).

Table 1
Data extracted from included studies: Main findings, methodological quality.

Citation	Main Findings (Clinical Anxiety population compared to healthy controls) * Significant findings unless stated otherwise.	Methodological Quality
(Agam et al., 2014a)	No significant difference in ΔERN, ERN or CRN amplitude.	(+)
(Agam et al., 2014b)	No significant difference in ΔERN amplitude. ERN and CRN alone not analysed.	(+)
(Baldwin et al., 2015)	Within-person variance was higher than between-group variance for all groups. Clinical Anxiety and Depression groups require more trials to have a dependable ERN/CRN amplitude measure than HC. i.e. Clinical Anxiety and Depression groups have greater within-person variance than HC.	(+)
(Carmi et al., 2018)	↑ ΔERN amplitude in high frequency deep transcranial magnetic stimulation group and ↓ ΔERN amplitude in sham group following successful treatment. ERN and CRN alone not analysed.	(++)
(Carmi et al., 2019)	↑ ΔERN amplitude in OCD compared to HC group. No significant difference between OCD and unaffected siblings' group. ERN and CRN alone not analysed.	(+)
(Carrasco et al., 2013a)	↑ ERN amplitude in OCD compared to other groups.	(+)
(Carrasco et al., 2013b)	↑ ERN amplitude in OCD and GAD/SAD groups not HC, with trend level effect of error trial number. Amplitude not significantly different between OCD and GAD/SAD groups.	(+)
(Chong et al., 2020)	↑ ERN and residualised ERN amplitude in sub-set of clinical anxiety group (i.e. those with GAD, OCD and/or SAD) compared to participants without an anxiety disorder diagnosis. Non-significant difference if whole clinical anxiety group compared to non-diagnosed group. CRN between groups not analysed.	(+)
(Clemans et al., 2012)	Less negative time-frequency ERN amplitude.	(+)
(Crane et al., 2018)	PTSD symptoms did not significantly associate with residualised ERN or ΔERN amplitude. CRN or ERN alone not analysed.	(+)
(Denefrio et al., 2019)	No significant group differences in in ERN or ΔERN amplitude. CRN between groups not analysed.	(+)
(Endrass et al., 2010)	↑ ERN and CRN amplitude in standard condition. ↑ ERN and CRN amplitude between reward and punishment conditions in HC only.	(+)
(Endrass et al., 2008)	↑ ERN amplitude	(+)
(Endrass et al., 2014)	↑ ERN amplitude in OCD and SAD than HC. ↑ CRN amplitude in SAD compared to HC.	(+)
(Gehring et al., 2000)	↑ ERN and ΔERN amplitude in OCD compared to HC group. CRN between groups not analysed.	(+)
(Gorka and Phan, 2017)	↑ anxiety symptoms associated with ↑ residualised ERN amplitude. ERN and CRN alone not analysed.	(+)

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Table 1 (*continued*)

Citation	Main Findings (Clinical Anxiety population compared to healthy controls) * Significant findings unless stated otherwise.	Methodological Quality
(Gorka et al., 2016)	↑ ERN amplitude in PTSD/alcohol use disorder co-morbid group compared to either disorder alone.	(+)
(Gorka et al., 2018)	↑ residualised ERN and ΔERN amplitude pre- to post- successful Selective Serotonin Reuptake Inhibitors treatment for clinical anxiety group only, no significant difference pre- to post- Cognitive Behavioural Therapy for any group. Increased residualised ERN and ΔERN amplitude at baseline significantly associated with greater reduction in fear-based anxiety symptoms for cognitive-behavioural therapy group only. CRN and ERN results alone not analysed.	(+)
(Gorka et al., 2017)	↑ Residualised ERN amplitude associated with ↑ fear-based anxiety symptoms. ΔERN, ERN, CRN amplitude alone not analysed.	(+)
(Grützmann et al., 2016)	↑ ERN amplitude	(+)
(Hajcak et al., 2008)	↑ ERN amplitude.	(+)
(Hanna et al., 2012)	↑ ERN amplitude in OCD compared to HC group. ↑ ERN amplitude in non-tic OCD compared to tic-OCD and HC.	(+)
(Hanna et al., 2016)	↑ ERN amplitude.	(+)
(Hanna et al., 2018)	↑ ERN amplitude in OCD compared to HC and OCD/ depression patients compared to HC.	(+)
(Hanna et al., 2020)	↑ ERN and residualised ERN amplitude in GAD compared to HC group. ↑ ERN and residualised ERN amplitude in Other Clinical Anxiety group compared to HC group.	(+)
(Hum et al., 2013)	↑ ERN, CRN amplitudes. In the Clinical Anxiety group only, no differences between neutral and emotional conditions.	(+)
(Johannes et al., 2001)	↑ ΔERN and ERN amplitude. ↑ ΔERN and ERN latency. CRN between groups not analysed.	(+)
(Khan et al., 2018)	↑ Residualised ERN amplitude with greater combat exposure, even when adjusting for broad anxiety and PTSD. The association between combat exposure and residualised ERN was not moderated by PTSD symptom severity.	(+)
(Klawohn et al., 2015)	↑ ERN amplitude in flanker task.	(+)
(Klawohn et al., 2020b)	↑ ΔERN and ERN amplitude in OCD group compared to both HC groups pre- attentional bias modification training. ↓ ΔERN and ERN amplitude post- training in OCD group only.	(+)
(Klawohn et al., 2014)	↑ ERN, CRN amplitude.	(+)
(Kujawa et al., 2016)	↑ ΔERN in SAD relative to HC.	(+)
(Ladouceur et al., 2018)	↑ Residualised ERN amplitude. ERN and CRN amplitude alone not analysed.	(+)
(Ladouceur et al., 2006)	↑ ERN. In clinical anxiety group ERN localised to anterior cingulate cortex.	(+)
(Lieberman et al., 2017)	↓ ΔERN associated with increased hyperarousal symptoms. ERN and CRN alone not analysed.	(+)
(Liu et al., 2017)	Experiment 1: N/A Experiment 2: ↑ ERN amplitude and latency	(+)
(Liu et al., 2014)	↑ ERN and dERN amplitude. CRN alone not analysed.	(+)
(Mathews et al., 2016)	↑ ERN amplitude in OCD compared to HC.	(+)
(McDermott et al., 2009)	Teenagers with high behavioural inhibition as children had ↑ ERN amplitude as teenagers, which led to higher risk for clinical anxiety.	(+)
(Meyer et al., 2013)	↑ ΔERN amplitude	(+)
(Meyer et al., 2017b)	↑ΔERN amplitude in clinical anxiety subset compared to non-clinical anxiety (remaining sample).	(+)
(Meyer et al., 2019)	↑ Residualised ERN amplitude in parent condition in children with clinical anxiety than HC. No difference in experimental condition. ERN and CRN amplitude alone not analysed.	(+)
(Meyer et al., 2018)	↑ ΔERN at time 1, increased likelihood of GAD at time 2. ↑ social anxiety symptoms increased the likelihood of GAD at time 2, more so if combined with ↑Δ ERN.	(+)
(Muir et al., 2020)	Residualised ERN or ERN did not significantly predict any diagnostic status. CRN alone not used in analyses.	(+)
(Nawani et al., 2018)	↑ ERN amplitude.	(+)
(Rabinak et al., 2013)	No significant difference between PTSD and HC in ΔERN, ERN or CRN amplitude.	(+)
(Riesel et al., 2019b)	↑ ERN (all types) and ΔERN amplitude in OCD and unaffected relatives compared to HC, but no difference between unaffected relatives and OCD group. ↑ ERN (all types) and ΔERN amplitude in affected relatives with family history of anxiety disorders.	(++)
(Riesel et al., 2015)	↑ ERN and CRN amplitudes pre- and post- Cognitive Behavioural Therapy treatment in OCD compared to HC. Whilst symptoms improved post-treatment there was no significant correlation between symptoms and ERN or CRN.	(+)
(Riesel et al., 2011)	↑ ERN amplitude in OCD group and unaffected relatives compared to HC.	(+)
(Riesel et al., 2019a)	↑ ERN amplitude in OCD compared to HC under speed instructions, but no significant differences between groups under accuracy instructions.	(+)
(Riesel et al., 2014)	↑ ERN and CRN amplitude.	(+)
(Roh et al., 2016)	↑ ERN and CRN amplitude overall. Only OCD group had ↑ ERN amplitude in fearful versus neutral condition.	(+)
(Roh et al., 2017)	↑ ERN and CRN amplitude across both conditions. In OCD group ↑ ERN amplitude in OCD provocation compared to neutral condition.	(+)
(Ruchsow et al., 2005)	↑ ERN and ΔERN amplitude.	(+)

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Table 1 (*continued*)

Citation	Main Findings (Clinical Anxiety population compared to healthy controls) * Significant findings unless stated otherwise.	Methodological Quality
(Schellekens et al., 2010)	↑ ERN amplitude in the alcohol-dependent Clinical Anxiety group compared to group with alcohol-dependence and no anxiety disorders or HC group. CRN not analysed between groups.	(+)
(Seow et al., 2020)	No significant association between any ERN or CRN measure and any anxiety symptoms.	(+)
(Stern et al., 2010)	↑ ERN. ↑ ERN amplitude associated with severity of anxiety symptoms in HC only.	(+)
(Suor et al., 2020)	Anxiety symptoms alone did not significantly predict residualised ERN amplitude. However, ↑ residualised ERN amplitude was associated with youth with both less externalising problems and greater anxiety symptoms. ERN and CRN amplitude alone not analysed.	(+)
(Swick et al., 2015)	No significant differences in ERN, ΔERN or CRN between groups.	(+)
(Valt et al., 2018)	↑ ERN overall. No differences in neutral or emotional condition.	(+)
(Voegler et al., 2018)	No significant ERN/CRN differences.	(+)
(Weinberg et al., 2012b)	↑ ΔERN amplitude and increased difference between error and correct trials than HC or co-morbid groups.	(+)
(Weinberg et al., 2015)	↑ ERN amplitude in GAD but only with no co-morbid depression compared to HC. ΔERN and CRN alone not analysed between groups.	(+)
(Weinberg et al., 2010)	↑ ERN and ΔERN amplitude. ↑ ERN associated with increased anxiety.	(+)
(Xiao et al., 2011)	↑ ΔERN difference in OCD patients only compared to HC. ↑ ERN amplitude in both OCD and GAD compared to HC, which associated with HAM-A scores.	(+)
(Zambrano-Vazquez and Allen, 2014)	Only worry group had ↑ dERN amplitude relative to HC. Worry correlated with dERN amplitude. ERN and CRN alone not analysed.	(+)

Note. HC = Healthy Controls, GAD = Generalised Anxiety Disorder, SAD = Social Anxiety Disorder, PTSD = Post-Traumatic Stress Disorder, OCD = Obsessive Compulsive Disorder. ERN = Error-Related negativity, CRN = Correct-Response negativity, ΔERN or dERN = delta ERN.

3.5. Anxiety disorders: Primary classifications

The following studies were classified by the primary inclusion criteria for the sample group. This means that some studies were counted in two classifications if they compared two groups with clinical anxiety with different primary classifications. Of these studies; five had participants with a primary diagnosis of PTSD, 35 with OCD, nine with GAD, one with panic disorder and three with SAD (Supplementary Table 1). Additionally 20 studies had mixed clinical samples i.e. they did not analyse some or all of their data by separating patients with anxiety disorder by group (Baldwin et al., 2015; Carrasco et al., 2013b; Chong et al., 2020; Gorka et al., 2018, 2017; Gorka and Phan, 2017; Hanna et al., 2020; Hum et al., 2013; Khan et al., 2018; Ladouceur et al., 2018, 2006; Lieberman et al., 2017; McDermott et al., 2009; Meyer et al., 2019, 2017b, 2013; Muir et al., 2020; Schellekens et al., 2010; Seow et al., 2020; Suor et al., 2020). None of the extracted studies had any individuals with selective mutism.

3.6. Anxiety disorders: Co-morbidities

Of the OCD samples 12 had at least one co-morbid anxiety disorder in their samples (Carrasco et al., 2013a; Endrass et al., 2014, 2010, 2008; Gehring et al., 2000; Klawohn et al., 2020b; Liu et al., 2014; Riesel et al., 2019a, 2019b, 2014; Weinberg et al., 2015; Zambrano-Vazquez and Allen, 2014), five for GAD (Denefrio et al., 2019; Hanna et al., 2020; Kujawa et al., 2016; Weinberg et al., 2015, 2012b), one for panic disorder (Valt et al., 2018) and three for SAD (Endrass et al., 2014; Kujawa et al., 2016; Voegler et al., 2018). There were also no studies with separation anxiety disorder, specific phobia or agoraphobia samples alone. However, individuals with separation anxiety disorder were found in 10 of the mixed clinical samples (Chong et al., 2020; Hanna et al., 2020; Hum et al., 2013; Ladouceur et al., 2018, 2006; McDermott et al., 2009; Meyer et al., 2019, 2017b, 2013; Suor et al., 2020), composing between 7% to30% (or to 36% if accounting for current subthreshold separation anxiety disorder (Meyer et al., 2019)) of the study sample as well as in an additional study (Carrasco et al., 2013a) as a co-morbidity to another anxiety disorder. Similarly, there were no studies with specific phobia samples alone. However individuals with specific phobia were noted in seven of the mixed clinical samples studies (Hanna et al., 2020; ; Ladouceur et al., 2018, 2006; Meyer et al., 2019, 2017b, 2013; Suor et al., 2020) composing 4% to 46% of their study samples, respectively. Specific phobia was also noted in 12 studies as a co-morbidity to another anxiety disorder (Carrasco et al., 2013a; Denefrio et al., 2019; Endrass et al., 2014; Gehring et al., 2000; Gorka and Phan, 2017; Klawohn et al., 2020b; Liu et al., 2014; Riesel et al., 2019a, 2019b, 2014; Weinberg et al., 2015; Zambrano-Vazquez and Allen, 2014). Additionally, there were no studies with agoraphobia samples alone. However, individuals with agoraphobia were noted in five of the mixed clinical samples (Chong et al., 2020; Hanna et al., 2020; Meyer et al., 2013; Schellekens et al., 2010; Seow et al., 2020) composing 3% to-15% of their study samples, respectively. Agoraphobia was also noted in eight studies as a co-morbidity to another anxiety disorder (Carrasco et al., 2013a; Gehring et al., 2000; Klawohn et al., 2020b; Riesel et al., 2019a, 2019b; Valt et al., 2018; Weinberg et al., 2015; Zambrano-Vazquez and Allen, 2014)

3.7. Risk of bias

Of the 66 studies included, 2 were rated as high quality (++) (Carmi et al., 2018; Riesel, et al., 2019b), with the remaining 64 rated as acceptable (+), (see Table 1).

3.8. Error-related ngativity (ERN) findings

Below, all ERN study findings are first summarised (i.e. across diagnoses), followed by the reporting of findings by primary diagnosis type, ending with findings from mixed clinical samples.

3.8.1. All studies

All 66 studies measured ERN, including, residualised ERN and ΔERN/dERN. All studies were rated with an acceptable level of bias, except two which were rated as high quality (Table 1). Of the 66 studies which measured standard ERN, of these 13 were not analysed in their respective study (i.e. they were only used to calculate ΔERN/dERN or residualised ERN) (Supplementary Table 1). Of the 32 studies which measured ΔERN/dERN, of these two were not analysed in their respective study (Supplementary Table 1). 12 studies measured residualised ERN, all of which were analysed (Supplementary Table 1). Of all studies utilising any ERN measures; five had participants with a primary diagnosis of PTSD, 35 with OCD, nine with GAD, one with panic disorder, three with SAD and 20 had mixed clinical samples (Supplementary Table 1). Of these studies, 60 used non-emotional attentional

control tasks, and of these 51 used an Eriksen Flanker Task (Supplementary Table 1). In the Eriksen Flanker Task (Eriksen and Eriksen, 1974), the congruent condition refers to when the 'flankers' (i.e. stimuli surrounding the target), are the same as the target letter, image, symbol etc e.g. "< < < < < < <". The incongruent condition is where the flankers differ from the target e.g. "< < < > < < <". Of the remaining non-emotional task studies, two used a Go/No-Go task, three used the Stroop task, two used an anti-saccade paradigm, one used a continuous performance task, one used a Simon Task, and another used both the visual search task as well as the Eriksen Flanker task (Supplementary Table 1). The remaining six studies used emotional attentional control tasks. Of these; three used an Eriksen Flanker task, two used a Go/No-Go task and one used a Response-choice task (Supplementary Table 1). Of the 54 studies which utilised any Eriksen Flanker Task, 36 utilised performance feedback which emphasised speed and accuracy, 12 utilised no feedback at all, four used monetary incentive and/or punishment, one used feedback to emphasise speed only and one noted that visual feedback was used, but did not specify what this was (Supplementary Table 1). No studies used both emotional and non-emotional attentional control tasks within the one study.

In total 57 of the 66 studies utilising any ERN measures found significant differences in ERN measures (Table 1). Five of these 57 studies used emotional attentional control tasks (Hum et al., 2013; Meyer et al., 2019; Roh et al., 2017, 2016; Valt et al., 2018), with the remaining studies using a non-emotional task.

Of the 53 studies which measured and analysed the standard ERN, 41 of these found significant differences (Table 1). Of the 12 studies measuring residualised ERN, nine found significant differences (Table 1). Of the 30 studies which analysed and measured ΔERN/dERN, 18 of these found significant differences (Table 1).

3.8.2. PTSD studies

Of the five PTSD studies all used a case-control design, except one which used a cohort design; however, all studies used a non-emotional Eriksen Flanker task (Supplementary Table 1). All studies had an acceptable level of bias and two of these studies found significant group differences in ERN measures for PTSD (Table 1). Of these; one study found increased ERN amplitude in the co-morbid PTSD and alcohol use disorder group than the healthy control group, or PTSD or alcohol use disorder groups alone, where performance feedback which emphasised speed and accuracy was utilised in the task (Gorka et al., 2016). The mean average age of PTSD groups was approximately 10 years older than the three studies that did not find significant difference in ERN amplitude (Supplementary Table 1). The other study found less negative time-frequency ERN amplitude, for both congruent and incongruent trials for PTSD versus healthy control groups (Clemans et al., 2012), however the mean age of the sample was not reported and feedback was not utilised during the Eriksen Flanker task (Supplementary Table 1).

3.8.3. OCD studies

Of the 35 OCD studies all were case-control studies except two which were controlled trials (Supplementary Table 1). All studies had acceptable levels of bias, except two which was rated as high quality (Table 1). All studies used a non-emotional attentional control task, except two which used emotional attentional control tasks, both using an emotional Eriksen Flanker task (Supplementary Table 1). All studies except three, found significant differences in ERN family measures for OCD (Table 1). Of these 32 studies with significant findings, 27 used an Eriksen Flanker task (Supplementary Table 1). Of these, 18 utilised performance feedback which emphasised speed and accuracy, one used feedback which emphasised speed only, three utilised monetary incentive and five did not use any performance feedback (Supplementary Table 1).

One of the controlled trials found that pre- attentional bias training increased ΔERN and ERN amplitude were found in the OCD compared to the healthy control groups, but post-training only the OCD group had reduced ΔERN and ERN amplitude (Klawohn et al., 2020b). However, the other OCD study with a controlled trial design found an increase in ΔERN amplitude when high-frequency deep transcranial magnetic stimulation was used and a decrease when sham stimulation was used following successful treatment (Carmi et al., 2018). On the other hand, another study found increased ERN amplitude in the OCD compared to the healthy control group which persisted from pre- to post- cognitive behavioural therapy treatment (Riesel et al., 2015).

In total, 20 studies found an increased ERN amplitude in OCD compared to the healthy control group (one in the standard condition (Endrass et al., 2010), and one only in the speed, but not accuracy condition (Riesel et al., 2019a)) (Table 1). Additionally, four studies found the same result using ΔERN/dERN measures (Table 1). Two different studies found an increase in both ERN amplitude and latency in the OCD compared to the healthy control groups (Johannes et al., 2001; Liu et al., 2017), with Johannes et al., (2001), finding the same results with ΔERN amplitude. Another study found an increase in ERN amplitude in the OCD compared to the healthy control group and an increase in ERN amplitude in the non-tic related OCD compared to tic-related OCD and healthy control groups (Hanna et al., 2012). Similarly, a different OCD study found that only the 'worry' OCD-subgroup had increased dERN amplitude relative to the healthy control group (Zambrano-Vazquez and Allen, 2014). Moreover, two studies found an increase in ERN amplitude in all types measured (including ΔERN) was found in the OCD and unaffected relatives groups compared to healthy controls, but no differences were found between unaffected relatives and OCD group (Carmi et al., 2019; Riesel et al., 2019b) However, the same increase in ERN amplitude of all types measured was found in affected relatives with a family history of anxiety disorders (Riesel et al., 2019b).

Of the two studies that used emotional attentional control tasks, one found an increase in ERN amplitude across both fearful and neutral conditions in the OCD compared to the healthy control group (Roh et al., 2016). However, only the OCD group had an increase in ERN amplitude in the fearful compared to the neutral condition. Similarly, the other study examining emotional attentional control also found an increase in ERN amplitude across both OCD-provocation and neutral conditions in the OCD compared to the healthy control group (Roh et al., 2017), where only the OCD group had an increase in ERN amplitude in the OCD-provocation compared to the neutral condition.

3.8.4. GAD studies

Of the nine GAD studies (Supplementary Table 1), all had acceptable levels of bias (Table 1). All studies had a case-control design, except one with a cohort study design (Meyer et al., 2018). All studies used a non-emotional attentional control task except one which used an emotional attentional control task (Denefrio et al., 2019). All studies found significant group differences in ERN, except three (Denefrio et al., 2019; Kujawa et al., 2016; Muir et al., 2020). Of the remaining six studies with significant ERN differences; in the cohort study there was an increase in ΔERN amplitude at time 1 with an increased likelihood of GAD at time 2 and an increase in social anxiety symptoms increased the likelihood of GAD at time 2, more so if combined with an increase in ΔERN amplitude (Meyer et al., 2018). Five studies found an increase in ERN amplitude measures in GAD compared to healthy control groups (Hanna et al., 2020; Weinberg et al., 2015, 2012b, 2010; Xiao et al., 2011). This included; standard ERN (Hanna et al., 2020; Weinberg et al., 2015, 2010; Xiao et al., 2011), ΔERN (Weinberg et al., 2012b, 2010), and residualised ERN (Hanna et al., 2020). All studies utilised a non-emotional Eriksen Flanker task with performance feedback to emphasise speed and accuracy.

3.8.5. SAD studies

Of the three case-control SAD studies, all used a non-emotional attentional control task (Table 1). All studies were rated with acceptable levels of bias and two of the three studies found a significant group differences in ERN measures (Table 1). Of these, one study one found increased ERN amplitude in SAD compared to healthy control groups

(Endrass et al., 2014). The other study found increased ΔERN in SAD relative to healthy controls (Kujawa et al., 2016). Both studies used a non-emotional Eriksen Flanker task which utilised performance feedback to emphasise speed and accuracy.

3.8.6. Panic disorder studies

The one Panic Disorder study (Valt et al., 2018), used a case-control design and had an acceptable level of bias. It utilised an emotional attentional control task. The study found an increased ERN amplitude in panic disorder compared to healthy control groups.

3.8.7. Studies with mixed clinical anxiety samples

Of the 20 mixed clinical samples studies, nine were case-control studies, seven were correlational studies, three were cohort studies and one was a controlled trial. All of these studies had an acceptable level of bias (Table 1). All studies except two (Muir et al., 2020; Seow et al., 2020) found significant changes in ERN measures (Table 1). All these studies finding significant changes used non-emotional attentional control tasks (Supplementary Table 1), except two which both used No/No-Go emotional attentional control tasks (Hum et al., 2013; Meyer et al., 2019). Of the 16 studies which showed significant changes in ERN family measures (and also used an Eriksen Flanker task), 11 utilised performance feedback which emphasised speed and accuracy, three provided no feedback, one used visual feedback, but did not specify what it was and when it was used, and another study used a monetary incentive if participants performed "extremely well", which all participants received no matter their performance (Supplementary Table 1).

Of the six correlational studies with significant findings, one found an increase in residualised ERN amplitude was associated with an increase in anxiety symptoms (Gorka and Phan, 2017) and, another study found increased residualised ERN amplitude was associated with increased fear-based anxiety symptoms (Gorka et al., 2017). However, another correlational study found a decreased ΔERN associated with increased hyperarousal symptoms (Lieberman et al., 2017). A different correlational study found that whilst anxiety symptoms alone did not significantly predict ERN amplitude, increased residualised ERN amplitude was found in youth with both reduced externalising problems and increased anxiety symptoms (Suor et al., 2020). Another correlational study found an increase in ERN amplitude and residualised ERN amplitude in sub-set of the clinical anxiety group i.e. individuals with GAD and/or OCD and/or SAD, compared to those without a clinical anxiety diagnosis (Chong et al., 2020). However, these significant differences did not remain when the whole clinical anxiety group was taken into account (i.e. including individuals with separation anxiety disorder, and/or panic disorder and/or specific phobia and/or an anxiety disorder not otherwise specified). Finally, the remaining correlational study found an increase in residualised ERN amplitude with greater combat exposure, after adjusting for broad anxiety and PTSD (Khan et al., 2018).

Three studies found increased ERN amplitude in clinical anxiety, (Hanna et al., 2020; Ladouceur et al., 2006; Schellekens et al., 2010), of these, two studies also found increased residualised ERN in clinical anxiety as well (Hanna et al., 2020; Ladouceur et al., 2006). Other studies found the same increased amplitude with residualised ERN alone (Ladouceur et al., 2018) and with ΔERN (Meyer et al., 2017b;2013). One case-control study found increased ERN amplitude in clinical anxiety (co-morbid GAD and SAD group) compared to the healthy control group with a trend level effect of error trial number (Carrasco et al., 2013b). A different study found an increase in ERN amplitude in clinical anxiety versus the healthy control group, and in the clinical anxiety group only, no differences were found between neutral and emotional conditions (Hum et al., 2013). Another case-control study found that increased residualised ERN and ΔERN amplitude pre- to post- successful selective serotonin reuptake inhibitors treatment for clinical anxiety group only, with no significant difference in residualised ERN and ΔERN amplitude pre- to post- Cognitive Behavioural Therapy for any group (Gorka et al., 2018). It was also found that increased residualised ERN and ΔERN amplitude at baseline significantly associated with greater reduction in fear-based anxiety symptoms for the cognitive-behavioural therapy group only. A different study found that within-person variance in ERN amplitude was higher than between-group variance for all groups, but that the clinical anxiety group requires more trials to have a dependable ERN amplitude measure than the healthy control group (Baldwin et al., 2015). One of the cohort studies found that teenagers with high behavioural inhibition as children had increased ERN amplitude as teenagers, which was related to higher risk for clinical anxiety (McDermott et al., 2009). The final study including a mixed clinical sample found an increased residualised ERN amplitude when a parent was present during testing in children with clinical anxiety compared to healthy controls, however, no differences were found when the experimenter alone (rather than the parent) was present (Meyer et al., 2019).

3.9. Correct-response negativity (CRN) findings

Below, all CRN study findings are first summarised (i.e. across diagnoses), followed by the reporting of findings by primary diagnosis type, ending with findings from mixed clinical samples.

3.9.1. All studies

In total, 62 studies measured CRN, including residualised CRN and of these only 44 analysed their respective CRN measure (Supplementary Table 1). Of the 62 studies measuring CRN, 55 derived CRN across all correct trial types (i.e. both congruent and incongruent) (Supplementary Table 1). Only two studies specified that they used the incongruent condition only, although the only results reported in said studies were the ΔERN, with the ERN and CRN alone not analysed (Supplementary Table 1). Three studies did not specify how they measured CRN in respect to using congruent/incongruent conditions (Supplementary Table 1). Another study used a subset of correct responses matched to error responses on the basis of reaction time, but also did not specify using congruent/incongruent conditions (Supplementary Table 1). And finally, one study used a mathematically determined combination of response-locked and stimulus-locked components (Supplementary Table 1).

21 studies did not analyse standard CRN in their respective study (i.e. CRN was only used to calculate ΔERN/dERN or residualised CRN) (Supplementary Table 1). Four studies measured residualised CRN, all of which were analysed (Supplementary Table 1). Altogether of the 44 studies which analysed any measure of CRN, all were rated with an acceptable level of bias, except one which was rated as high quality (Table 1). All studies had a case-control design except three which had a cohort study design, four which used a correlational study design and one which was a controlled trial (Supplementary Table 1). Of these studies, three had participants with a primary diagnosis of PTSD, 25 with OCD, six with GAD, three with SAD and 10 had mixed clinical samples (Supplementary Table 1). No studies with panic disorder used CRN measures. All studies used non-emotional attentional control tasks, except three which used emotional attentional control tasks (two of which used Eriksen Flanker Tasks, one which used a Go/No-Go task) (Supplementary Table 1). All studies using non-emotional attentional control tasks used Eriksen Flanker tasks except two which used Go/No-Go tasks, and one which used a Simon Task (Supplementary Table 1). In total nine of the 44 studies analysing CRN measures found significant differences in CRN measures (Table 1). No studies which found significant differences used residualised CRN measures. All used non-emotional Eriksen Flanker tasks, except two which used an emotional Eriksen Flanker task, and one with an emotional Go/No-Go task (Supplementary Table 1). Of the eight studies with significant CRN findings which utilised an Eriksen Flanker Task, five utilised performance feedback which emphasised speed and accuracy, two provided no feedback and one included a punishment condition where slow or incorrect response were punished with a loss of 20 cents (Supplementary Table 1). No studies used both emotional and non-emotional attentional control

tasks within the one study.

3.9.2. PTSD studies

Of the three case-control PTSD studies, all used a non-emotional Eriksen Flanker task (Supplementary Table 1). All the PTSD studies had an acceptable level of bias and also had no significant differences in CRN measures (Table 1).

3.9.3. OCD studies

Of the 25 OCD studies (Supplementary Table 1), all had an acceptable level of bias, except one which was rated as high quality (Table 1). All studies had a case-control design, except one which was a controlled trial (Supplementary Table 1). All studies used non-emotional Eriksen Flanker tasks, except two which used an emotional Eriksen Flanker task (Roh et al., 2017, 2016). Of all OCD studies, only six found a significant group difference in CRN measures and of these, all found an increase in CRN amplitude in the OCD compared to healthy control group (Table 1), including both studies which used an emotional Eriksen Flanker task.

3.9.4. GAD studies

Of the six studies with GAD samples, all used a non-emotional Eriksen Flanker task with a case-control study design, except one study which used a cohort study design (Supplementary Table 1). All studies had an acceptable level of bias, but none found any significant group differences in CRN measures (Table 1).

3.9.5. SAD studies

Of the three SAD case-control studies, all used non-emotional attentional control tasks (two with the Eriksen Flanker task and one with a Go/No-Go task) (Supplementary Table 1). All studies were rated with an acceptable level of bias, but only one of the studies found significant differences in CRN measures (Table 1). This study found an increase in CRN amplitude in SAD compared to healthy control groups (Endrass et al., 2014) and utilised a non-emotional Eriksen Flanker task with performance feedback which emphasised speed and accuracy.

3.9.6. Studies with mixed clinical anxiety samples

Of the 10 mixed clinical sample studies, four used correlational study designs, four used case-control designs and two used a cohort study design (Supplementary Table 1). All of these studies used non-emotional attentional control tasks (nine with an Eriksen Flanker task, one with a Go/No-Go task), except one which used an emotional Go/No-Go task (Supplementary Table 1). All had acceptable levels of bias, but only two studies found any significant differences in CRN measures (Table 1). This included the study which utilised an emotional Go/No-Go attentional control task, where increased CRN amplitude was found in the clinical anxiety compared to the healthy control group (Hum et al., 2013). The other study found that within-person variance in CRN amplitude was higher than between-group variance for all groups, but that the clinical anxiety group required more trials to have a dependable CRN amplitude measure than the healthy control group, when utilising a non-emotional Eriksen Flanker Task with no feedback on performance (Baldwin et al., 2015).

4. Discussion

4.1. Overall findings

The aim of this review was to compare the ERN and CRN findings of attentional control (both emotional and non-emotional) in those with clinical anxiety to healthy controls. A total of 56 of the 66 studies (~85%) measuring ERN found significantly greater ERN amplitudes were associated with clinical anxiety. More specifically, the majority of studies showed significantly greater ERN amplitude in comparison to healthy controls, whilst some showed positive correlations with severity of anxiety symptoms, successful treatment, or an increased likelihood of clinical anxiety (Table 1). The findings of only one study were an exception to this general pattern of results; this correlational clinical anxiety study found that a significant *decrease* in ΔERN was associated with an increase in PTSD related hyperarousal symptoms (Lieberman et al., 2017). Relatedly, whilst another study noted the expected pattern of an increased ERN and ΔERN amplitude in OCD patients compared to healthy controls at baseline, they did note that post- attentional bias training ERN and ΔERN amplitude were significantly *reduced* in the OCD patients only, but not healthy controls (Klawohn et al., 2020b). In contrast to the ERN findings, only nine of the 44 studies (~20%) analysing CRN found significant differences in CRN measures associated with clinical anxiety, all of which were in comparison to healthy controls (Baldwin et al., 2015; Endrass et al., 2010, 2014; Hum et al., 2013; Klawohn et al., 2014; Riesel et al., 2014, 2015, Roh et al., 2016, 2017)

With regards to emotional and non-emotional attentional control tasks, only five of the 57 studies reporting on significant differences in ERN amplitude used emotional attentional control tasks (Hum et al., 2013; Meyer et al., 2019; Roh et al., 2017, 2016; Valt et al., 2018). Of the nine studies observing significant differences in CRN, only three used an emotional attentional control task (Hum et al., 2013; Roh et al., 2017, 2016). Taken together, only six of all the 66 studies extracted on both ERN and CRN used emotional attentional control tasks. Therefore, there is not enough evidence to draw any firm conclusions with respect to emotional versus non-emotional attentional control and future studies should explore this in more detail.

With respect to residualised measures of ERN and CRN, only 12 of the extracted studies utilised these measures (Supplementary Table 1). All 12 used residualised ERN, and only four used residualised CRN (Supplementary Table 1). Of these 12 studies, five analysed residualised ERN or residualised CRN alone, without also analysing ERN, CRN or ΔERN (Supplementary Table 1). Of the seven remaining studies which used multiple measures, only one analysed residualised CRN (Supplementary Table 1), however, it only also analysed ΔERN, not standard CRN, making it difficult to draw any firm conclusions about the potential improved specificity of the residualised CRN measure compared to standard CRN.

Regarding residualised ERN, two studies analysed residualised ERN and the standard ERN alone (Supplementary Table 1), with both showing standard ERN findings to be the same as the residualised ERN findings. Three studies analysed residualised ERN and the ΔERN alone (Supplementary Table 1), of which two found ΔERN findings to be the same as the residualised ERN findings (Crane et al., 2018; Gorka et al., 2018). Together, this suggests that the residualised ERN, is at least consistent with standard and ΔERN measures.

Only two studies analysed residualised ERN as well as standard ERN and ΔERN findings (Hanna et al., 2020; Seow et al., 2020). Whilst Seow et al., (2020), found non-significant findings across all three measures, it is relevant to note that Hanna et al., (2020) found that residualised ERN and standard ERN showed significant differences in a GAD compared to a healthy control group, whereas ΔERN did not. Additionally, one study which analysed residualised ERN and the ΔERN alone, found that residualised ERN showed a significant association with anxiety symptoms, whereas ΔERN did not (Gorka and Phan, 2017). This provides some preliminary evidence in accordance with Meyer et al., (2017a) that residualised ERN may be more sensitive to unique ERN neural activity than ΔERN.

Of the 54 studies which utilised any Eriksen Flanker Task, only 12 utilised no performance feedback, of which nine found significant findings, suggesting that the mere presence of feedback perhaps does not significantly alter findings. Other factors of feedback, such as monetary incentive and/or punishment were only utilised in four studies, so are difficult to draw firm conclusions on. Similarly, 36 utilised performance feedback which emphasised speed and accuracy (Supplementary Table 1), and only one used feedback to emphasise speed only. This makes it difficult to draw any comparisons with the findings of Riesel et al., (2019a), who found ERN amplitude differences between groups

only under a condition which emphasised speed, but not an accuracy condition whilst using a monetary incentive for both conditions.

4.2. Implications of findings

An increase in ERN amplitude across clinical anxiety diagnoses is consistent with findings that individuals with clinical anxiety have increased response monitoring (Simons, 2010) and therefore appear to place greater value on errors (Hajcak et al., 2005). This fixation on errors appears to be reflecting worry, which is in line with core anxiety symptomology (Simons, 2010), rather than an increased motivation to attend to tasks. Increased motivation would be more reflected by consistent increases in *both* ERN and CRN amplitude compared to healthy controls (Simons, 2010) i.e. placing greater value on both errors and correct responses, which is not the case here. Albeit, evidence that greater motivation is linked to increased CRN amplitude, whilst conceptualised by Simons, (2010) has limited empirical evidence at this stage and is restricted only to healthy control samples (Bonnefond et al., 2011, Imhof and Rüsseler, 2019), so this interpretation of the CRN is to be taken with caution. This suggests that our findings are consistent with the conceptualisation of increased ERN amplitude representing a trait marker of clinical anxiety (Meyer, 2017). Interestingly, all studies including samples of children and adolescents with clinical anxiety also showed the consistent pattern of an increase in ERN amplitude. This suggests that the potential use of ERN as a trait marker of clinical anxiety is robust even throughout development. It is also worth noting that 19 of the 21 mixed clinical anxiety samples showed significant ERN results, and again, all except one (Lieberman et al., 2017) were consistent with the ERN pattern (i.e. increased ERN amplitude with clinical anxiety), further reinforcing that ERN may be a robust *transdiagnostic* trait marker of clinical anxiety.

Increased ERN amplitude was found in individuals with clinical anxiety across studies which measured emotional attentional control as well as those which measured non-emotional attentional control. Although, to our knowledge, no studies appear to have specifically investigated between-task ERN or CRN differences in a single sample of individuals with clinical anxiety completing an equivalent emotional and non-emotional attentional control task, this finding was still somewhat unexpected given past related findings in the literature. For example, within-task differences between neutral and emotional stimuli have been found in studies utilising emotional attentional control tasks e.g. neutral versus fearful faces (Roh et al., 2017; 2016). Fearful faces elicited an increased ERN amplitude in individuals with OCD compared to neutral faces, but not in healthy individuals (Roh et al., 2017; 2016). In healthy controls, differential activation of brain regions, including the amygdala and was found depending on the emotional salience of the stimuli during attentional control tasks (Pessoa et al., 2002). However, this distinction seemed to disappear if the task involved high attentional load as this does not leave enough resources for emotional valence to be conveyed through top-down processing (Pessoa et al., 2002; Sutherland et al., 2017). Therefore, it could perhaps be cautiously stated that the lack of ERN or CRN differences found between emotional and non-emotional attentional control tasks for individuals with clinical anxiety could reflect their core worry symptomology, which could lead to this higher attentional load compared to healthy controls. Nevertheless, as only six studies extracted used an emotional control task, further research is necessary to explore this in more detail.

Overall, the current review's findings suggest that the ERN may measure clinical anxiety as a trait construct that is consistent transdiagnostically. As no current studies have investigated the sensitivity, specificity, nor incremental validity of the ERN to make anxiety disorder diagnoses, it would not yet be appropriate to go as far as to describe the ERN as a diagnostic marker of anxiety disorders. However, there appears to be some evidence from the longitudinal studies extracted that the increased ERN amplitude is associated with a greater susceptibility to anxiety disorder diagnosis, at least in children (Meyer et al., 2013) and adolescents (Meyer et al., 2018). However, use of ERN amplitude as a susceptibility marker for anxiety disorder diagnosis would need to be verified in prospective longitudinal study designs for an adult population. The findings also highlight that evidence to date on the use of ERN as a response marker for treatment development appears to be mixed, which may be a function of the heterogeneity of treatment modality. For example, extracted studies showed that successful treatment with modalities such as cognitive behavioural therapy (Carrasco et al., 2013b; Gorka et al., 2018; Kujawa et al., 2016; Ladouceur et al., 2018; Riesel et al., 2015) or selective serotonin reuptake inhibitors (Carrasco et al., 2013b; Kujawa et al., 2016) did not significantly alter ERN amplitude. Conversely other extracted studies have shown that successful treatment with selective serotonin reuptake inhibitors (Gorka et al., 2018) or high-frequency deep transcranial magnetic stimulation (Carmi et al., 2018) are counterintuitively associated with an *increase* in ERN amplitude. However, application of attentional bias modification training has successfully demonstrated that ERN amplitude can be successfully *reduced* in individuals with OCD (Klawohn et al., 2020b), although anxiety and OCD symptoms were not measured post-intervention, so it is unclear if this would function as a treatment per se. Together this suggests the need for further research to gain a better mechanistic understanding of these ERN related changes and perhaps further focusing on treatment interventions which directly target pathophysiological brain activity (i.e. brain stimulation methods), and attentional bias (i.e. computerised interventions).

Response markers are, in the context of anxiety disorders, essentially precursor requirements for prognostic markers i.e. for helping to determine *which* from an array of potential treatment options would be most effective, and *when* they would be most effective (FDA-NIH Biomarker Working Group, 2016). It is therefore a logical extension of the current mixed evidence of response marker findings, that no extracted studies specifically aimed to examine the use of ERN as a prognostic marker for clinical anxiety. However, one extracted study did infer the use of ERN amplitude as a potential prognostic marker (Gorka et al., 2018). Authors suggested that individuals with clinical anxiety with increased pre-treatment ERN amplitude who reported fear-based anxiety symptoms (e.g. phobia, panic etc), may be better suited to cognitive-behavioural therapy than treatment with selective serotonin reuptake inhibitors. Nevertheless, until there is clarification and refinement around the use of ERN as a response marker for treatment development in clinical anxiety, ERN is unlikely to be utilised as a prognostic marker for anxiety disorders.

Whilst currently, the diagnostic and prognostic process for clinical anxiety are subjective, it is critical to consider the potential utility that a collection of objective markers, including the ERN could provide (Boksa, 2013). Whilst it is unlikely that a single measure such as the ERN, could fulfill all potential roles of a marker (i.e. diagnostic, prognostic, treatment response, susceptibility), it may be considered one element in an array of objective biological markers (Cosci and Mansueto, 2020). Specifically, the ERN may offer a better understanding of psychological processes related to anxiety such as attentional control (Davies et al., 2001; Eysenck et al., 2007), action monitoring (Luu et al., 2004) and threat sensitivity (Weinberg et al., 2016). Finding objective measures on the basis of sound neuroscientific evidence is crucial to progressing our understanding of anxiety disorders and could lead to novel treatment approaches (LeDoux and Pine, 2016).

4.3. Limitations

Whilst the review findings suggest that the ERN may measure a transdiagnostic trait marker of clinical anxiety, this interpretation is limited by past findings indicating that a) the ERN is sensitive to state-specific manipulations including e.g. monetary incentive (Pailing and Segalowitz, 2004) and caffeine (Tieges et al., 2004) and b) that the ERN is also related to other cognitive processes including working memory (Miller et al., 2012) and is related to other psychological disorders not

related to anxiety (Pasion and Barbosa, 2019). This suggests that perhaps the ERN should not be considered in isolation and could be examined together with other markers in order to strengthen its specificity. There were also several anxiety disorders that had either limited or nil literature available and thus were not included in the review. Specifically, no studies had any participants with selective mutism, either as a primary diagnosis or co-morbid to another disorder. There were also no studies based on samples of specific phobia, separation anxiety disorder or agoraphobia patients alone. In addition, more common anxiety disorders in the general population (such as specific phobia and SAD, each having a lifetime prevalence of ~13% for adults) (Kessler et al., 2012) were underrepresented in the extracted studies as the primary diagnosis, where no studies were based on samples of specific phobia alone, and only three of the 66 studies had samples for SAD. On the other hand, rarer anxiety disorders such as OCD, with a lifetime prevalence of only ~3% for adults (Kessler et al., 2012) were overrepresented with 35 of the 66 extracted studies including OCD as the primary diagnosis. Therefore, whilst our findings indicate that the ERN has value as a transdiagnostic marker of clinical anxiety, more research is needed to confirm it as a marker across *all* anxiety disorders. Similarly, only six extracted studies used emotional attentional control tasks, making it difficult to draw firm conclusions on the effects of emotion on attentional control and determining if there are any differences between emotional and non-emotional attentional control in clinical anxiety. Likewise, of the 12 extracted studies which used residualised ERN, only one analysed standard ERN, ΔERN and residualised ERN in a single study, also making it difficult to draw firm conclusions on the specificity of the residualised ERN measure. Finally, there were no studies exploring samples of older adults (i.e. aged 60+ years), as the highest mean age found in a clinical sample was only 46.5 years (and 45.4 years for healthy controls). This is a significant gap in the literature especially given that past research has shown that there is potentially an age-related decline in attentional control (Bugg et al., 2007). Therefore, this gap in the literature may limit any conclusions that may be made about the practical clinical utility of markers for older individuals.

4.4. Future directions

The current review highlights the need for additional research in a number of areas. Future studies are needed in this area using more common clinical anxiety populations, such as patients with specific phobia and SAD, in order to confirm the clinical utility of ERN as a transdiagnostic trait marker of clinical anxiety as a whole. Further research is also needed to explore ERN in attentional control in clinical anxiety across the lifespan, especially in older adults i.e. 60 + years old. Given that there is a selection of literature looking at childhood changes in ERN with clinical anxiety (Ladouceur et al., 2006; McDermott et al., 2009; Meyer et al., 2013), this may call for greater efforts for longitudinal studies in adult population groups, which so far have been neglected, even in healthy control groups. This is especially important to help confirm if ERN amplitude can be verified as a marker of anxiety disorder susceptibility in adults as well. Furthermore, future studies should explore the level of sensitivity, specificity, and incremental validity of the ERN to make anxiety disorder diagnoses, to fully consider its potential as an objective diagnostic marker, in the context of a collection of related biological markers of clinical anxiety. Additionally, future research should investigate different measures of attentional control in individuals with anxiety disorders, with a broader variety of ERN and CRN measures. Different tasks should also be utilised that measure emotional attentional control, as they may help in refining a better understanding of the impact emotional stimuli has on links between clinical anxiety and EEG correlates of attentional control, including mechanisms. Finally, future research should continue to investigate the possibility that the ERN may also act as a marker of treatment response. If relevant, such findings would assist in further uncovering the mechanisms between clinical anxiety and attentional control and may aid in the development of novel treatment approaches.

4.5. Conclusions

We extracted 66 papers exploring ERN and/or CRN as EEG correlates of attentional control in individuals with clinical anxiety. Findings indicate the promising utility of ERN in attentional control as a robust, transdiagnostic trait marker of clinical anxiety. However, future research should explore this prospect in more common anxiety disorders and in older adults (aged over 60 years) to ensure that this marker is indeed transdiagnostic and has clinical utility across the lifespan.

Author contribution

JM, MK, PF, BF and KH were involved in the writing of the protocol. JM conducted the literature searches. JM and MW independently conducted the first two screening phrases and checked the methodological quality of the studies. JM wrote the manuscript, with all authors contributing significantly to manuscript revision. All authors have approved the final article.

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Declaration of Competing Interest

JM, MW, MK, BF and KEH reported no biomedical financial interests or potential conflicts of interest.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jad.2021.04.049.

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