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

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Abstract

Altered performance monitoring is implicated in obsessive-compulsive disorder (OCD), Gilles de la Tourette syndrome (GTS), attention-deficit/hyperactivity disorder (ADHD) and autism. We conducted a systematic review and meta-analysis of electrophysiological correlates of performance monitoring (error-related negativity, ERN; error positivity, Pe; feedback-related negativity, FRN; feedback-P3) in individuals with OCD, GTS, ADHD or autism compared to control participants, or associations between correlates and symptoms/traits of these conditions. Meta-analyses on 97 studies (5890 participants) showed increased ERN in OCD (Hedge's g=0.54[CIs:0.44,0.65]) and GTS (g=0.99[CIs:0.05,1.93]). OCD also showed increased Pe (g=0.51[CIs:0.21,0.81]) and FRN (g=0.50[CIs:0.26,0.73]). ADHD and autism showed reduced ERN (ADHD: g=-0.47[CIs:-0.67,-0.26]; autism: g=-0.61[CIs:-1.10,-0.13]). ADHD also showed reduced Pe (g=-0.50[CIs:-0.69,-0.32]). Implications of these findings in terms of shared and distinct performance monitoring alterations across these neurodevelopmental conditions are discussed.

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Keywords: Performance monitoring; attention-deficit/hyperactivity disorder (ADHD); autism; electroencephalography (EEG); error-related negativity (ERN); error positivity (Pe); feedback-P3; feedback-related negativity (FRN); Gilles de la Tourette syndrome (GTS); obsessive-compulsive disorder (OCD)

1. Introduction

1.1 Performance monitoring

Within the context of cognitive control, *performance monitoring* refers to a set of neural processes that support the continuous monitoring of thoughts and actions. This mechanism ensures our cognition and behaviour remain consistent with our current goals and can be adapted in response to changes in the environment (Holroyd & Coles, 2002; Ullsperger et al., 2014; Yeung et al., 2004). For example, on our daily commute to work or school we notice and adapt our behaviour in line with events that occur on the journey, such as changing route if a road is closed or returning home if we forget something important. Being able to detect any errors we might have committed during a task, or to adequately monitor someone else's feedback on our behaviour, is crucial to adapt to the surrounding environment. Findings from electroencephalography (EEG) and functional magnetic resonance imaging (fMRI) studies indicate that performance monitoring (including error detection and feedback processing) is mediated in part by the anterior cingulate cortex (ACC), which receives signals from other brain regions that indicate whether a specific action was better or worse than expected (Holroyd & Coles, 2002; Ullsperger et al., 2014; Yeung et al., 2004). Changes in ACC activity are thought to flag when a mismatch occurs in this prediction, i.e. when an action has more positive or negative outcomes than intended, to facilitate adaptive changes in behaviour in response to internal and external environmental demands (Holroyd & Coles, 2002; Ullsperger et al., 2014; Yeung et al., 2004).

1.2 Electrophysiological correlates of performance monitoring

Performance monitoring is an ongoing, fast and adaptive process. As such, EEG, which has a high temporal resolution and can capture moment-to-moment changes in brain function in the millisecond time-range, is particularly appropriate for investigating neural activity associated with performance monitoring. Over the last thirty years, EEG studies have identified robust electrophysiological correlates of performance monitoring. For example, when the EEG signal is averaged in relation to participants' motor responses to create a response-locked event-related potential (ERP), several components (i.e. positive and negative deflections) of the ERP waveform reflect different aspects of performance monitoring. The earliest of these components is the error-related negativity (ERN or Ne), a negative deflection in the ERP waveform that occurs around 0-100ms following an erroneous response and is maximal at frontocentral electrodes (Falkenstein et al., 1991; Gehring et al., 1993). Source localisation and concurrent EEG+fMRI studies indicate that the ERN is generated by the ACC (Debener et al., 2005; Yeung et al., 2004). The ERN is believed to reflect activity of the ACC as it detects a mismatch between the expected and actual outcome of the erroneous action; this change in ACC activity acts as a signal to other brain regions that the outcome was worse than expected (Holroyd & Coles, 2002; Ullsperger et al., 2014; Yeung et al., 2004). A corresponding negative component, the correct-related negativity (CRN), occurs following correct responses in the same time-range and at the same electrodes as the ERN but is smaller in amplitude (Falkenstein et al., 1991; Gehring et al., 1993). Larger ERN than CRN is consistent with the idea that ACC activity in the context of performance monitoring reflects mismatches between actions and outcomes; the mismatch between the expected and actual outcome of a behaviour is smaller when the behaviour is correct than when it is erroneous (Holroyd & Coles, 2002).

Following the ERN, the error positivity (Pe) is a positive deflection in the response-locked ERP waveform that is maximal at centro-parietal electrodes between 150-500ms (Falkenstein et al., 1991; Falkenstein et al., 2000). A corresponding positive deflection (the Pc) occurs following correct responses but is smaller in amplitude than the Pe (Falkenstein et al., 1991; Falkenstein et al., 2000). The Pe is believed to reflect the conscious evaluation of the detected error and the initiation of corrective cognitive and behavioural processes (Falkenstein et al., 2000; Ullsperger et al., 2010). In support, the Pe has been shown to be present for consciously detected errors and absent for errors of which the participant is unaware (Nieuwenhuis et al., 2001; Overbeek et al., 2005). In some studies, the amplitude of the Pe correlates with adaptive slowing in responses after an error has been made (post-error slowing), a behavioural strategy implemented to reduce the likelihood of further error commissions (Hajcak et al., 2003, but see Overbeek et al., 2005). Source modelling EEG studies and investigations of aware vs. unaware errors with fMRI indicate that the Pe is generated by the anterior insula, a region involved in processing the salience of internal and external events, interoception, and the integration of sensory and motor information (Dhar et al., 2011; Ullsperger et al., 2010). Source-modelling studies also show that the orbitofrontal cortex (OFC) is activated in a time-range following the Pe, which may reflect further evaluative and decision-making processes related to the error (Dhar et al., 2011). A recent study also found that the Pe can occur in the absence of the ERN, suggesting that these components might reflect partially independent error-monitoring mechanisms (Di Gregorio et al., 2018).

A similar series of components can be seen in electrophysiological activity averaged in relation to performance feedback presented to participants. The earliest component, the feedback-related negativity (FRN), is a negative deflection between ~250-350ms post-feedback

that is maximal at frontocentral sites (Miltner et al., 1997). The FRN is larger for performance feedback that is negative (i.e. when the participant is told their response was incorrect) rather than positive (when the feedback indicates that the response was correct) (Miltner et al., 1997; Hajcak et al., 2006) or surprising rather than expected (Bellebaum & Daum, 2008; Hauser et al., 2014). Source-localisation and concurrent EEG+fMRI studies indicate that the FRN is generated by the ACC (Bellebaum & Daum, 2008; Hauser et al., 2014; Miltner et al., 1997). Similar to the ERN, the FRN is thought to reflect activity of the ACC as it detects a mismatch in the signals it receives, indicating that a behaviour was better or worse than predicted (Chase et al., 2011; Hauser et al., 2014). One important difference between the ERN and FRN is that the former is generated by *internal* monitoring while the latter is triggered by *external* monitoring of performance. These internal and external monitoring processes appear to coordinate during situations when we learn new behaviours. Specifically, during early phases of learning, the FRN is larger than the ERN, indicating that correct behaviour is evaluated via the provision of external performance feedback (Bellebaum & Coloso, 2014; Holroyd & Coles, 2002; Pietschmann et al., 2008). Later in the learning process, once the to-be-learned behaviour is better consolidated, the ERN becomes larger than the FRN, indicating more reliance on internally-driven representations and monitoring (Bellebaum & Coloso, 2014; Holroyd & Coles, 2002; Pietschmann et al., 2008).

Following the FRN, the feedback-locked P3 component is a positive deflection in the ERP waveform that is maximal around 300-600ms at parietal electrodes (Miltner et al., 1997). Like the FRN, the feedback-P3 is also larger for unexpected than expected performance feedback (Pfabigan et al., 2011; Yeung & Sanfey, 2004) and some studies indicate this component is particularly sensitive to rewarding outcomes, i.e. performance feedback indicating behaviour was better than expected (Bellebaum & Daum, 2008; Hajcak et al., 2005). The feedback-P3 has been

interpreted by some authors as reflecting activity of the locus coeruleus physiological arousal system in motivation-based decision-making (Nieuwenhuis et al., 2005a) and by others as indexing the updating of working memory representations based on performance feedback (Fischer & Ullsperger, 2013).

In addition to these ERP components, performance monitoring can also be indexed by oscillatory electrophysiological activity in the theta frequency range (4-8Hz) over frontocentral electrode sites (Cavanagh & Frank, 2014). Similar to the ERN and FRN ERP components, the magnitude of frontocentral theta activity (measured as the power, μ^2, of the electrophysiological signal) or the degree of theta phase-locking across experimental trials (the consistency in the phase of oscillatory theta activity across trials) are larger for error vs. correct response trials (Cohen, 2011; Luu et al., 2004) and for negative or unexpected feedback compared to positive or expected feedback (Cavanagh et al., 2010; Cohen et al., 2007). Theta activity in these contexts has been source-localised to the ACC and may contribute to the amplitude of the ERN and FRN, either by phase resetting of ongoing oscillations or additional amplitude in those oscillations (Cavanagh et al., 2010; Cohen et al., 2007; Cohen, 2011; Luu et al., 2004). Thus, frontocentral theta, like the ERN and FRN, is thought to reflect the detection of a mismatch in predicted vs. actual behaviour by the ACC (Cavanagh & Frank, 2014).

1.3 Altered performance monitoring in neurodevelopmental conditions characterised by restricted, repetitive, compulsive and impulsive behaviours

Alterations in performance monitoring have been proposed to contribute to the restricted, repetitive, inflexible, compulsive or impulsive behaviours that characterise several neurodevelopmental conditions. For instance, it has been proposed that the excessive and

repetitive compulsive behaviours in obsessive-compulsive disorder (OCD) could arise from a faulty and overactive performance monitoring system, which erroneously indicates that a performed behaviour (e.g. hand-washing) did not achieve the required outcome (e.g. clean, contamination-free hands) and should be repeated (Gehring et al., 2000; Nieuwenhuis et al., 2005b; Norman et al., 2019). In support, individuals with OCD show atypically increased amplitude of the ERN component during error commission compared to control participants without OCD (Gehring et al., 2000; Nieuwenhuis et al., 2005b), indicative of overactive error monitoring in OCD. Recent meta-analyses have confirmed that increased ERN amplitude is a robust finding in OCD and is unaffected by factors such as age, sex, and severity of OCD symptoms (Moser et al., 2016; Riesel et al., 2019). Further, a previous systematic review indicated that although some individual studies report enhanced amplitude of the CRN on correct-response trials in OCD, findings are markedly less consistent than those reporting enhanced ERN amplitude on error trials (Endrass & Ullsperger, 2014). These findings suggest that overactive performance monitoring may be specifically related to errors in OCD.

Interestingly, the previous meta-analysis by Riesel (2019) and the systematic review by Endrass & Ullsperger (2014) found that increased ERN amplitude is not present in OCD in all contexts; increases were found during performance of cognitive control tasks (e.g. the Flanker) but not during other experimental paradigms such as probabilistic learning tasks. Furthermore, Endrass and Ullsperger (2014) also highlighted in their systematic review that other electrophysiological correlates of performance monitoring, including the Pe and FRN, appear not to be altered in OCD. These findings suggest a high degree of specificity in performance monitoring alterations in OCD, which may be useful for identifying more precise neurocognitive alterations involved in the condition and novel targets for treatment (Shephard et al., 2021a).

However, to our knowledge, there have been no meta-analyses of performance monitoring components other than the ERN in OCD, and no further systematic reviews of these components since Endrass and Ullsperger (2014). An updated meta-analysis of performance monitoring components in OCD is therefore needed to confirm these findings.

Similar to OCD, it has been suggested that overactive performance monitoring is involved in the repetitive motor and phonic tic symptoms of Gilles de la Tourette syndrome (GTS) (Johannes et al., 2002; Müller et al., 2003). Indeed, some previous studies have reported increased ERN amplitude in GTS compared to control participants without tics (Johannes et al., 2002; Schüller et al., 2018), a pattern similar to the increased ERN observed in OCD (Riesel, 2019). However, whether increased ERN amplitude reflects the involvement of overactive error-monitoring as a causal mechanism in the production of tics is unclear; several authors instead propose that increased performance monitoring reflects a compensatory or adaptive mechanism that helps individuals with GTS monitor and voluntarily suppress their tics (Jackson et al., 2007; Schüller et al., 2018). Furthermore, some studies have reported no alterations in the ERN or other performance monitoring components in GTS (Shephard et al., 2016a, 2016b).

The inconsistency in findings of altered electrophysiological correlates of performance monitoring in GTS was confirmed in a recent systematic review (Morand-Beaulieu & Lavoi, 2019), although the authors did not comprehensively examine factors that might contribute to the variability in findings. One important factor that is known to influence neurocognitive function in GTS is age. The typical clinical course of GTS is that tics attenuate during adolescence and the majority of individuals have no or minimal/mild tics in adulthood (Groth et al., 2017). It is thought that adults with GTS represent an atypical and perhaps more severe form of the condition with additional neurocognitive alterations not seen in children with the more typical

remitting course of GTS (Jackson et al., 2015). Another important factor is the presence of co-occurring conditions. For example, previous studies have shown that co-occurring symptoms of attention-deficit/hyperactivity disorder (ADHD) introduce impairments in neurocognitive function in GTS, including performance monitoring (Shephard et al., 2016a, 2016b). Thus, further work is needed to clarify the nature of altered performance monitoring in GTS, and in particular whether previous inconsistent findings in electrophysiological indices of performance monitoring might reflect differences in age and the presence of co-occurring conditions across samples.

In contrast to OCD and GTS, in ADHD it has been proposed that underactive or hypofunctioning performance monitoring may cause difficulties in the evaluation and adjustment of erroneous or inappropriate behaviours; this in turn leads to poor self-regulatory control more broadly and the inattentive, hyperactive and impulsive symptoms of the condition (Sergeant et al., 2003; Shiels & Hawk, 2010). In support, previous studies have reported reduced amplitude of the ERN and Pe during error commission (Groom et al., 2013), reduced FRN amplitude during feedback processing (Hauser et al., 2014) and reduced error-related theta activity (Groom et al., 2010) in children and adults with ADHD compared to control participants without ADHD. These findings suggest that ADHD is associated with extensive performance monitoring difficulties. However, findings of reduced ERN but not Pe (McLoughlin et al., 2009) and reduced Pe but not ERN (Wiersema et al., 2005) have also been reported, suggesting a more complex pattern of alterations in error monitoring in ADHD. Further, some studies have reported enhanced FRN in ADHD, especially to negative feedback; this pattern has been interpreted as reflecting a heightened sensitivity to negative outcomes of behaviour (van Meel et al., 2005) and/or an

overreliance on external rather than internal performance monitoring (Shephard et al., 2016a; Thoma et al., 2015).

Previous meta-analyses of electrophysiological correlates of performance monitoring in ADHD have also yielded inconsistent findings. Geburek et al. (2013) reported significantly reduced ERN but not Pe amplitude in seven studies examining these components in children and adults with ADHD during two experimental tasks (Go/Nogo and Flanker tasks). In contrast, Kaiser et al. (2020) reported significantly reduced Pe but not ERN amplitude in ten studies examining error monitoring in children and adults with ADHD. Importantly, a number of published studies investigating these components were not included in either of those meta-analyses (e.g. Shephard et al., 2016b; Wiersema et al., 2005). To our knowledge, there has been no published meta-analysis of feedback-locked performance monitoring components in ADHD. A more comprehensive meta-analysis of both response- and feedback-locked components may help to clarify the nature of performance monitoring alterations in ADHD.

Finally, inefficient performance monitoring has been proposed to contribute to symptoms of autism, such that reduced ability to continuously monitor, evaluate and adapt behaviour in response to prediction-outcome mismatches may underlie the inflexible, restricted and repetitive behaviour symptoms of the condition (D'Cruz et al., 2016; Mundy et al., 2003). In line with this proposal, previous studies have reported reduced ERN (Santesso et al., 2011; South et al., 2010; Vlamings et al., 2008) and Pe (Santesso et al., 2011; Vlamings et al., 2008) amplitudes during error commission in autistic children and adults compared to non-autistic control participants. However, several studies have reported no alterations in the ERN (Clawson et al., 2017; Henderson et al., 2015) and Pe (South et al., 2010) or increased ERN (Suzuki et al., 2017) in autistic individuals. Similarly, mixed findings of reduced (Bellebaum et al., 2014) and unaltered

(McPartland et al., 2012) FRN amplitude have been reported in autistic children and adults. A previous systematic review of performance monitoring components in autism concluded that internal performance monitoring indexed by the ERN/Pe appears to be impaired while external performance monitoring reflected by the FRN and other feedback-locked components is unaffected (Hüppen et al., 2016). Nevertheless, no meta-analysis on electrophysiological correlates of performance monitoring in autism has been published, which would help to clarify the presence or absence or alterations associated with the condition.

1.4 Motivation and aims for the current systematic review and meta-analysis

The evidence from electrophysiological studies to date indicates that alterations in performance monitoring may be involved in the repetitive, inflexible, compulsive or impulsive symptoms of OCD, GTS, ADHD and autism. However, more comprehensive meta-analyses are needed to clarify the pattern of alterations associated with each condition across different performance monitoring contexts (e.g. internal vs. external) and correlates (e.g. ERN vs. Pe). Furthermore, an important limitation of the previous meta-analytic work is that comparisons of performance monitoring have not been made across these neurodevelopmental conditions. Such comparisons are important because OCD, GTS, ADHD and autism frequently co-occur in the same individuals (Jensen et al., 2015; Lai et al., 2019; Lebowitz et al., 2012; Martin et al., 2018). Identifying neurocognitive alterations that are shared between these conditions may be helpful in understanding the mechanisms that contribute to their co-occurrence. Based on the previous work summarised above, it could be hypothesised that increased electrophysiological correlates of performance monitoring may be shared between OCD and GTS, while reduced electrophysiological markers of performance monitoring might be a common alteration between

ADHD and autism. If these hypotheses are correct, alterations in electrophysiological correlates of performance monitoring may be useful as transdiagnostic markers of the neurobiological mechanisms involved in repetitive, inflexible, compulsive and impulsive behaviours across common neurodevelopmental conditions.

The aim of the current work was to attempt to address these limitations by conducting a systematic review and meta-analysis of electrophysiological correlates of performance monitoring in OCD, GTS, ADHD and autism. We included studies that compared these correlates between individuals with one of these conditions to control participants without these conditions, or that examined associations between the correlates and symptoms or traits of OCD, GTS, ADHD and autism. We aimed to (1) test whether performance monitoring alterations are present across contexts (e.g. internal vs. external monitoring) and electrophysiological components (e.g. ERN vs. Pe) in each neurodevelopmental condition, and (2) compare the magnitude and direction of effect sizes for each component between OCD, GTS, ADHD and autism to better understand whether performance alterations are shared across these commonly co-occurring neurodevelopmental conditions. An additional aim (3) was to investigate whether developmental stage (childhood and adolescence vs. adulthood) influences performance monitoring alterations in GTS given that adulthood GTS is thought to be a more severe form of the condition. For completeness, we also investigated whether age affected performance monitoring alterations in OCD, ADHD and autism although we did not expect developmental differences in these conditions. A final aim (4) was to assess whether the co-occurrence of one or more of these neurodevelopmental conditions modifies performance monitoring alterations, for example, whether individuals with OCD without GTS differ from those with OCD and co-occurring GTS.

2. Methods

2.1 PRISMA statement and pre-registration

Methodology and reporting for this systematic review and meta-analysis are in line with the PRISMA statement (see PRISMA Checklist in eAppendix 1). The protocol was pre-registered on PROSPERO (CRD42019134612).

2.2 Search strategy and selection criteria

A systematic literature search was conducted to identify eligible articles using three electronic databases (MEDLINE, EuropePMC, Scopus) and reference lists of eligible articles and review articles. The searches included peer-reviewed journal articles written in any language that were accepted for publication from the beginning of time until the final search date (8th February 2021). Articles published in languages other than those understood by the research team (English, Portuguese, Spanish, Italian) were translated using online software. Keywords used in the searches were: “performance monitoring”, “action monitoring”, “EEG”, “electrophysiology”, “event-related potentials”, “ERP”, “feedback-related negativity”, “error-related negativity”, “theta”, “autism”, “ASD”, “attention-deficit/hyperactivity disorder”, “ADHD”, “hyperkinetic disorder”, “obsessive-compulsive disorder”, “OCD”, “Gilles de la Tourette syndrome”, “GTS”, “TS”, “Tourette syndrome”, “tics”. The specific search conducted in all three databases is given in eAppendix 2.

Studies were considered eligible for inclusion if they met one of the following criteria: (1) empirical studies that compared electrophysiological correlates of performance monitoring between individuals with OCD/GTS/ADHD/autism to control participants without these

conditions and/or to each other, or (2) empirical studies that examined associations between symptoms or traits of OCD/GTS/ADHD/autism and electrophysiological correlates of performance monitoring. Articles were excluded if they were reports of case studies or review articles without empirical data.

2.3 Data selection, extraction and coding

Titles and abstracts of studies retrieved from the searches were screened independently by two authors (AB and ES) to identify those that potentially met inclusion criteria. Disagreements in the eligibility of articles were resolved through discussion between AB and ES. Next, the full text of each article marked as eligible for inclusion was assessed and data were extracted using standardised forms by two independent authors (ES and one of AB/AW/CYO/II/PFZ). Extracted information included: study population and design, study location, participant characteristics (inclusion and exclusion criteria, age, sex, socioeconomic status, racial/ethnic background, intellectual ability), clinical assessments and characteristics (diagnostic instruments, measures of symptoms, presence of co-occurring conditions, medications), electrophysiological outcome measures (experimental task in which performance monitoring was studied, electrophysiological indices of performance monitoring), summary statistics, statistical model results and effect sizes. Data not available from publications were requested from corresponding, first or senior authors. Disagreements during data extraction were resolved through discussion between the first and senior authors (AB and ES).

For electrophysiological outcome measures, we focused on amplitude rather than latency of ERP components since alterations in amplitudes have been more frequently reported in previous work and because many studies do not report latency as well as amplitude. For

oscillatory theta correlates of performance monitoring, we extracted whichever measures of magnitude were reported in the articles, such as power (amplitude squared), coherence, phase synchrony or inter-trial phase coherence. We initially intended and began to extract data on behavioural measures of performance monitoring. However, during the data extraction process it became clear that too few studies reported specific behavioural measures of performance monitoring, such as post-error slowing, and that it would be complicated to select a measure of performance from each study that reflected performance monitoring specifically rather than another cognitive control process. For example, in the commonly used Flanker task there are at least four behavioural measures (accuracy and reaction time for congruent and incongruent conditions) that index performance ability but are difficult to attribute to performance monitoring rather than other aspects of cognitive control, such as inhibition or attention. We therefore did not include behavioural measures of performance monitoring in the review and meta-analysis.

2.4 Assessment of study quality

Study quality was rated independently by each author in the pair of researchers who conducted data extraction for each article. We used the Newcastle-Ottawa Scale (NOS, Wells et al., 2004) to classify studies as good, fair or poor quality according to the criteria specified in the NOS. Disagreements concerning study quality were resolved through discussion between the first and senior authors (AB and ES).

2.5 Data synthesis and analysis

A narrative synthesis was created with studies grouped according to neurodevelopmental condition (OCD, GTS, ADHD, autism) and electrophysiological correlate of performance

monitoring (e.g. ERN, FRN). Meta-analyses were conducted in *R 4.0.2* (R Core Team, 2020) to estimate the pooled effect size across studies for each electrophysiological component for each neurodevelopmental condition. The standardised mean difference (Hedge's g) was calculated for all studies and used as the measure of effect size in meta-analyses. For studies that reported findings from analyses that were not based on group differences, such as correlation or regression, the effect sizes were converted to Hedge's g using the R package *esc* (Lüdecke, 2019). If different studies reported data on the same electrophysiological measure from the same sample of participants, the study with the largest sample size for analysis and/or clearest reporting of results was selected.

Two studies (Groen et al., 2013; Groom et al., 2010) reported results for participants with ADHD tested once while taking stimulant medication and once while off-medication (Groom et al., 2010), or results for a group of participants with ADHD taking stimulant medication compared to a group of participants not taking stimulants (Groen et al., 2013). In these cases, we selected the data from the group of participants with ADHD who were tested off-medication for inclusion in the meta-analysis. Our rationale for this is that it is standard practice to assess EEG in participants with ADHD following a 24-48-hour stimulant wash-out period, since stimulants are known to enhance electrophysiological correlates of cognitive control (e.g. Groom et al., 2010). For medications other than stimulants, we intended to conduct a meta-regression analysis to examine the effect of medication on the pooled effect sizes. However, many studies did not report medication status of participants and or medication status was reported in an inconsistent way across studies (e.g. *n* participants on medications in some studies, a list of the medications taken but not by how many participants in other studies). Therefore, we did not include an analysis of medication in the current narrative synthesis or meta-analysis.

Some studies reported data on the same electrophysiological component measured on multiple different experimental tasks or conditions. In these cases, we computed effect sizes for the components in each condition separately and effect sizes were nested within the study in meta-analysis to account for non-independence of data. Many studies reported results for the same component measured at several different electrode sites (e.g. the ERN at FCz and Cz). For these studies, we created an average of the mean and SD for the component across the electrode sites; these averages were then used to compute one Hedge's g effect size across electrodes. It was also common for studies to report data on a component computed in more than one way, e.g. the ERN computed for error trials only, correct trials only, and as a difference score for error vs. correct trials. In these cases, we selected the data from the method that most clearly isolates the performance monitoring aspect of the component (e.g. the difference score for the ERN) to compute effect size.

If studies included participant groups that were defined as having a condition that was investigated in the current review (OCD, GTS, ADHD, autism) and a co-occurring condition that was not the focus of the current review (e.g. OCD and co-occurring anxiety), then only the group without the co-occurring condition was included in the narrative synthesis and meta-analysis. Studies that included a group of participants defined as having co-occurring presentations of OCD, GTS, ADHD or autism were excluded from the analyses of performance monitoring related to those conditions individually. The data were instead included in a separate section of the narrative synthesis focused on co-occurring presentations of OCD, GTS, ADHD and autism.

Multi-level random-effects meta-analytic models were fitted to the data in *metafor* (Viechtbauer, 2010) with effect sizes nested within studies for those that reported multiple effect sizes for the same component to account for non-independence of data. The Restricted

Maximum-Likelihood (REML) estimator was used with the Knapp-Hartung confidence interval adjustment (Langan et al., 2019). Heterogeneity was assessed with I^2 using the *dmetar* package (Harrer et al., 2019). Publication bias was assessed using funnel plots and the rank correlation test for asymmetry in *metafor* (Viechtbauer, 2010). Significant findings in the meta-analyses were followed by subgroup analyses conducted to test for moderating effects of developmental stage (coded 0 = childhood/adolescence; 1 = adulthood), whether effect size was converted or not (coded 0 = not converted; 1 = converted), and study quality rating (coded 0 = good, 1 = fair, 2 = poor) on the pooled effect size estimates. All R data and code are available here: https://osf.io/y7zts/

3. Results

3.1 Sample

After full text reading, 107 non-duplicate studies met inclusion criteria (6304 participants in total; 5341 participants from case-control studies: 52% with a diagnosis or high-traits of a condition under study; 963 participants from cohort studies: 12% with a clinical diagnosis) (Figure 1, Table 1). Of these, 41 studies investigated OCD (939 participants with a diagnosis of OCD; 156 with high obsessive-compulsive (OC) traits; 283 with varying levels of OC traits; 1143 control participants without OCD or with low OC traits), six studies investigated GTS (91 participants with a diagnosis of GTS or tic disorder; 120 control participants without GTS or tics), 46 studies investigated ADHD (1124 participants with a diagnosis of ADHD; 977 control participants without ADHD; 567 with varying levels of ADHD traits) and 21 studies investigated autism (526 autistic participants; 468 non-autistic control participants). Four studies investigated these conditions as co-occurring presentations in the same individuals (44 participants with co-

occurring ADHD+GTS and 9 participants with co-occurring OCD+tics). Further details of the samples for each condition are described in the following sections. All 107 studies were included in narrative syntheses and 97 studies (5890 participants) were included in meta-analysis (Figure 1).

[Figure 1]

[Table 1]

3.2 OCD

3.2.1 ERN in OCD

Of the 41 studies investigating OCD, 39 examined the ERN (Table 2). Studies mainly focused on adults (k=33) rather than children or adolescents (k=6). All 39 studies provided sufficient information for effect sizes to be computed and were included in meta-analysis. Ten studies reported the ERN in more than one experimental task or condition. Meta-analyses conducted on 52 effect sizes from the 39 studies (Table 2) showed that ERN amplitude was significantly increased in participants with OCD compared to those without (g=0.54, se=0.05, CIs (95%)=[0.44,0.65], t=10.15, p<0.0001, Figure 2). Cross-study heterogeneity was considerable (I^2=43.49%) and publication bias was significant (Tau=0.30, p=0.0016, Figure 2). Subgroup analysis showed that the pooled effect size was not significantly moderated by developmental stage (children/adolescent vs adult participants) (F(1,50)=0.0008, p=0.98), conversion of effect size prior to meta-analysis (F(1,50)=1.20, p=0.28) or quality rating (F(1,50)=0.26, p=0.62).

[Table 2]

[Figure 2]

3.2.2 Pe in OCD

Nine studies examined the Pe in OCD (Table 3). Most studies (8/9) investigated the Pe in adult participants. Eight of the nine studies provided sufficient information for effect sizes to be computed and were included in meta-analysis. Meta-analysis on eight effect sizes from the eight studies revealed that Pe amplitude was significantly larger in OCD than controls (g=0.51, se=0.13, CIs (95%)=[0.21,0.81], t=4.05, p=0.0049, Figure 3). Heterogeneity was moderate (I^2=34.35%) and publication bias was not significant (Tau=0.07, p=0.90, Figure 3). Subgroup analyses were non-significant for developmental stage (F(1,6)=2.98, p=0.13), conversion of effect size (F(1,6)=0.08, p=0.78) and quality rating (F(1,6)=2.41, p=0.17). The remaining study for which an effect size could not be computed reported no significant difference in Pe amplitude between OCD and control groups (Table 3).

[Table 3]

[Figure 3]

3.2.3 FRN in OCD

Eight studies investigated the FRN in OCD, all of which were conducted with adults rather than children or adolescents (Table 4). All eight studies provided sufficient data to compute effect sizes and were included in meta-analysis. Four studies reported the FRN in more than one experimental condition or task. Meta-analysis on 13 effect sizes from the eight studies showed that FRN amplitude was significantly larger in OCD than controls (g=0.50, se=0.11, CIs (95%)=[0.26,0.73], t=4.65, p=0.0006, Figure 4). Cross-study heterogeneity was low (I^2=20.75%) and publication bias was not significant (Tau=0.30, p=0.16, Figure 4). The pooled effect size was not significantly moderated by whether effect size was converted or not prior to meta-analysis (F(1,11)=3.58, p=0.08) or study quality rating (F(1,11)=0.29, p=0.60).

[Table 4]

[Figure 4]

3.2.4 Other performance monitoring components in OCD

Other performance monitoring components examined in OCD were error-related theta activity (two studies) and the feedback-P3 (two studies) (Table 5). These studies were included in narrative synthesis only since the sample size for each component was too small for meta-analysis. A descriptive summary of the findings is presented in Table 5. The findings for error-related theta activity were mixed, with one study reporting significantly increased theta power in adults with OCD compared to controls (Riesel et al., 2014) and the other study reporting no differences in theta power in OCD compared to controls (Carmi et al., 2019). Findings for the feedback-P3 were more consistent, with both studies reporting no differences in feedback-P3 amplitude between OCD and controls (Endrass et al., 2013) or no associations between feedback-P3 amplitude and OCD traits (Doñamayor et al., 2014). However, Endrass et al. (2013) reported different patterns of feedback-P3 amplitude modulation in response to positive and negative feedback within the OCD and control groups (Table 5).

[Table 5]

3.3 GTS

3.3.1 ERN in GTS

Of the six studies investigating electrophysiological correlates of performance monitoring in GTS, five examined the ERN (Table 6). Two studies investigated the ERN in children or adolescents and three studies in adults. All five studies provided sufficient data for computation of effect sizes. Meta-analysis on five effect sizes from the five studies indicated that ERN amplitude was significantly greater in GTS than controls (g=0.99, se=0.34, CIs (95%)=[0.05,1.93], t=2.92, p=0.0433, Figure 5). Heterogeneity was high (I^2=77.05%) though

publication bias was not significant (Tau=0.20, p=0.82, Figure 5). Developmental stage did not moderate the pooled effect size (F(1,3)=0.20, p=0.68). Subgroup analyses were not conducted for the moderators 'conversion of effect size' and 'study quality rating' due to insufficient variability in these factors across studies (Table 6).

[Table 6]

[Figure 5]

3.3.2 Other performance monitoring components in GTS

Other performance monitoring components examined in GTS were the Pe (three studies) and the feedback-locked P2 and FRN (one study). Since there were too few of these studies for meta-analysis, they were included in narrative synthesis only. A descriptive summary of these studies is provided in Table 7. Narrative synthesis indicated mixed findings of no alterations in Pe amplitude between GTS and controls in two studies (Eichele et al., 2016; Shephard et al., 2016b) but significantly reduced Pe amplitude in the third study (Schüller et al., 2018). The Pe was measured in children and adolescents in all three studies. The only study examining feedback-locked components in GTS reported no differences in FRN amplitude but a trend for reduced feedback-P2 amplitude in children and adolescents with GTS compared to controls (Shephard et al., 2016a).

[Table 7]

3.4 ADHD

3.4.1 ERN in ADHD

Of the 46 studies investigating performance monitoring components in ADHD, 33 examined the ERN (Table 8). Twenty-eight studies provided sufficient data for the computation of effect sizes and were included in meta-analysis; the remaining five studies were included in

narrative synthesis only. Of the 28 studies included in the meta-analysis, one study reported effect sizes for the ERN in more than one experimental condition; seventeen studies were conducted with children and adolescents and 11 with adults. Meta-analysis on 30 effect sizes from the 28 studies showed that ERN amplitude was significantly reduced in ADHD compared to controls (g=-0.47, se=0.10, CIs (95%)=[-0.67,-0.26], t=-4.64, p<0.0001, Figure 6). The pooled effect size remained significant when one outlying effect size (Balogh et al., 2017 (1), Figure 6, Table 8) was excluded (g=-0.41, se=0.09, CIs (95%)=[-0.59,-0.23], t=-4.76, p<0.0001). Cross-study heterogeneity was high (I^2=76.25%) but publication bias was not significant (Tau=-0.05, p=0.72, Figure 6). Subgroup analysis indicated that none of the factors investigated significantly moderated the pooled effect size (developmental stage: F(1,28)=4.28, p=0.05; effect size conversion: F(1,28)=0.004, p=0.95; study quality rating: F(1,28)=0.25, p=0.62). Two of the five studies included in narrative synthesis also reported significantly reduced ERN in ADHD (Table 8).

[Table 8]

[Figure 6]

3.4.2 Pe in ADHD

Twenty-nine studies investigated the Pe in ADHD (Table 9). Effect sizes could be computed for 26 of these studies, which were included in meta-analysis. The remaining three studies were included in narrative synthesis. Of the 26 studies included in meta-analysis, 17 were conducted with children or adolescents and three studies reported effect sizes for the Pe in more than one experimental condition. Meta-analysis conducted on 30 effect sizes from the 26 studies revealed significantly reduced Pe amplitude in ADHD compared to controls (g=-0.50, se=0.09, CIs (95%)=[-0.69,-0.32], t=-5.62, p<0.0001, Figure 7). The pooled effect size remained

significant when one outlying effect size (Balogh et al., 2017 (3), Figure 7, Table 9) was excluded (g=-0.46, se=0.07, CIs (95%)=[-0.60,-0.32], t=-6.55, p<0.0001). Heterogeneity was high (I^2=66.18%) but publication bias was not significant (Tau=-0.12, p=0.36, Figure 7). Developmental stage (F(1,28)=0.04, p=0.85), conversion of effect size (F(1,28)=0.18, p=0.67) and study quality rating (F(1,28)=0.73, p=0.40) were not significant moderators of the pooled effect. Two of the three studies included only in narrative synthesis also reported significantly reduced Pe amplitude in ADHD (Table 9).

[Table 9]

[Figure 7]

3.4.3 FRN in ADHD

Thirteen studies investigated the FRN in ADHD (Table 10). Effect sizes could be computed for 11 studies, which were included in meta-analysis. Of these, three studies reported effect sizes for the FRN in more than one experimental condition. The majority of studies investigated the FRN in children and adolescents (Table 10). Meta-analysis on 15 effect sizes from 11 studies showed no significant difference in FRN amplitude between ADHD and controls (g=-0.07, se=0.31, CIs (95%)=[-0.99,0.72], t=0.21, p=0.83, Figure 8). These results were unchanged when three outlying effect sizes (Ibanez et al., 2012 (1-3), Figure 8, Table 10) were excluded (g=-0.22, se=0.15, CIs (95%)=[-0.55,0.11], t=-1.49, p=0.16). Heterogeneity was high (I^2=92.93%) and publication bias was significant (Tau=0.62, p=0.0008, Figure 8). The two studies not included in meta-analysis also reported no significant difference in FRN amplitude between ADHD and controls (Table 10).

[Table 10]

[Figure 8]

3.4.4 Feedback-P3 in ADHD

Eight studies investigated the feedback-P3 in ADHD (Table 11). Six studies provided sufficient data to compute effect sizes and were included in meta-analysis. Two of these studies reported the FRN in more than one experimental condition. Three of the six studies included in meta-analysis and five of the eight studies overall were conducted with children and adolescents rather than adults. Meta-analysis on nine effect sizes from the six studies revealed no significant difference in feedback-P3 amplitude between ADHD and controls (g=0.03, se=0.43, CIs (95%)=[-0.96,1.01], t=0.06, p=0.95, Figure 9). These findings did not change when two outlying effect sizes (Ibanez et al., 2012 (1-2), Figure 9, Table 11) were excluded (g=-0.18, se=0.28, CIs (95%)=[-0.87,0.50], t=-0.65, p=0.54). Heterogeneity was high (I^2=94.28%) and publication bias was significant (Tau=0.67, p=0.01, Figure 9). The two studies that could not be included in meta-analysis also reported no association between the feedback-P3 and ADHD (Table 11).

[Table 11]

[Figure 9]

3.4.5 Other performance monitoring components in ADHD

Other components examined in ADHD were error-related theta activity (two studies), the feedback-N1 (one study), the feedback-P2 (three studies) and the feedback-LPP (four studies) (Table 12). These studies were included in narrative synthesis since the sample sizes were too small for meta-analysis. The two studies investigating error-related theta activity reported significantly reduced theta power (Groom et al., 2010; Keute et al., 2019) and inter-trial theta phase coherence (Groom et al., 2010) in ADHD compared to controls. The three studies examining the feedback-P2 reported mixed findings of no difference in amplitude between ADHD and controls (Groen et al., 2013; Shephard et al., 2016a) and a lack of learning-related

reduction in feedback-P2 amplitude associated with ADHD (Groen et al., 2008; Shephard et al., 2016a). The feedback-N1 did not differ between ADHD and controls (van Meel et al., 2005). The feedback-LPP was significantly negatively correlated with ADHD symptoms in one study (Althaus et al., 2010) but was not associated with ADHD in the remaining three studies (Gong et al., 2014; Groen et al., 2013; van Meel et al., 2011).

[Table 12]

3.5 Autism

3.5.1 ERN in autism

Of the 21 studies investigating performance monitoring components in autism, fifteen studies examined the ERN (Table 13). Effect sizes could be computed for all 15 studies, of which one study reported the ERN in more than one experimental condition. All studies except one were conducted with children and adolescents rather than adults. Meta-analysis on 18 effect sizes from the 15 studies showed significantly reduced ERN amplitude in autistic participants compared to controls (g=-0.61, se=0.23, CIs (95%)=[-1.10,-0.13], t=-2.66, p=0.02, Figure 10). These findings did not change when one outlying effect size (McMahon et al., 2015 (4), Figure 10, Table 13) was excluded (g=-0.36, se=0.14, CIs (95%)=[-0.65,-0.06], t=-2.56, p=0.02). Heterogeneity was high (I^2=91.95%) but publication bias was not significant (Tau=-0.19, p=0.29, Figure 10). Study quality rating was not a significant moderator of the pooled effect size (F(1,16)=2.25, p=0.15). Developmental stage and conversion of effect size were not examined as moderators due to insufficient variability in these factors across studies (Table 13).

[Table 13]

[Figure 10]

3.5.2 Pe in autism

Ten studies investigated the Pe in autism (Table 14). Effect sizes could be computed for four of these studies. All studies except one were conducted with children or adolescents. Meta-analysis on four effect sizes from the four studies revealed no significant difference in Pe amplitude between autistic and non-autistic participants (g=-0.29, se=0.26, CIs (95%)=[-1.13,0.54], t=-1.12, p=0.35, Figure 11). Heterogeneity was moderate (I^2=49.7%) and publication bias was not significant (Tau=0.33, p=0.75, Figure 11). The six studies not included in the meta-analysis also reported no Pe amplitude alterations associated with autism (Table 14).

[Table 14]

[Figure 11]

3.5.3 FRN in autism

Five studies investigated the FRN in autism (Table 15). Effect sizes could be computed for four of the studies, with one of the four studies reporting the FRN in more than one experimental condition. All studies except one were conducted with children or adolescents rather than adults. Meta-analysis on five effect sizes from the four studies was attempted but the model did not converge. Due to the small sample size and the lack of convergence of the model, all studies investigating the FRN in autism were included in narrative synthesis only. A descriptive summary of these studies is shown in Table 15. Two of these studies reported significantly smaller FRN amplitude in autistic compared to non-autistic participants (Bellebaum et al., 2014; Larson et al., 2011) while another study reported no differences in FRN related to autism (McPartland et al., 2012). A further two studies reported reduced (Gonzalez-Godea et al., 2016) or increased (Stavropoulos et al., 2014) FRN in conditions in which social feedback was used in autistic participants compared to controls but no differences in FRN when non-social (monetary) feedback was used.

[Table 15]

3.5.4 Other performance monitoring components in autism

Other components examined in autism were the feedback-P2 (two studies), the feedback-P3 (four studies), the feedback-LPP (one study) and feedback-locked theta power and inter-trial phase coherence (one study). Due to the small sample sizes, these studies were included in narrative synthesis only. A descriptive summary of the studies is shown in Table 16. The feedback-P2 did not differ between autistic and non-autistic participants in either of the studies (Groen et al., 2008; McPartland et al., 2012). Two of four studies reported no autism-related alterations in the feedback-P3 (Bellebaum et al., 2014; Larson et al., 2011) while the other two studies reported reduced effects of feedback type (positive vs negative feedback) associated with autism (Althaus et al., 2010; Groen et al., 2008). Feedback type effects on the feedback-LPP were also associated with autistic traits in the one study that investigated this component (Althaus et al., 2010). One study investigated feedback-locked theta activity and reported no differences in theta power but significantly reduced theta inter-trial phase coherence in autism compared to controls (van Noordt et al., 2017).

[Table 16]

3.6 Co-occurring presentations of OCD, GTS, ADHD or autism

Three studies investigated electrophysiological markers of performance monitoring in participants with co-occurring presentations of OCD, GTS, ADHD or autism. All studies were conducted with children and adolescents rather than adults. Two studies focused on co-occurring GTS and ADHD (Shephard et al., 2016a, 2016b) and one study on OCD and co-occurring tics (Hanna et al., 2012). A narrative synthesis of these studies is presented in Table 17. Note that the

findings from participants without the co-occurring condition (e.g. GTS without co-occurring ADHD) were included in the sections above focused on OCD, GTS, ADHD and autism.

One study investigated the ERN and Pe in relation to the co-occurrence of GTS and ADHD (Shephard et al., 2016b) and reported significantly smaller ERN and Pe amplitudes on error trials in children and adolescents with ADHD with and without co-occurring GTS compared to those without ADHD with or without GTS. The other study investigating co-occurring GTS and ADHD (Shephard et al., 2016a) focused on the FRN and feedback-locked P2 during a learning and reversal task. They found smaller feedback-P2 amplitude in children and adolescents with GTS without ADHD compared to those with GTS and co-occurring ADHD and control participants, but no differences in FRN. Moreover, among young people with GTS, those with more severe ADHD symptoms showed larger feedback-P2 and FRN amplitudes during key task phases when learning and reversal were most difficult and required greater reliance on external feedback. The study examining OCD and co-occurring tics (Hanna et al., 2012) found increased ERN amplitude in children with OCD with and without co-occurring tics compared to controls, and increased ERN in OCD without co-occurring tics compared to OCD with tics.

[Table 17]

4. Discussion

4.1 Increased electrophysiological correlates of internal and external performance monitoring in OCD

The current findings confirm those from previous meta-analyses (Moser et al., 2016; Riesel, 2019) showing that amplitude of the ERN is increased during error commission in individuals with OCD compared to participants without OCD. Given that the ERN is generated

by the ACC, these findings are also consistent with a recent meta-analysis of fMRI studies, which reported significantly increased ACC activity during error processing in OCD (Norman et al., 2019). Further, similar to Riesel's (2019) findings, the current meta-analysis showed that increased ERN in OCD is not influenced by developmental stage, indicating that both paediatric and adult forms of the condition are associated with overactive error monitoring, although considerably fewer studies have been conducted with children and adolescents compared to adults. These findings provide further support for the idea that overactive error detection by the ACC is a key neurocognitive alteration in OCD (Endrass & Ullsperger, 2014; Gehring et al., 2000; Nieuwenhuis et al., 2005b; Norman et al., 2019; Riesel, 2019). However, it should be noted that cross-study heterogeneity was considerable. This may reflect variability in the tasks used to measure the ERN across studies, given Riesel's (2019) finding that the ERN increase in OCD was present for cognitive control tasks but not for other tasks. Alternatively, the cross-study heterogeneity may reflect inter-individual variability in the neurocognitive mechanisms involved in OCD. Indeed, it is unlikely that all individuals with OCD will show the same neurocognitive alterations, as highlighted by recent models of the neurobiology of the condition (Dougherty et al., 2018; Shephard et al., 2021a; van den Heuvel et al., 2016). Further, publication bias was significant, suggesting that studies reporting larger effect sizes were more likely to have been published and consequently the current pooled effect size may be an overestimate.

Importantly, the current meta-analyses indicate that overactive error monitoring extends beyond the detection of errors by the ACC in OCD. The Pe component, thought to be generated by the insula and linked with the evaluation and correction of errors (Dhar et al., 2011; Hajcak et al., 2003; Ullsperger et al., 2010), was also significantly increased in OCD with a similar pooled

effect size to that of the ERN. These findings suggest that individuals with OCD also have difficulties in evaluating errors they have made, perhaps getting "stuck" in determining the importance of the error and deciding how the error should be corrected, or by overestimating the significance of the error. It should be noted, however, that considerably fewer studies contributed to the meta-analysis of the Pe (9 studies) than to the ERN (39 studies) and further work examining the Pe in OCD is needed to confirm these findings. Nevertheless, overactive insula function during error commission has been reported in fMRI studies of OCD (e.g. Huyser et al., 2011; Stern et al., 2011) and a recent fMRI meta-analysis confirmed that significantly increased neural activity during errors was found in both ACC and the insula in OCD (Norman et al., 2019).

In addition to increased ERN and Pe correlates of internal performance monitoring, the current meta-analyses also revealed significantly increased FRN amplitude in OCD, a correlate of external performance monitoring. The pooled effect size for the FRN was of similar magnitude to those of the ERN and Pe, but was based on fewer studies than the ERN. This finding will therefore require confirmation in future when more studies investigating the FRN in OCD have been published. Still, it is perhaps unsurprising that individuals with OCD show increased FRN given that the same neural mechanism is thought to underlie the ERN and FRN, i.e. the detection of mismatches in prediction-action outcomes by the ACC (Holroyd & Coles, 2002; Ullsperger et al., 2014; Yeung et al., 2004). The current findings suggest that this mismatch detection mechanism is overactive in OCD, regardless of the way in which the information concerning the mismatch is perceived (i.e. internally and externally).

Much of the previous work in OCD has focused exclusively on the ERN, with several authors suggesting that increased ERN could represent an endophenotypic marker of OCD (e.g.

Riesel, 2019; Riesel et al., 2011; Riesel et al., 2015). One previous systematic review that did include studies investigating other correlates of performance monitoring, such as the FRN, concluded that overactive performance monitoring is limited to the ERN in OCD (Endrass & Ullsperger, 2014). In contrast to this previous work, the pattern of findings from the current meta-analyses implicate a more widespread overactivity in performance monitoring in OCD, involving both the ACC and insula and internal and external monitoring. An important avenue for future work will be to examine how altered performance monitoring is related to the phenomenology of OCD symptoms, such as reassurance-seeking from others (perhaps related to increased external performance monitoring) and uncertainty concerning whether a compulsion was performed correctly or an obsession is valid (perhaps associated with internal performance monitoring).

4.2 Increased electrophysiological correlates of error detection in GTS

The current systematic review highlighted that few studies have investigated electrophysiological correlates of performance monitoring in GTS. The only component investigated in a sufficient number of studies to be included in meta-analysis was the ERN, which was significantly increased in amplitude in participants with GTS compared to participants without GTS, with a large pooled effect size (g=0.99). However, this was based on only five studies and cross-study heterogeneity was high. This finding should therefore be interpreted cautiously until further studies investigating the ERN in GTS have been conducted. In contrast to our predictions, the heterogeneity was not explained by the subgroup factor developmental stage, indicating that cross-study differences in ERN alterations were not

explained by differences between children/adolescents compared to adults with GTS, although this may reflect the small number of studies and lack of power to detect moderating effects.

While the current findings should be considered preliminary due to the small number of studies subjected to meta-analysis, they provide initial support for the idea that error detection mediated by the ACC is increased in GTS. However, whether this reflects overactivity in performance monitoring that contributes to tic symptoms, similar to the overactive monitoring mechanism thought to contribute to OCD symptoms, or increased engagement of performance monitoring to facilitate tic control, is unclear. In support of the latter proposal, a recent fMRI study found that increased ACC activity mediated successful tic suppression in GTS (van der Salm et al., 2018).

Narrative synthesis of studies investigating other correlates of performance monitoring in GTS revealed mixed findings of reduced Pe amplitude in one study (Schüller et al., 2018) and unaltered Pe amplitude in the other two studies (Eichele et al., 2016; Shephard et al., 2016b). It should be noted that these studies reported the same pattern for the ERN: increased ERN amplitude in GTS in Schüller et al. (2018) and unaltered ERN amplitude in GTS in Eichele et al. (2016) and Shephard et al. (2016b). These contrasting findings likely did not reflect age-related differences in performance monitoring since all three studies were conducted with children and adolescents. Interestingly, consistent with the increased Pe amplitude reported by Schüller et al. (2018), a recent fMRI study reported hyperactivity in a network of regions involved in error monitoring in GTS, which included the insula (Fan et al., 2018). Only one study examined correlates of external performance monitoring in GTS and reported no significant differences in the FRN or feedback-P2 associated with GTS in children and adolescents (Shephard et al., 2016a). Given the small number of studies investigating the Pe and feedback-locked correlates of

performance monitoring, it is not possible to draw conclusions concerning the presence or absence of alterations in GTS.

It will be important for future work examining performance monitoring in GTS to try to distinguish whether enhancements in this process reflect the engagement of mechanisms to facilitate tic control or whether they play a contributing role in the generation of tics. It is known that tic symptoms worsen during periods of stress (Conelea et al., 2011; Silva et al., 1995). Thus, one possibility for future experiments would be to compare performance monitoring under conditions of (mild and appropriate) stress and under non-stressful conditions. A review on the role of stress in tics highlighted four stress factors that are known to exacerbate tics, including atypically high or low sensory stimulation, frustrating or anger-inducing situations, anxiogenic stimuli, and fatigue / sleep loss (Godar & Bortolato, 2017). At least some of these factors can be experimentally manipulated without adverse consequences for the participant. For example, long and boring tasks or those with feedback suggesting the participant performed too poorly to earn a reward elicit frustration (e.g. Deveney et al., 2013). Moreover, the urge to tic can be measured with rating scales (Woods et al., 2005). If performance monitoring enhancements are involved in tic control, one could expect these to be present particularly under conditions of stress when the urge to tic is rated as higher and in which greater control of tics is required.

4.3 Reduced electrophysiological correlates of internal but not external performance monitoring in ADHD

The current systematic review and meta-analysis extended previous meta-analytic work (Geburek et al., 2013; Kaiser et al., 2020) by including a larger number of studies investigating electrophysiological correlates of performance monitoring in ADHD. Results of the current

meta-analyses revealed significantly reduced ERN and Pe amplitude in children and adults with ADHD compared to participants without ADHD, indicative of difficulties in detecting (ERN) and evaluating (Pe) errors in behaviour. These EEG findings are consistent with previous fMRI meta-analyses showing significantly reduced functional activity in the ACC and insula in ADHD (Hart et al., 2013; Norman et al., 2016; see also Rubia et al., 2011), which are involved in the generation of the ERN and the Pe, respectively. The pooled effect sizes for the ERN and Pe (both gs ~ -0.50) were not moderated by age, suggesting that underactive or hypofunctioning internal performance monitoring mechanisms are present across developmental stages in ADHD. Evidence of weaker internal performance monitoring in ADHD also emerged from our narrative synthesis of studies that could not be included in the meta-analyses. These studies reported reduced error-related theta power and inter-trial theta phase coherence in people with ADHD. Cross-study heterogeneity was high for the ERN and Pe, though publication bias was not significant. High heterogeneity has been reported in meta-analyses of many other neurocognitive functions in ADHD (e.g. Huang-Pollock et al., 2012; Shephard et al., 2021b) and might reflect variation in the clinical or neurocognitive presentation of the condition and/or co-occurring symptoms of other conditions, which may not have been thoroughly assessed in the studies included in our meta-analyses.

To our knowledge, our meta-analysis is the first to include electrophysiological indices of external performance monitoring in ADHD. The meta-analyses of the FRN and feedback-P3 indicated no significant differences between ADHD and controls in either component. These findings suggest that while mechanisms of internal performance monitoring (indicated by the ERN and Pe) appear to be weaker in ADHD, performance monitoring based on external cues (indexed by the FRN and feedback-P3) may be unaltered. It should be noted, though, that some

atypicalities in other feedback-locked correlates of performance monitoring were found to be associated with ADHD in narrative synthesis, but too few of these studies were available to be included in meta-analyses. For example, two studies reported that individuals with ADHD did not show a reduction in feedback-P2 amplitude during learning, while individuals without ADHD showed a significant decrease in this component as learning progressed across trials (Groen et al., 2008; Shephard et al., 2016a). These findings might suggest that individuals with ADHD have difficulty with external performance monitoring when feedback must be used to adapt and guide future behaviour (i.e., in the context of learning).

However, several studies included in the meta-analysis of the FRN and the feedback-P3 also examined these components during learning, and the pooled effect sizes were non-significant. Still, as noted, cross-study heterogeneity for the FRN and feedback-P3 was high, reflecting large variation in effect sizes reported in individual studies included in the meta-analyses. It may be the case that the FRN and/or feedback-P3 do show alterations in ADHD during learning contexts similar to the feedback-P2, but there were too few published studies to examine experimental task as a moderator of the pooled effect sizes for the FRN and feedback-P3. This will be an important step for further research. It should also be highlighted that a larger number of studies investigated the ERN/Pe (28/26) than FRN/feedback-P3 (11/6) and therefore further studies are needed to confirm our preliminary findings of atypical internal but typical external performance monitoring in ADHD.

Overall, underactive or weaker performance monitoring in ADHD, especially when external cues are not provided and one is required to internally monitor one's own performance, is likely to cause difficulties in regulating behaviour to meet the demands of an ongoing task or activity. Being less able to internally detect and process errors is likely to cause reduced

vigilance and poorer behavioural adaptation in ADHD, with consequent worsening of performance accuracy and difficulties in self-regulation (Sergeant et al., 2003; Shiels & Hawk, 2010). In learning environments such as at school, it might be beneficial for people with ADHD to obtain external feedback on their performance instead of having to rely on internal monitoring when performing a task or carrying out an activity. School- and home-based interventions should therefore consider including activities that help people with ADHD to integrate internal and external cues to assess their performance and behaviour, since this might help to increase self-regulation in different situations of everyday life. Still, further work is needed to clarify in which situations external, feedback-based performance monitoring is and is not altered in individuals with ADHD.

4.4 Reduced electrophysiological correlates of error detection in autism

As far as we are aware, there has been no previous meta-analysis of electrophysiological correlates of performance monitoring in autism. The current meta-analysis of the ERN revealed significantly reduced amplitude in autistic participants compared to non-autistic controls with a moderate-to-large pooled effect size, indicative of reduced error detection in autism. There was no evidence of publication bias but cross-study heterogeneity was high, which may reflect the large phenotypic variability in autism and possibly also the fact that the ERN was measured in six different experimental tasks across 15 studies. In contrast, meta-analysis of the Pe was not significant, suggesting no differences in evaluating errors or initiating corrective strategies between autistic and non-autistic participants. This pattern of findings is consistent with meta-analyses of fMRI studies in autism reporting consistent hypoactivity of the ACC (which generates the ERN) across a range of neurocognitive task conditions but more limited

hypoactivity of the insula (which generates the Pe) that occurs mainly during social contexts (Di Martino et al., 2009). It should be noted that the meta-analysis of the Pe was based on only four studies, but the findings were supported by narrative synthesis of six other studies not included in the meta-analysis, which also reported no Pe alterations in autism. Most studies investigating the ERN and Pe were conducted with children and adolescents (except for one in adults) and therefore it is not clear if reduced ERN and typical Pe are also seen in autistic adults.

Few studies have investigated feedback-locked correlates of external performance monitoring in autism. Narrative synthesis of five studies investigating the FRN yielded mixed results, with two of the studies reporting reduced FRN amplitude in autism and the remaining studies reporting no alterations in FRN or FRN increases or reductions only when social rather than non-social stimuli were used as feedback. More consistent findings were reported for the feedback-P3, with reduced amplitude in autism in two of four studies and reduced differentiation in amplitude for positive vs. negative feedback in the other two studies. These findings should be considered preliminary due to the small number of studies included, but they might suggest that some autistic individuals have difficulties with detecting mismatches between predicted outcomes of behaviour and external performance feedback (indicated by reduced FRN in some studies), especially when feedback is socially oriented, and in using external feedback information to guide future behaviour (indicated by reduced feedback-P3). Importantly, however, not all autistic individuals appear to experience this difficulty.

Previous authors have suggested that weaker or less efficient performance monitoring might result in difficulties in detecting and adjusting suboptimal behaviour in different situations of everyday life and in turn this contributes to the inflexible, restricted and repetitive behaviour symptoms of autism (D'Cruz et al., 2016; Mundy et al., 2003). The current findings provide

some support for the involvement of reduced error detection in autism, but there is little evidence for extensive difficulties in performance monitoring in autistic individuals. It is also not clear if reduced error detection indicated by smaller ERN amplitude relates specifically to restricted, repetitive and inflexible autistic symptoms. Further empirical studies are needed to clarify the impact of different factors such as age, sex and co-occurring conditions on electrophysiological correlates of performance monitoring in autism, and to extend the current literature by involving autistic adults with heterogeneous behavioural and symptomatologic profiles as well as autistic children.

4.5 Cross-condition comparison

A key goal of the current work was to compare alterations in performance monitoring between OCD, GTS, ADHD and autism to better understand whether atypicalities might be shared across any or all of these frequently co-occurring neurodevelopmental conditions. Our findings revealed increased ERN amplitude in both OCD and GTS. Further, the one study that examined performance monitoring in individuals with OCD and co-occurring tics (Hanna et al., 2012) also reported similarly increased ERN in children with OCD both with and without co-occurring tics compared to controls without OCD or tics. These findings suggest some degree of overlap in neurocognitive mechanisms between OCD and GTS, specifically in terms of shared enhancements in the detection of errors mediated by the ACC. This finding is important because increased ERN is frequently cited as an endophenotypic marker of OCD (see e.g. Riesel, 2019), but the current findings indicate a lack of specificity of this marker to OCD. While recent work by Riesel and others (Gillan et al., 2017; Riesel et al., 2019a) highlights that increased ERN may act as a transdiagnostic marker for OCD and anxiety disorders, to our knowledge no work has emphasised that the ERN may also represent a shared neurocognitive atypicality in OCD and

GTS. Similarly, neurobiological models of OCD that consider the overlap in neurocognitive and neurocircuit alterations between OCD and GTS mainly focus on shared atypicalities in neurocognitive functions associated with sensorimotor and reward circuitry, such as sensory phenomena and excessive habit formation (Robbins et al., 2012; Robbins et al., 2019; Shephard et al., 2021a). These models may need to be updated to include overactive error detection and ACC function as an additional commonality.

In contrast to the findings in OCD and GTS, the current work revealed similarly reduced ERN amplitude across ADHD and autism, suggesting that reduced functioning of the ACC in the context of detecting errors in behaviour may represent a shared neurocognitive alteration between these conditions. This finding is interesting since previous EEG studies attempting to identify shared neurocognitive alterations between ADHD and autism have mainly reported distinct atypicalities that were associated with ADHD but not autism or vice versa (e.g., Bellato et al., 2021; Groom et al., 2017; Tye et al., 2013, 2014; Shephard et al., 2019), though the ERN was not examined in those studies. Yet, some models of the co-occurrence of ADHD and autism do suggest that these conditions share neurocognitive mechanisms (Rommelse et al., 2010). In our systematic search of the literature, we did not find any studies that investigated electrophysiological correlates of performance monitoring in people with co-occurring ADHD and autism. Further work is therefore needed to clarify whether and how the co-occurrence of these conditions is likely to affect performance monitoring. Previous work investigating other aspects of neurocognitive function suggest that ADHD and autism have additive effects, with individuals with both conditions showing neurocognitive alterations associated with both ADHD and autism (Tye et al., 2013, 2014; Shephard et al., 2019).

It is also important to highlight the opposing alterations in error detection across the four neurodevelopmental conditions, with increases in OCD and GTS and reductions in ADHD and autism. This pattern of findings might suggest that OCD and GTS are more similar to one another in terms of neurobiology than to ADHD and autism, and vice versa for ADHD and autism compared to OCD and GTS. Yet, all four of these conditions frequently co-occur (Jensen et al., 2015; Lai et al., 2019; Lebowitz et al., 2012; Martin et al., 2018). Further work will be needed to better understand how contrasting alterations in error detection manifest in individuals with OCD or GTS and co-occurring ADHD or autism. The one study that has investigated this in GTS and co-occurring ADHD suggests that reduced error detection associated with ADHD might cancel out increased error detection associated with GTS, although the participants with GTS without ADHD did not show increased error detection either (Shephard et al., 2016b). This issue may be particularly important in the case of OCD because enhanced error monitoring indexed by the ERN has been suggested as an endophenotypic marker (Riesel, 2019) and as a treatment target (Carmi et al., 2018). Understanding how co-occurring ADHD and autism affect error detection and ACC function in OCD may therefore have clinical implications in that individuals with OCD and co-occurring ADHD or autistic symptoms may not be identifiable based on enhanced ERN and hence treatment targeted at reducing ERN-indexed ACC hyperactivity may not be appropriate.

Another striking difference revealed by comparison of the meta-analyses of the four conditions was the lower cross-study heterogeneity in OCD (23-43%) compared to ADHD (66-93%), autism (50-92%) and GTS (77%). These findings indicate that cross-study variability in experimental tasks used and different participant samples as well as clinical and demographic characteristics may have less influence on altered performance monitoring in OCD than they do

in the other three conditions. Further work will be needed to identify which factors in ADHD, autism and GTS influence performance monitoring. It will also be important to investigate how other neurocognitive or neurobiological atypicalities associated with these conditions, such as sensory processing, autonomic arousal and broader aspects of self-regulation, affect performance monitoring alterations. A better understanding of how increases and decreases in electrophysiological correlates of performance monitoring relate to real-life cognition and behaviour is also needed.

Overall, our findings suggested dysregulated functioning (either overactive or underactive) of the ACC in all four neurodevelopmental conditions under study. This is in line with recent work reporting that the ACC acts as a connectivity "hub" in the brain (Tang et al., 2019). Hubs are regions that are characterised by particularly dense connections with many other brain regions (van den Heuvel & Sporns, 2013). It has been suggested that the ACC, because of its hub-like nature, may be in a position to mediate function (and dysfunction) in many other brain networks and could act as a transdiagnostic neural alteration in several neurodevelopmental and psychiatric conditions (Tang et al., 2019). The current findings of shared increases or reductions in the ACC-mediated ERN across four common neurodevelopmental conditions are in line with this proposal.

4.6 Limitations

The current work should be considered in light of several limitations. First, the number of studies included in the meta-analyses of several components was small and consequently the pooled effect sizes for these should be considered preliminary until further studies have been conducted and meta-analyses can be repeated. Although we did not find a moderating effect of

developmental stage on any of the components included in meta-analyses, there was some imbalance in terms of the age ranges of participants across the conditions under study. Specifically, most studies in autism focused on children and adolescents while most studies on OCD focused on adults. Due to a lack of published studies, the feedback-locked correlates of external performance monitoring could only be included in meta-analysis of OCD and ADHD and not in GTS and autism.

While we were able to focus the meta-analyses of ADHD on participants who were not taking stimulant medication at the time of the EEG, we were not able to investigate effects of other medications on the pooled estimates for any of the neurodevelopmental conditions due to inconsistent reporting and lack of information on medication status in many studies. We also did not examine behavioural measures of performance monitoring. This was because few studies reported specific behavioural correlates of performance monitoring (e.g. post-error slowing) and general measures of task performance such as accuracy and reaction time are difficult to attribute to performance monitoring capacity rather than other aspects of cognitive control. However, this is an important limitation because increased or decreased ERN amplitude (for example) could reflect different alterations in performance monitoring in the context of better or worse behaviour.

Concerning the cross-condition and co-occurring condition comparisons, the studies included in our systematic review and meta-analyses recruited a maximum of one or two samples of participants with different clinical diagnoses of the four conditions under study, and only three studies recruited participants defined as having co-occurring symptoms or ‘double’ diagnoses. Further, while we extracted information on co-occurring conditions for every study included in this review, this information was often not reported or described in a way that could not be

quantified for meta-analysis across studies. Thus, it is likely that many of the participants included in analysis of the components in each condition also had symptoms or diagnoses of the other conditions as well. Future studies comparing participants with these different conditions as well as co-occurring presentations are therefore needed to confirm our findings and interpretations. A useful approach may be to adopt dimensional research frameworks, such as the NIH Research Domain Criteria (RDoC, Cuthbert and Insel, 2013), and dimensional classification systems, such as the Hierarchical Taxonomy of Psychopathology (HiTOP, Kotov et al., 2017), rather than or as well as comparing groups of participants with specific diagnoses. A dimensional approach would help to clarify whether performance monitoring alterations are associated with the restricted, repetitive, inflexible, compulsive or impulsive symptoms across OCD, GTS, ADHD and autism. Combining RDoC and HiTOP systems has recently been suggested as a particularly useful method of identifying the underlying neurocognitive and neurobiological correlates of symptomatologic domains that are shared between different neurodevelopmental and psychiatric conditions (Michelini et al., 2021).

5. Conclusions

We conducted the first systematic review and meta-analysis of a range of electrophysiological correlates of internal (ERN, Pe) and external (FRN, feedback-P3) performance monitoring in OCD, GTS, ADHD and autism. We found evidence of atypically increased ERN amplitude in OCD and GTS and atypically reduced ERN amplitude in ADHD and autism. These findings suggest shared increases in error detection in OCD and GTS and shared reductions in error detection in ADHD and autism. OCD was additionally associated with significantly increased Pe and FRN amplitudes, indicative of more extensive performance

monitoring overactivity. ADHD was also associated with reduced Pe, indicative of difficulties in evaluating errors in performance, but no consistent atypicalities in feedback-locked correlates of external performance monitoring.

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