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Review

Electrophysiology of obsessive compulsive disorder: A systematic review of the electroencephalographic literature

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ABSTRACT

Obsessive-compulsive disorder (OCD) is a chronic disease that causes significant decline in the quality of life of those affected. Due to our limited understanding of the underlying pathophysiology of OCD, successful treatment remains elusive. Although many have studied the pathophysiology of OCD through electroencephalography (EEG), limited attempts have been made to synthesize and interpret their findings. To bridge this gap, we conducted a comprehensive literature review using Medline/PubMed and considered the 65 most relevant studies published before June 2018. The findings are categorised into quantitative EEG, sleep related EEG and event related potentials (ERPs). Increased frontal asymmetry, frontal slowing and an enhancement in the ERP known as error related negativity (ERN) were consistent findings in OCD. However, sleep EEG and other ERP (P3 and N2) findings were inconsistent. Additionally, we analysed the usefulness of ERN as a potential candidate endophenotype. We hypothesize that dysfunctional frontal circuitry and overactive performance monitoring are the major underlying impairments in OCD. Additionally, we conceptualized that defective fronto-striato-thalamic circuitry causing poor cerebral functional connectivity gives rise to the OCD behavioural manifestations. Finally, we have discussed transcranial magnetic stimulation and EEG (TMS-EEG) applications in future research to further our knowledge of the underlying pathophysiology of OCD.

1. Introduction

Obsessive compulsive disorder (OCD) is a psychosocially debilitating condition that causes significant reduction in the quality of life of those affected (Angelakis, Gooding, Tarrier, & Panagioti, 2015; Richter, Summerfeldt, Antony, & Swinson, 2003). The proposed lifetime prevalence of OCD ranges from 2 to 3% (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012; Ruscio, Stein, Chiu, & Kessler, 2010). As described in the DSM-5 (American Psychiatric Association, 2013), if left untreated, OCD is typically a chronic disease with waxing and waning symptoms.

Existing treatment modalities for OCD, including Selective Serotonin Re-uptake Inhibitors (SSRIs) and Cognitive Behaviour Therapy (CBT), often fail to achieve a clinically significant improvement of symptoms (Alarcon, Libb, & Spitler, 1993; Ballenger, 1999; Taylor, Abramowitz, & McKay, 2012). This inadequacy has motivated researchers worldwide to broaden their investigations in order to further our understanding of the underlying pathophysiology of OCD.

Over the past few decades, a myriad of electrophysiological studies have discovered various neurobiological changes that appear to be related to the behavioural and psychological deficits of OCD. Electroencephalography (EEG) is an affordable and effective method to analyse the electrophysiology of the brain. We have categorized the eligible studies from the literature on EEG changes in OCD into three broad areas, namely, quantitative electroencephalography (QEEG), sleep related EEG changes and abnormalities in event related potentials (ERP).

QEEG is a method of EEG signal acquisition, followed by refined signal processing, mathematical transformations, advanced data analysis and large database comparisons. This facilitates the precise measuring of various EEG parameters (Tong & Thakor, 2009). Sleep related EEG signals are acquired during sleep via clinical polysomnography and digitally analysed to identify the sleep microstructure (Kubicki & Herrmann, 1996). Event related potentials (ERP) denote voltage changes that occur specifically related to the brain's response to a stimulus and are expressed through EEG epochs that are time locked to a

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stimulus (Coles & Rugg, 1995).

Several previous reviews have contributed towards some of the subtopics that we discuss in this review (Endrass & Ullsperger, 2014; Nota, Sharkey, & Coles, 2015). However, the current review is the first to compile all EEG related areas together to synthesize the broad knowledge base of electrophysiological research in OCD. This review presents an exhaustive literature evaluation on electrophysiological anomalies linked with OCD with an emphasis on the degree of consistency between studies. To the authors' knowledge, this is the first in-depth review that comprehensively analyses all the relevant components of electrophysiology in OCD sufferers.

2. Methods

A comprehensive search of the relevant literature was performed using the electronic database of MEDLINE/PubMed. Cross-sectional studies published in English up to June 2018 were included. The search terms used were obsessive-compulsive disorder, OCD, electroencephalography, EEG, quantitative electroencephalography, QEEG, polysomnography, event related potentials, ERP. Since OCD affects both paediatric and adult populations, studies on both were included. The inclusion criteria were: (i) full length original articles published in peer-reviewed journals in English language, (ii) patients with OCD according to DSM or ICD-10 criteria, (iii) the article included a detailed description of the EEG measurement method and results, (iv) the study involved analysing EEG in individuals with OCD with or without a comparison sample of healthy controls. Initially, all the relevant articles were included via inspection of the title and the abstract. Full text versions were then assessed in more detail and only the most pertinent articles were included for review. The reference lists of the selected articles were manually searched for additional relevant articles. The study selection process is summarized in Fig. 1. The final eligible list of studies included 65 studies. The following general information was recorded from the 65 studies: (a) year of publication, (b) EEG technique, and (c) key findings from their EEG recordings. Characteristics of participants in each study were recorded as follows: (a) sample size (for experimental and control groups), (b) mean age, (c) male:female ratio in the sample/group. The studies were categorized under our three main groups as QEEG, sleep related EEG changes and abnormalities in ERPs. We then analysed the results of studies under each category in a qualitative manner to explore how each of these differences may illuminate the pathogenesis of OCD.

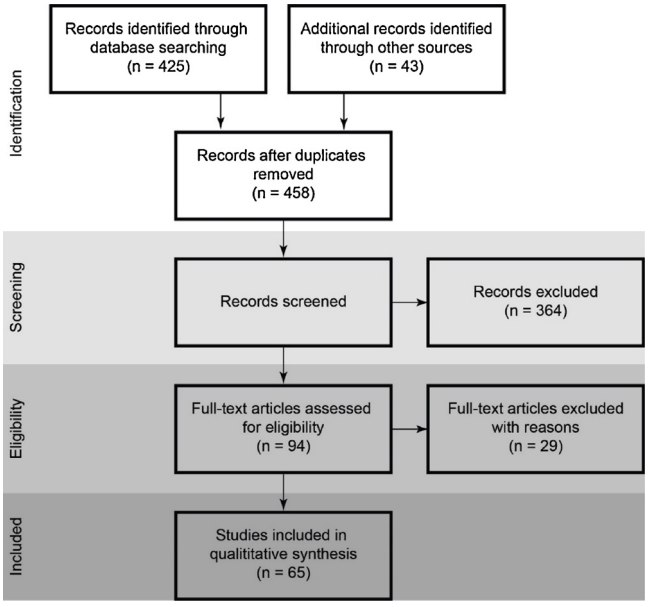

Fig. 1. Study selection process. This diagram depicts the flow of information through the different phases of our systematic review. It maps out the number of records identified, included and excluded.

3. Quantitative EEG in OCD

QEEG has been utilized in a growing number of studies assessing various disorders (Sun et al., 2011; Wickramasuriya, Wijesinghe, & Mallawaarachchi, 2015) including OCD. The OCD related QEEG findings are summarized in the following sections and in Table 1.

3.1. Frontal asymmetry

Frontal asymmetry is characterized by higher powers of neural oscillations in one hemisphere compared to the other and is a neurofunctional characteristic linked with several psychiatric disorders (Jetha, Schmidt, & Goldberg, 2009; Segrave et al., 2011). Frontal asymmetry has also been associated with OCD in many studies. The common forms of asymmetries include, left sided increase in frontal alpha (Desarkar, Sinha, Jagadheesan, & Nizamie, 2007; Ischebeck, Endrass, Simon, & Kathmann, 2014) and theta (Kopřivová et al., 2011; Perros, Young, Ritson, Price, & Mann, 1992; Tot, Özge, Çömelekoğlu, Yazici, & Bal, 2002) band powers. Asymmetries in other band forms have also been reported (Bolwig, Hansen, Hansen, Merkin, & Prichep, 2007; Kuskowski et al., 1993; Molina et al., 1995; Velikova et al., 2010). However, a few studies failed to reproduce this phenomenon (Drake, Pakalnis, & Newell, 1996; Grützmann et al., 2017; Karadag et al., 2003). This ambiguity could be due to the nature of frontal asymmetry, which is known to reflect underlying processes of psychiatric disorders such as, excessive avoidance (e.g. – anxiety, depression) or approach (e.g. – substance abuse, mania), rather than the disorders themselves (Davidson, 1998). Therefore, the presence or absence of frontal asymmetry might depend on common comorbidities of OCD such as depression or substance use (Zitterl et al., 2000). An alternative theory is that individuals with OCD show a combination of avoidance to certain situations and approach to specific behaviours (compulsions) (Najmi, Kuckertz, & Amir, 2010). An imbalance in this approach-avoidance process could potentially lead to frontal asymmetry (Harmon-Jones, 2003).

3.2. Deviations in delta activity

Significantly higher resting, overall delta activity (Desarkar et al., 2007a; Kamaradova et al., 2016; Karadag et al., 2003; Molina et al., 1995; Pogarell et al., 2006; Tot et al., 2002) and delta-1 (1–1.9 Hz) activity (Locatelli, Bellodi, Grassi, & Scarone, 1996) are found in OCD patients, compared to healthy controls, predominantly seen over frontotemporal regions. It is noteworthy that the higher delta activity reported in these studies was not accompanied by higher activity in other band forms, suggesting that neural oscillations are slowed in OCD without an increase in power across the spectrum. It is postulated that frontal slowing of this nature can be explained by the activation of frontal generators of delta activity (Michel, Lehmann, Henggeler, & Brandeis, 1992). Further localization was accomplished by analysing EEG data via standardized low-resolution electromagnetic tomography (sLORETA) in two studies, which observed this phenomenon to be confined to the insula (Kamaradova et al., 2016; Velikova et al., 2010). In contrast, significantly reduced delta power was observed in two studies, the results of which may be unreliable due to the small sample size (Kuskowski et al., 1993) and the absence of a control group (Hansen, Prichep, Bolwig, & John, 2003).

3.3. Deviations in theta activity

3.3.1. Resting state theta activity

When compared to healthy controls, increased resting frontotemporal overall theta (Desarkar et al., 2007a; Desarkar, Sinha,

Table 1
Summary of quantitative electroencephalographic findings in individuals with obsessive-compulsive disorder.

Authors (Year)	OCD Sample	Comparison Sample	Electrophysiological Measure And Related Methodology	Key Findings
Perros et al. (1992)	13 patients (9 M, 4 F; 35.5 ± 12.4y) with DSM-III-R OCD	11 HC (5 M, 6 F; 40.1 ± 9.02y)	EEG recording while resting with eyes closed for 4 min.	Increased relative power in theta-2 power band with generally reduced variability predominantly in left frontotemporal regions.
Kuskowski et al. (1993)	13 patients (5 M, 8 F; 34.7 ± 11.6y) with DSM-III OCD.	10 HC (3 M, 7 F; 37.1 ± 11.9y)	EEG recording while resting with eyes closed for 9 min.	Significantly reduced log EEG power predominantly over the right hemisphere and confined to delta, beta1 and beta2 bandwidths.
Prichep et al. (1993)	27 patients (20 M, 7 F; 40.6y) with DSM-III-R OCD.	–	EEG recording while resting with eyes closed for 20 min. and subsequently treating with SSRIs/TCAs and assessing response with CGI score in 12 weeks	Patients were observed to be in 2 distinctive clusters. Cluster 1 (n = 10) – Significant excess of frontal theta power bilaterally, 80% were non-responders to treatment. Cluster 2 (n = 17) – Excess alpha power, 82.4% were responders to treatment. The results were obtained relative to age appropriate population norms.
Molina et al. (1995)	13 patients (9 M, 4 F; 32.1 ± 12.9y) with DSM-III-R OCD	13 HC (9 M, 4 F; 37.4 ± 17.5y)	EEG recording while resting with eyes closed for 15 min.	Total power of the global band was increased in bilateral frontal electrodes. Increased power was observed in the beta band over FP1 and FP2, theta band over FP2 and delta band over right frontal and temporal electrodes.
Drake et al. (1996)	20 patients (6 m, 14 F; 8-41y) with DSM-III-R OCD	12 HC (6 M, 6 F; 20-61y)	EEG recording while resting with eyes closed and most artefact free 1 min. epochs were used for calculations	Modal and maximal alpha frequency were reduced in bilateral frontal regions. Spectral mobility between right and left frontal regions showed no significant difference. Temporal and occipital regions showed no changes.
Locatelli et al. (1996)	37 patients (23 M, 14 F; 31.59 ± 7.26y) with DSM-III-R OCD	30 HC (11 M, 19 F; 28.1 ± 9.7)	EEG recording from temporal electrodes while, 1) resting, eyes closed 2) odour stimulation, eyes closed	When resting, increased delta-1 and decreased alpha-2 power in temporal regions. During odour stimulation, OCD patients failed to show increased beta-1 power, which was seen in controls.
Simpson et al. (2000)	6 patients (3 M, 3 F; 34.5 ± 2.2y) with DSM-IV OCD	–	EEG recording in 3 test conditions. 1) Clean control 2) Imaginal exposure 3) Live exposure	Mean log alpha power decreased slightly at anterior sites (Fz) and increased greatly at posterior sites (Pz, Oz) in live exposure, compared to clean control. No significant shifts in beta, theta or delta bands. Results were compared against the 3 test conditions.
Leocani et al. (2001)	10 patients (3 M, 7 F; 28.1 ± 7.2y) with DSM-IV OCD	10 HC (2 M, 8 F; 27 ± 4.7y)	EEG, EOG and EMG recorded while subjects performed brisk extensions of a thumb with eyes open	Pre-movement beta desynchronization occurred significantly later and post-movement beta synchronization was lower in patients when compared to controls.
Tot et al. (2002)	22 patients (5 M, 17 F; 28 ± 7y) with DSM-IV OCD	20 HC (6 M, 14 F; 30 ± 2y)	EEG recording while resting with eyes closed for 30 min. during resting state and HV	Increased delta and theta activity in left frontotemporal regions in resting state, which correlates with increasing Y-BOCS scores, female sex and good treatment response. Reduced left frontal beta activity during HV.
Hansen et al. (2003)	20 patients (12 M, 8 F; 33y) with DSM-III-R OCD	–	EEG recording while resting with eyes closed for 20 min.	Excess of absolute alpha activity in frontal, frontopolar and posterior temporal regions with deficits in delta band power and normal beta and theta activity. The results were obtained relative to age appropriate population norms.
Karadag et al. (2003)	29 patients (7 M, 22 F; 33.7 ± 10.9y) with DSM-IV OCD	29 HC (7 M, 22 F; 33.2 ± 11.9y)	EEG recording while resting with eyes closed	Increased relative theta power and decreased alpha and beta power over frontotemporal sites. Higher absolute delta power at F7 and higher theta power at temporal sites. Hemispheric asymmetries were not observed. In addition, EEG findings were characterized into OCD subgroups.
Bucci et al. (2004)	32 patients (17 M, 15 F; 28.7 ± 7.1y) with DSM-IV OCD	32 HC (17 M, 15 F; 27.5 ± 5.6y)	EEG recording while resting with eyes closed, followed by a neuropsychological evaluation	Decreased alpha-1 band power diffusely, but more prominent in frontal regions. Low alpha-1 was negatively correlated with time to complete the test. (Lower the alpha-1 band power, slower the performance).
Sherlin and Congedo (2005)	8 patients (4 M, 4 F; 17-29y) with DSM-IV-TR OCD	8 HC (5 M, 3 F; 17-29y)	EEG recording while resting with eyes closed	Excess beta current source density in the cingulate gyrus with beta-1 more prominent anteriorly and beta-2 more prominent posteriorly in the cingulate gyrus.
Fontenelle et al. (2006)	17 patients (6 M, 11 F; 33.29 ± 11.09y)	–	EEG recording while resting with eyes closed and subsequently assessing response after treating	Significantly lower beta band power in the rostral anterior cingulate and medial frontal gyrus regions in responders to treatment. Results were compared according to response to treatment.
Pogarell et al. (2006)	18 patients (8 M, 10 F; 32.4 ± 11.8y) with DSM-IV + ICD-10 OCD	18 HC (8 M, 10 F; 33.3 ± 11.3y)	EEG recording while resting with eyes closed for 5 min.	Slower mean background EEG activity, more prominent in anterior regions. Widespread increase in delta power and decreased beta and alpha-2 power. A positive correlation for obsessions and a negative correlation for compulsions were found in all powers.

(*continued on next page*)

Table 1 (*continued*)

Authors (Year)	OCD Sample	Comparison Sample	Electrophysiological Measure And Related Methodology	Key Findings
Bolwig et al. (2007)	18 patients (11 M, 7 F; 42y) with DSM-III-R OCD, responding to SSRI	–	EEG recording while resting with eyes closed for 20 min.	An excess of alpha band power was seen in corpus striatum, orbitofrontal and frontotemporal regions, which is more prominent in the right hemisphere than the left. After successful treatment, the anomalies were significantly reduced. The results were obtained relative to age appropriate population norms.
Desarkar et al. (2007)a	20 patients (10 M, 10 F; 31.45 ± 8.91y) with DSM-IV OCD	19 HC (10 M, 9 F; 29.85 ± 7.78y)	EEG recording while resting with eyes closed for 20 min.	Mean theta coherence values were significantly higher in fronto-occipital region with no correlation to medication status or symptom severity (Y-BOCS score). No differences were observed for other band powers.
Desarkar et al. (2007) b	20 patients (10 M, 10 F; 31.45 ± 8.91y) with DSM-IV OCD	19 HC (10 M, 9 F; 20.85 ± 7.78y)	EEG recording while resting with eyes closed for 20 min.	Widespread increase in theta band power. Predominantly left sided delta and alpha power increase in frontotemporal area and beta-2 power increase in left frontal area.
Velikova et al. (2010)	37 patients (15 M, 22 F; 31 ± 10.5y) with DSM-IV OCD	37 HC (15 M, 22 F; 31.5 ± 10.5y)	EEG recording while resting with eyes closed for 5 min.	Significant increase in current source density of delta at the insula and beta at frontal, parietal and limbic regions. Decreased interhemispheric coherence was also observed.
Kopřivová et al. (2011)	50 patients (20 M, 30 F; 29.2 ± 5y) with DSM-IV OCD	50 HC (23 M, 27 F; 28.4 ± 5.6y)	EEG recording while resting with eyes closed	Low frequency (2-6 Hz) power excess in the medial frontal cortex localized more towards the left side. Theta excess was positively correlated with non-response to SSRIs.
Min et al. (2011)	16 patients (12 M, 4 F; 24y) with DSM-IV OCD	16 HC (7 M, 9 F; 23y)	EEG recording while performing a colour and shape discriminating task	Significantly lower pre-stimulus total alpha power at parietal regions and theta power at frontal regions in OCD patients when compared to controls.
Ischebeck et al. (2014)	20 patients (8 M, 12 F; 33.1 ± 9.3y) with DSM-IV OCD	20 HC (9 M, 11 F; 32.6 ± 9.4y)	EEG recording while performing a passive viewing task	Frontal alpha-1 power was more dominant in the left hemisphere across all conditions, suggesting increased avoidance motivation in OCD.
Kamaradova et al. (2016)	20 patients (15 M, 5 F; 31.45 ± 8.1y) with MINI OCD.	15 HC (9 M, 6 F; 33.33 ± 6.47y)	EEG recording while resting with eyes closed after testing for cognitive impairment	In OCD patients with cognitive impairment (n = 11), significant increase in theta power at middle frontal gyrus and delta power at insula were seen.
Grützmann et al. (2017)	113 patients (52 M, 61 F; 31.5 ± 9.5y) with DSM-IV OCD	113 HC and 37 1st degree relatives	EEG recording while resting and while performing a passive viewing task	No significant difference in frontal alpha asymmetry was observed between patients and HC.

M – Males, F – Females, HC – Healthy controls, OCD – obsessive compulsive disorder, EEG – electroencephalography, TCA – tri-cyclic anti-depressants, CGI – Clinical Global Impressions rating system, EOG – Electro-oculogram, EMG – Electro-myogram, HV – Hyperventilation, Y-BOCS – Yale-Brown Obsessive Compulsive Scale, LORETA – Low-resolution electromagnetic tomography, MINI – Mini-international neuropsychiatric interview, min. - minutes.

Jagadheesan, & Nizamie, 2007; Kamaradova et al., 2016; Karadag et al., 2003; Kopřivová et al., 2011; Molina et al., 1995; Prichep et al., 1993; Tot et al., 2002) and theta-1 (6–7.5 Hz) (Perros et al., 1992) band power was observed among OCD sufferers. Furthermore, excess theta band power is generally associated with SSRI resistance (Kopřivová et al., 2011; Prichep et al., 1993). However, one study reported a correlation between excess frontal theta activity and good treatment response to SSRIs (Tot et al., 2002), which was attributed to the younger sample population and possible genetic differences.

3.3.2. Functional/task related theta activity

In contrast to the resting findings, a study that utilized a colour discrimination task documented reduced frontal theta power in OCD patients, compared to controls (Min, Kim, Park, & Park, 2011). In healthy participants, frontal theta activity is reported to increase during attention demanding tasks (Scheeringa et al., 2009). Furthermore, it is postulated that theta activity reflects the brain's mechanism of selecting between competing processes, for example, when suppressing distractions during a focused activity (Nigbur, Ivanova, & Stürmer, 2011). It is theorized that frontal theta activity, particularly generated by the anterior cingulate cortex, reflects active inhibition (Wang, Ulbert, Schomer, Marinkovic, & Halgren, 2005). Since OCD sufferers are known to be affected by deficits in inhibitory regulation (Richter et al., 2012), it seems conceivable that patients may present with abnormally low task related frontal theta activity (functional), but excessive resting theta activity (non-functional).

3.4. Deviations in alpha activity

It is generally accepted that lower alpha power is associated with increased fMRI blood oxygen level dependent response, and thought to reflect increased cortical activation (Laufs et al., 2003). However, due to the presence of excessive individual variations in alpha activity, interpreting such results is a challenge.

3.4.1. Resting state alpha activity

An elevation of resting frontotemporal alpha activity, compared to healthy controls was observed in several OCD studies (Bolwig et al., 2007; Desarkar et al., 2007a; Hansen et al., 2003; Ischebeck et al., 2014; Prichep et al., 1993). However, decreased overall alpha (Drake et al., 1996; Karadag et al., 2003), alpha-1 (8–10 Hz) (Bucci et al., 2004) and alpha-2 (10–13 Hz) (Locatelli et al., 1996; Pogarell et al., 2006) activity was noted by others. This ambiguity may be due to differences in study design or the heterogeneous nature of OCD symptoms. It is therefore, unclear whether resting alpha activity is modulatd in OCD and remains an open area for future research.

3.4.2. Functional/task related alpha activity

Additionally, two studies reported reduced cognitive task related alpha activity in OCD patients, compared to healthy controls (Min et al., 2011; Simpson, Tenke, Towey, Liebowitz, & Bruder, 2000). The cognitive tasks used in these studies include a colour and shape discrimination task and a task involving exposure to OCD symptom provoking conditions, respectively. It has been suggested that reduced alpha activity was related to difficulties in suppressing distracters or task-irrelevant details (Crawford, Knebel, Vendemia, Kaplan, & Ratcliff, 1995). People with OCD are frequently preoccupied with distracters and have an intrinsic deficit in inhibiting obsessive thoughts, which may explain the alpha reduction in OCD patients during cognitive tasks.

Furthermore, alpha reduction was observed to shift from posterior to anterior topography during live exposure to OCD symptom-provoking situations, indicating frontal activation (Simpson et al., 2000). This finding is consistent with brain imaging studies describing frontal activation during symptom provocation in OCD (McGuire et al., 1994).

3.5. Deviations in beta activity

3.5.1. Resting state beta activity

Beta activity alterations are linked with cerebral metabolism (Gamma et al., 2004), where decreased beta activity correlates with decreased cerebral blood flow and therefore, decreased metabolism in that region. Although beta power band abnormalities have been observed by many studies, their findings have been mostly inconsistent. Both significant reductions (L. Fontenelle, Mendlowicz, Ribeiro, Piedade, & Versiani, 2006; Karadag et al., 2003; Kuskowski et al., 1993; Tot et al., 2002) and increases (Desarkar et al., 2007a; Molina et al., 1995; Sherlin & Congedo, 2005) in beta activity are reported in literature. Several functional neuroimaging studies have linked OCD with altered cerebral metabolism rates (Perani et al., 1995; Whiteside, Port, & Abramowitz, 2004). However, the neurophysiological basis of beta activity in OCD remains unestablished.

3.5.2. Functional/task related beta activity

OCD patients failed to show an increase in the temporal region beta band power during odour stimulation; a finding that was consistently present in healthy controls (Locatelli et al., 1996). As olfactory stimulation is expected to produce a desynchronization of electrical activity with an enhancement in beta band power (Chapman, Xu, Haykin, & Racine, 1998), OCD patients seem to have impaired neuro-functional responses to temporal activation procedures.

3.6. Summary of QEEG findings

Frontal asymmetries in alpha and theta bands appear to be prominent in OCD patients. They also show increased resting frontotemporal slow wave (delta and theta) activity. Both alpha and beta power bands show inconsistent findings with both increased and decreased activity.

4. Sleep related EEG in OCD

Sleep related problems (SRP) are extremely common among OCD populations (Nota et al., 2015; Paterson, Reynolds, Ferguson, & Dawson, 2013; Storch et al., 2008). In reported paediatric OCD populations, 92% experienced at least one SRP and 27.3% experienced five or more concurrent SRPs (Storch et al., 2008). Additionally, studies indicate that adult OCD populations experience similar SRPs such as sleep latency, reduced sleep duration and sleep phase shifting (Bobdey, Fineberg, Gale, Patel, & Davies, 2002; Donse, Sack, Fitzgerald, & Arns, 2017; Turner et al., 2007). Sleep is generally assessed using clinical polysomnography, which incorporates EEG and several other physiological measurements. In OCD patients, EEG has been used to measure rapid eye movement (REM) sleep onset and durations, delta sleep percentage and sleep stage (Šušmáková, 2004).

The polysomnography parameters of interest in OCD are the REM sleep related measures. Experimental studies have found that the serotonergic system is important in regulating REM sleep (Steriade & McCarley, 2013). It has been postulated that OCD is linked with serotonergic deficits (Insel et al., 1983), which may be the causative factor involved in REM sleep abnormalities in individuals with OCD.

REM latency has been extensively studied among OCD populations and is defined as the time from sleep onset to the first epoch of REM sleep. Early studies described shortening of REM latency in OCD patients (Insel et al., 1982; Rapoport et al., 1981). Short REM latencies have been found to be a consistent finding in primary depressive disorders (Friess et al., 2008; Kupfer, 1976; Rao, Hammen, & Poland, 2009). Therefore, this finding in OCD sufferers could be due to the presence co-morbid depression. However, the subsequent studies failed to observe a significant difference in REM latency between OCD patients and healthy controls (Hohagen et al., 1994; Kluge, Schüssler, Dresler, Yassouridis, & Steiger, 2007; Robinson, Walsleben, Pollack, & Lerner, 1998; Voderholzer et al., 2007).

Table 2
Summary of sleep electroencephalography findings in individuals with obsessive-compulsive disorder.

Authors (year)	Sample size		Electrophysiological measure and related methodology	Key findings							
	Patients	Controls		Total sleep time	Sleep efficiency	awakenings	REM efficiency	REM density	REM latency	Delta sleep	Stage 4 sleep
Rapoport et al. (1981)	9	9	All night polysomnography with EEG, EOG, EMG.	↓	↓	NA	NA	•	↓	↑	•
Insel et al. (1982)	14	14	All night polysomnography with EEG, EOG, EMG.	↓	↓	↑	↓	•	↓	↓	↓
Hohagen et al. (1994)	22	22	All night polysomnography with EEG, EOG, EMG.	•	↓	↑	NA	•	•	NA	•
Robinson et al. (1998)	13	13	Structured sleep log with polysomnography.	•	•	↑	NA	NA	•	•	•
Kluge et al. (2007)	10	10	Polysomnography with EEG, vertical and horizontal EOG, EMG and ECG.	•	•	•	NA	NA	•	•	↓
Voderholzer et al. (2007)	62	62	All night polysomnography with EEG, EOG, EMG.	↓	↓	NA	NA	↑	•	•	NA

↓ - Significantly lower than controls, ↑ - Significantly higher than controls, • - No significant difference from controls, NA – Not assessed, EEG – electroencephalogram, EOG – Electro-oculogram, EMG – Electromyogram, ECG – Electrocardiogram, REM – Rapid eye movement.

REM density represents the total duration of rapid eye movement during a REM period. Attenuated REM density is related to diseases that diffusely affect brain function (King et al., 1981). Although early studies (Hohagen et al., 1994; Insel et al., 1982; Rapoport et al., 1981) did not find a significant difference in the REM density of OCD patients, the largest sleep study (n = 62) in OCD (Voderholzer et al., 2007), showed a significant increase in REM density. Since enhanced REM density is linked with depression (Lahmeyer, Poznanski, & Bellur, 1983), we postulate that this finding is related to comorbid depression in OCD patients. A summary of these findings is presented in Table 2 and Fig. 2.

The first sleep EEG study described some important electrophysiological alterations in brain activity of paediatric OCD sufferers (Rapoport et al., 1981). Shortened REM latency, increased delta sleep percentage and increased stage 4 sleep were some of the findings of this study. However, no significant abnormalities in REM density were found.

Furthermore, a recent study analysed EEG arousal regulation as a predictor of response to three treatment modalities; SSRIs, CBT or a combination (Dohrmann, Stengler, Jahn, & Olbrich, 2017). Through EEG monitoring, they defined four vigilance stages leading to sleep (concentration, relaxed wakefulness, drowsiness and sleep onset). Their key finding was that OCD sufferers with a shorter concentration stage were more likely to respond to all treatment modalities with a preference towards combination therapy.

4.1. Summary of sleep EEG findings

Increased REM density, decreased sleep efficiency and duration were significant findings in individuals with OCD. Since patients with depression are known to exhibit similar sleep irregularities, these observations may be due to comorbid depression in OCD.

5. Event related potentials in OCD

Event related potentials (ERP) denote voltage changes attributed to the brain's response to a stimulus. They are reliably measured by recording EEG during specific sensory, cognitive or motor events (Bressler & Ding, 2006; Coles & Rugg, 1995). Abnormal ERPs are linked to many psychiatric disorders (Neshige, Barrett, & Shibasaki, 1988; Pritchard, 1986; Spronk, Arns, Bootsma, van Ruth, & Fitzgerald, 2008) including OCD. A summary of ERP findings in OCD is presented in Table 3.

5.1. Error related negativity in OCD

Error related negativity (ERN) is an ERP that is observed when the participant erroneously responds to a stimulus. It is most commonly measured with executive function and inhibition tasks (Gehring, Liu, Orr, & Carp, 2012; Yeung, Botvinick, & Cohen, 2004). ERN is the negative deflection of the EEG observed in the medial frontal regions, approximately 50–150 ms following an erroneous response (Gehring, Goss, Coles, Meyer, & Donchin, 1993). ERN related abnormalities in OCD have been extensively analysed in a previous review of literature (Endrass & Ullsperger, 2014). Since ERN reflects action monitoring processes, the consistent ERN enhancement in OCD patients may reflect overactive performance monitoring (Tamnes, Walhovd, Torstveit, Sells, & Fjell, 2013). Furthermore, it is noteworthy that in most of the studies, ERN related abnormalities were observed in the absence of any significant behavioural differences in error rates or error reaction times (Endrass, Klawohn, Schuster, & Kathmann, 2008; Nawani et al., 2017; Xiao et al., 2011).

Many studies that utilized Stroop, Go/NoGo, Eriksen Flanker (Eriksen & Eriksen, 1974) and other tasks reported increased ERN amplitudes in OCD patients, compared to healthy controls. In the Stroop test, the participant is instructed to say the colour of each word in each column instead of reading the word shown. It is a demonstration of the reaction time and is often used to illustrate the nature of

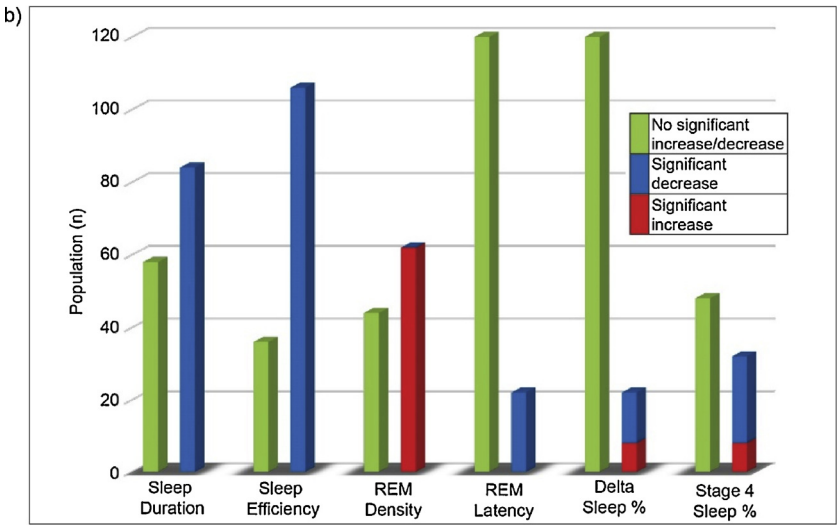

Fig. 2. Sleep related EEG abnormalities in OCD.
The x-axis represents the different parameters measured by the studies under review. The y-axis represents the total number of individuals with OCD across all six studies that showed either a significant increase/decrease or failed to show a significant difference in those parameters compared to controls. According to the findings, increased rapid eye movement (REM) sleep density and decreased sleep duration and efficiency appear to be the most consistently present sleep related findings in OCD patients when compared to healthy controls.

automatic processing. In the Go/NoGo task, participants are required to either respond or withhold a response depending on whether a Go stimulus or a NoGo stimulus is presented. The Flanker Task uses a set of response inhibition tasks to assess the ability to suppress responses when inappropriate. An early ERN study of OCD patients revealed an enhanced ERN amplitude and duration while performing a modified Stroop task (Gehring, Himle, & Nisenson, 2000). These findings were replicated in adult (Johannes et al., 2001; Nawani et al., 2017; Xiao et al., 2011) and paediatric (Hajcak, Franklin, Foa, & Simons, 2008; Hanna et al., 2012) populations. Furthermore, the combination of EEG and functional magnetic resonance imaging (fMRI) enables better localization of the neural source of enhanced ERN. The findings of a study (Grützmann et al., 2016) employing EEG and fMRI concurrently, revealed higher ERN to be more prominent in amygdala and subgenual anterior cingulate cortex in OCD, suggesting a stronger affective response towards errors in OCD patients.

In addition, one study that replicated the ERN enhancement (Ruchsow et al., 2005) further reported that early error positivity (reflecting automatic error processing) and late error positivity (reflecting error response awareness) were not different between patients and controls. These findings led them to deduce that compulsivity in OCD patients is related to hyper-functioning error monitoring.

Furthermore, two studies (Endrass et al., 2008; Endrass, Riesel, Kathmann, & Buhlmann, 2014) that replicated the ERN enhancement, further observed that correct response negativity (CRN - response locked fronto-central negativity associated with execution of correct responses) was also significantly enhanced in individuals with OCD. This finding further supports the view that performance monitoring is overactive in OCD. The same parameters, when assessed with a punishment condition for erroneous responses, showed no significant difference between patients and controls (Endrass et al., 2010). Furthermore, a four-choice object reversal learning task was administered to analyse whether feedback guided learning is compromised in OCD (Endrass, Koehne, Riesel, & Kathmann, 2013). Feedback related negativity (FRN - negative deflection in the EEG occurring around 250 ms after feedback onset) was found to be reduced during negative feedback. This observation indicates that overactive performance monitoring in OCD sufferers is somewhat attenuated during learning processes.

In contrast, one study observed no significant difference in the ERN amplitude between OCD sufferers and healthy controls (Nieuwenhuis, Nielen, Mol, Hajcak, & Veltman, 2005). However, a majority of the OCD sample they recruited were on medication. Since pharmacological agents are known to alter the ERN amplitude (De Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004), this observation could be a direct result of medication effects. Furthermore, another study that observed no significant difference in the ERN amplitude between OCD and healthy samples, found that checking behaviours in particular, were associated with enhanced ERN across multiple psychiatric conditions including generalized anxiety disorder and major depressive disorder (Weinberg, Kotov, & Proudfit, 2015).

5.1.1. *Attempts at modulation of ERN in OCD*

Some ERN studies in OCD have shifted their focus to attempt modulation of the ERN enhancement with the aim of enhancing our knowledge of the underlying pathophysiology. A study that utilised a probabilistic learning task observed that the enhanced ERN in OCD patients was lessened with increasing conflict following errorful learning (interfering and conflicting items are presented in addition to the relevant stimulus) (Hammer, Kordon, Heldmann, Zurowski, & Münte, 2009). Furthermore, a recent study (Klawohn, Endrass, Preuss, Riesel, & Kathmann, 2016) assessed the ERP in OCD patients during a combined flanker and n-back test. This test attempted manipulating attention resource allocation in order to assess whether hyperactive error monitoring in OCD is situationally flexible. They observed a significant reduction in the ERN enhancement with dual task demands. They concluded that altering the attentional focus might improve overactive error monitoring in OCD sufferers.

Moreover, another recent study (Roh, Chang, Yoo, Shin, & Kim, 2017) showed that further ERN enhancement is experimentally possible by presenting individually tailored, OCD symptom provoking images.

5.1.2. *ERN as an endophenotype for OCD*

Endophenotypes are objectively measurable components that are closely related to an underlying susceptibility for a disease along the pathway between disease manifestation and distal genotype (Flint & Munafò, 2007; Gottesman & Gould, 2003). Studies suggest that it is highly plausible that the susceptibility for an illness will manifest itself in some fashion before the emergence of its clinical signs and symptoms. An endophenotype is defined as being, 1) associated with illness in the population, 2) heritable, 3) primarily state independent (manifests regardless of the presence of symptoms), 4) present in unaffected family members at a higher rate than in the general population and 5) co-segregated with the disease within families (Gottesman & Gould, 2003).

ERN as a candidate endophenotype for OCD was suggested in a paediatric OCD study (Hajcak et al., 2008), where it was observed that the ERN remained unchanged after successful treatment led to symptomatic improvement. Subsequently, first degree asymptomatic relatives of OCD sufferers were noted to have significantly enhanced ERN,

Table 3
Summary of event related potentials in obsessive-compulsive disorder.

Authors (year)	OCD sample	Controls	Methodology	Findings
5.1. ERROR RELATED NEGATIVITY (ERN) IN OCD				
Gehring et al. (2000)	9 (4 M, 5 F; 39.6y)	9 (4 M, 5 F; 40.5y)	EEG recording while performing a modified Stroop test	Enhanced height and duration of ERN in medial frontal regions. ERN enhancement is positively correlated with OCD symptom severity.
Johannes et al. (2001)	10 (6 M, 4 F; 22-40y)	10 (10 M, 0 F; 25-29y)	EEG recording while performing a choice reaction time task	Enhanced ERN amplitude and longer ERN latency with a posterior topography.
Nieuwenhuis et al. (2005)	16 (6 M, 10 F; 30.9 ± 8.5y)	16 (6 M, 10 F; 30.9 ± 7.6y)	EEG monitoring while performing a probabilistic learning task.	ERN amplitude was higher in OCD patients when compared to controls, but the difference was not statistically significant.
Endrass et al. (2008)	20 (12 M, 8 F; 33.5 ± 8y)	20 (12 M, 8 F; 34.8 ± 8.6y)	EEG monitoring while performing a modified Flanker task	Enhanced ERN and CRN when compared to controls. Depicts overactive performance monitoring in OCD. Enhanced CRN is positively correlated to symptom severity
Endrass et al. (2010)	22 (11 M, 11 F; 32.2 ± 7.8y)	22 (11 M, 11 F; 31.2 ± 8.4y)	EEG monitoring while performing a modified Flanker task with a punishment condition	Enhanced ERN and CRN in standard condition. No significant difference between groups when punishment is introduced to erroneous responses.
Xiao et al. (2011)	25 (15 M, 10 F; 19-60y)	27 (15 M, 12 F; 19-54y)	EEG monitoring while performing a modified Flanker task	Ne component of ERN was significantly enhanced in OCD patients when compared to healthy controls.
Endrass et al. (2013)	25 (9 M, 16 F; 33.4 ± 9.4y)	25 (8 M, 17 F; 30.1 ± 8.3y)	EEG monitoring while performing a deterministic four-choice object reversal task	FRN was reduced during negative feedback in a four-choice object reversal-learning task.
Grützmann et al. (2016)	20 (11 M, 9 F; 32.3 ± 8.6y)	22 (11 M, 11 F; 30.8 ± 8.5y)	EEG monitoring while performing a modified Flanker task	Concurrent EEG and fMRI revealed enhanced ERN to be localized to amygdala and subgenual ACC suggesting a stronger affective response towards errors.
5.1.1. ATTEMPTS AT MODULATION OF ERN IN OCD PATIENTS				
Hammer et al. (2009)	16 (10 M. 6 F; 37y)	16 (10 M, 6 F; 36.7y)	EEG monitoring while performing a probabilistic learning task.	Enhanced ERN was observed in OCD patients when compared to controls. This enhancement was reduced with increased conflict after errorful learning.
Klawohn et al. (2016)	22 (13 M, 12 F; 28.3 ± 6.3y)	22 (13 M, 12 F; 27.9 ± 5.8y)	EEG monitoring while performing a modified Flanker task ± n-back task	Enhanced ERN in OCD. Significant reduction in ERN during dual task demands.
Roh et al. (2017)	20 (9 M, 11 F;32.2 ± 9.4y)	20 (9 M, 11 F; 32.7 ± 6.6y)	EEG monitoring while performing a Flanker task	Enhanced ERN can be further enhanced by presenting patients with OCD symptom provoking images.
5.1.2. ERN AS AN ENDOPHENOTYPE FOR OCD				
Hajcak et al. (2008)	18 (13 M, 5 F; 13.3 ± 2.8y)	18 (8 M, 10 F; 11.9 ± 2.6y)	EEG monitoring while performing a reaction time task	ERN enhancement was noted in paediatric populations. No change in ERN after symptom improvement with treatment.
Riesel et al. (2011)	30 (43.6 ± 11.7y)	30 1° relatives +30 HC	EEG monitoring while performing a modified Flanker task	Both asymptomatic first-degree relatives and OCD patients showed an enhancement in ERN when compared to healthy controls.
Carrasco et al. (2013)	40 (13.9 ± 2.4y)	19 normal siblings + 40 HC	EEG monitoring while performing a modified Flanker task	Enhanced ERN amplitude was noted in both paediatric OCD patients and their unaffected siblings.
Ruchsow et al. (2005)	11 (6 M, 5 F)	11 (6 M, 5 F)	EEG monitoring while performing a Go/NoGo task	Significant increase in ERN amplitude of OCD patients. No significant correlation with OCD severity or age. No differences in early or late error positivity.
Hanna et al. (2012)	44 (20 M, 24 F; 10-19y)	44 (22 M, 22 F; 10-18y)	EEG monitoring while performing a modified Flanker task.	Enhanced ERN in paediatric populations, which is poorly correlated to their symptom severity.
Endrass et al. (2014)	24 (7 M, 17 F; 31.5 ± 8.7y)	24 (7 M, 17 F; 32.1 ± 8.4y)	EEG monitoring while performing a modified Flanker task	ERN amplitudes were larger in OCD group when compared to healthy controls without any correlation to symptom severity.
Riesel et al. (2014)	72 (29 M, 41 F; 35.5 ± 10.8y)	72 (35 M, 37 F; 38.8 ± 12.8y)	EEG monitoring while performing a modified Flanker task	Enhancement in ERN and CRN of OCD patients is not correlated with their symptom severity.
Nawani et al. (2017)	16 (13 M, 3 F; 29.3 ± 5.9y)	17 (12 M, 5 F; 29 ± 4.5y)	EEG monitoring while performing a modified Flanker task	Enhanced ERN at Fz and Cz with no correlation to symptom severity.
5.2. OTHER EVENT RELATED POTENTIALS (ERP) IN OCD				
Malloy et al. (1989)	18 (34 ± 12.8y)	18 (28 ± 8.7y)	EEG monitoring while performing a Go/NoGo task	Significantly lower P3 amplitude in the orbitofrontal regions during the NoGo task
Towey et al. (1990)	10 (6 M, 4 F; 39.5 ± 7)	10 (4 M, 6 F; 28.3 ± 7y)	EEG monitoring while performing an auditory oddball task	OCD patients failed to show increased latency of P3 and N2 when task difficulty was increased. OCD patients showed a greater N2 negativity than controls.
Towey et al. (1993)	17 (13 M, 4 F; 35.9 ± 7.3y)	16 (5 M, 11 F; 31.6 ± 7.7y)	EEG monitoring while performing an auditory oddball task	Greater N2 amplitude of OCD patients was correlated with less severe obsessions and better response to SSRIs.
Towey et al. (1994)	18 (14 M, 4 F; 30 ± 9.1y)	15 (10 M, 5 F; 32.5 ± 9.6y)	EEG monitoring while performing a modified selective attention task	Larger N2 amplitude and smaller P3 amplitudes in OCD patients when compared to healthy controls
Morault et al. (1998)	21 (6 M, 15 F; 37.3 ± 10.9y)	21 (6 M, 15 F; 35.3 ± 10.9y)	EEG monitoring while performing a verbal auditory oddball task	Significantly reduced N2 amplitude and shorter N2 and P3 latencies in OCD patients are positively correlated to treatment response
Miyata et al. (1998)	23 (11 M, 12 F; 33.1 ± 15y)	18 (4 M, 14 F; 30.8 ± 14y)	EEG monitoring while performing an auditory oddball task	Greater N2 negativities and short P3 and N2 latencies in OCD patients with no correlation to symptom severity.

(*continued on next page*)

Table 3 (*continued*)

Authors (year)	OCD sample	Controls	Methodology	Findings
Sanz et al. (2001)	19 (19 M, 9 F; 20.5y)	19 (9 M, 10 F; 25.8y)	EEG monitoring while performing an auditory oddball task	Lower amplitude and longer latency of P3 in OCD patients when compared to controls. P3 amplitude increased after treatment with no change in P3 latency.
Mavrogiorgou et al. (2002)	21 (12 M, 9 F; 33.9 ± 12y)	21 (12 M, 9 F; 36.4 ± 10.9y)	EEG monitoring while performing an auditory oddball task	Larger P3b amplitude and latency in OCD patients noted only in the right hemisphere. No changes were noticed in P3a.
Kıvırcık, Yener, Alptekin, and Aydın (2003)	31 (15 M, 6 F; 27 ± 9.8y)	30 (14 M, 16 F; 27.4 ± 9.1y)	EEG monitoring while performing an auditory oddball task	P3 duration was found to be shorter in OCD patients when compared to healthy controls.
Kim et al. (2007)	15 (8 M, 7 F; 25.7 ± 4.8y)	15 (8 M, 7 F; 25 ± 2.9y)	EEG monitoring while performing Go/NoGo task	N2 amplitude was significantly smaller during the NoGo task in OCD patients when compared to controls
Gohle et al. (2008)	63 (30 M, 33 F; 33.7 ± 10.2y)	63 (27 M, 36 F; 36.1 ± 12.8y)	EEG monitoring while performing an auditory oddball task	Significantly larger P3b amplitude in OCD patients with no difference in P3a component when compared to controls
Ischebeck et al. (2011)	20 (7 M, 13 F; 32.8 ± 9.9y)	20 (7 M, 13 F; 32.7 ± 9.3y)	EEG monitoring while performing a visual novelty recognition task	OCD patients showed increased novelty P3 amplitude when compared to healthy controls
Andreou et al. (2013)	71(37 M, 34 F; 34.7 ± 10.8y)	71 (32 M, 39 F; 36.5 ± 12.2y)	EEG monitoring while performing an auditory oddball task	Increased P3 activity in the left orbitofrontal, prefrontal, parietal and temporal regions which reduced significantly after treatment
Yamamuro et al. (2015)	20 (12 M, 8 F; 12.6 ± 2.2y)	20 (15 M, 5 F; 12.25 ± 2.7y)	EEG monitoring while performing an auditory oddball task	Reduced P3 amplitudes in OCD group when compared to controls with significant correlation to their OCD symptom severity
Riesel et al. (2017)	70 (32 M, 38 F; 35.2 ± 10.9y)	70 (32 M, 38 F; 38.6 ± 12.3y)	EEG monitoring while performing an arrowhead Flanker task	N2 amplitude was significantly greater in OCD patients when compared to controls.

M – Males, F – Females, OCD – obsessive-compulsive disorder, EEG – electroencephalography, ERN – error related negativity, FRN – feedback related negativity, CRN – correct response negativity.

compared to healthy controls (Riesel, Endrass, Kaufmann, & Kathmann, 2011), a finding that supports this phenomenon to be a trait-like marker for OCD. This conclusion was strengthened by the same finding being present in asymptomatic siblings of paediatric OCD sufferers (Carrasco et al., 2013). Furthermore, several studies (Endrass et al., 2014; Hanna et al., 2012; Nawani et al., 2017; Riesel, Kathmann, & Endrass, 2014; Ruchsow et al., 2005) concluded that the enhancement of ERN in OCD patients is independent of their symptom severity. Considering the above findings, ERN enhancement in OCD appears to satisfy the criteria required for a candidate endophenotype.

5.2. Other ERPs related to OCD

Other ERPs commonly linked with OCD are P3 and N2. P3 is observed approximately 300 ms after stimulus onset and is thought to be related to attentional resource allocation, working memory updating (Polich & Kok, 1995) and the mental model of the environment required to make a suitable response (Linden, 2005). P3 is further subcategorized into P3a (reflects reactions to novel stimuli) and P3b (reflects updating of working memory in response to task-relevant events). N2 is a negative deflection peaking at around 200 ms after a stimulus and is thought to be linked to cognitive processes of conflict monitoring, stimulus identification and distinction (Hoffman, 1990). However, the functional processes that both of these ERPs reflect can vary by task specific parameters (Ritter, Simson, & Vaughan, 1983).

Individuals with OCD have shown reduced P3 amplitudes, compared to healthy controls during tasks such as, Go/NoGo (Malloy, Rasmussen, Braden, & Haier, 1989), auditory oddball (Sanz, Molina, Martin-Loeches, Calcedo, & Rubia, 2001; Yamamuro et al., 2015) and selective attention (Towey et al., 1994) tasks. As P3 is known to be a good index of cognitive processing of selective attention, uncertainty resolution and decision confidence (Stuss & Picton, 1978), these functions may be impaired in OCD sufferers. Additionally, lower P3 amplitude in OCD patients was linked to misallocation of cognitive resources to task relevant stimuli (Towey et al., 1994).

In contrast, an increase in P3b (Gohle et al., 2008; Ischebeck, Endrass, Simon, & Kathmann, 2011; Mavrogiorgou et al., 2002), and an overall increase in P3 (Andreou et al., 2013) reported in several studies, suggested over-focussed attention and hyper-arousal in OCD patients. It is known that hyper-arousal is related to an overactive noradrenergic system (Foote, Bloom, & Aston-Jones, 1983). Interestingly, noradrenergic activity in the locus coeruleus system is found to be involved in P3 generation (Pineda, Foote, & Neville, 1989).

The N2 amplitude was significantly larger in individuals with OCD, compared to healthy controls in studies utilising auditory oddball (Miyata et al., 1998; Towey et al., 1990, 1993), Flanker (Riesel, Klawohn, Kathmann, & Endrass, 2017) and selective attention (Towey et al., 1994) tasks. This may be explained by hyper-arousal and over-focused attention in OCD patients (Näätänen, Gaillard, & Mäntysalo, 1978). However, smaller N2 amplitudes were reported in studies that utilized the Go/NoGo (Kim, Kim, Yoo, & Kwon, 2007) and auditory oddball tasks (Morault, Guillem, Bourgeois, & Paty, 1998). It is generally accepted that N2 reflects a decision process that controls behavioural responses (Upton, Enticott, Croft, Cooper, & Fitzgerald, 2010) and conflict monitoring (Cavanagh, Zambrano-Vazquez, & Allen, 2012). This involves stimulus identification, categorization and extraction of information required for subsequent cognitive operations (Renault, Ragot, Lesevre, & Remond, 1982). Therefore, the attenuated N2 observed in OCD patients may reflect poorly controlled stimulus information processing, affecting both behavioural responses and inhibition.

Additionally, when compared to healthy controls, numerous studies concluded that shorter P3 and N2 latencies were observed in individuals with OCD (Miyata et al., 1998; Morault et al., 1998; Towey et al., 1990). Furthermore, increasing the difficulty of the task failed to increase the P3 and N2 latencies in OCD patients, compared to controls (Towey et al., 1990). This could reflect cortical hyper-arousal in OCD, resulting in speeding of cognitive processing, leading to shortened P3 and N2 latencies (Beech, Ciesielski, & Gordon, 1983).

5.3. Summary of ERP findings

Significant enhancement of ERN amplitude was a consistent finding among OCD patients and indicates over-active action monitoring during cognitive tasks. There is significant evidence suggesting that ERN might be a potential candidate endophenotype for OCD. Furthermore, other ERP findings include, enhanced P3 and greater negativity of N2, indicating cortical hyper-arousal and impaired response inhibition respectively. Additionally, shorter N2 and P3 latencies were observed in OCD patients, which demonstrate speeding of cognitive processing.

6. TMS-EEG and the future of OCD

Transcranial magnetic stimulation (TMS) is a non-invasive brain stimulation technique with investigational and therapeutic potential in psychiatric disorders (Bersani et al., 2013), including OCD (Bersani et al., 2013; Daskalakis, Christensen, Fitzgerald, & Chen, 2002; Fitzgerald, Brown, & Daskalakis, 2002). Concurrent use of TMS with EEG (TMS-EEG) has enabled measurement of TMS-evoked cortical activity to determine the neural changes (Chung, Rogasch, Hoy, & Fitzgerald, 2015; Daskalakis, Farzan, Radhu, & Fitzgerald, 2012; Rogasch & Fitzgerald, 2013). This novel technique has aided in revealing the underlying pathophysiology of various psychiatric disorders (Canali et al., 2014; Frantseva et al., 2012) and therefore, holds potential for broadening our understanding of OCD by either using EEG to measure changes as a result of stimulation or using stimulation to specifically change EEG biomarkers of OCD.

A recent TMS-EEG study (Radhu et al., 2017) investigated the GABA mediated frontal cortical inhibition in OCD and schizophrenia patients. However, they reported no significant findings for OCD patients. We believe that more targeted investigations evaluating the known brain pathologies in OCD using TMS-EEG are essential in order enhance our knowledge.

7. Discussion

This review analysed various aspects of EEG in OCD such as, QEEG, sleep EEG and ERPs. Frontal hemispheric asymmetry, impaired sleep functions and overactive performance monitoring were some of the major documented underlying differences observed in individuals with OCD, compared to healthy controls.

According to QEEG findings, frontal asymmetries in alpha (Desarkar et al., 2007a; Ischebeck et al., 2014) and theta (Kopřivová et al., 2011; Perros et al., 1992; Tot et al., 2002) bands appeared to be significantly prominent in OCD patients, compared to healthy controls. This could be due to comorbidities such as depression or substance abuse that affect frontal electrical activity (Zitterl et al., 2000). However, the traditional belief that depressive disorders exhibit frontal alpha asymmetry (Henriques & Davidson, 1991; Pizzagalli et al., 2002) was challenged by a recent meta-analysis (van der Vinne, Vollebregt, van Putten, & Arns, 2017) concluding that the heterogeneity among studies renders this finding insignificant with limited diagnostic value. Therefore, both meta-analytic and large sample, functional relationship focused studies of asymmetry in OCD are required for further understanding.

OCD patients show increased resting frontotemporal slow wave (delta and theta) activity, which is linked to central nervous system vulnerability (Hughes & John, 1999). However, cognitive task related frontal theta power was found to be reduced in OCD patients, compared to healthy controls (Min et al., 2011). Frontal theta activity, which reflects active inhibition (Wang et al., 2005), is expected to increase during tasks that require working memory or attention (Scheeringa et al., 2009). Since OCD patients suffer from inhibitory regulation

deficits (Richter et al., 2012), this frontal theta reduction is plausible.

Furthermore, studies have found both increased and decreased resting alpha band power in OCD patients. Therefore, it is not clear whether resting alpha activity is modulated in OCD sufferers. Furthermore, reduced frontal alpha activity during a cognitive task was observed in OCD patients (Min et al., 2011; Simpson et al., 2000). This phenomenon was attributed to deficits in suppressing task-irrelevant details or distractions (Crawford et al., 1995), a common characteristic of OCD patients.

There are both increased and decreased resting frontotemporal beta band power findings among OCD patients. These discrepancies were explained by altered cerebral metabolism, which directly affects beta power distribution (Gamma et al., 2004). Furthermore, several studies observed abnormalities in cerebral glucose metabolism in treatment resistant OCD patients (Perani et al., 1995; Whiteside et al., 2004). However, investigations are needed to study changes in beta activity during cognitive tasks.

With reference to sleep EEG in OCD patients, increased REM density, decreased sleep efficiency and duration were significant findings. Since patients with depression are known to exhibit similar sleep irregularities (Lahmeyer et al., 1983), these observations may be due to comorbid depression. However, due to the inconsistencies in the literature, further investigations are required to strengthen our understanding on sleep characteristics of OCD sufferers.

ERPs indicate the brain's response to diverse stimuli and were considerably altered in OCD patients, compared to healthy controls. Significant enhancement of ERN amplitude was a consistent finding among OCD patients and indicates over-active action monitoring during cognitive tasks. The presence of this anomaly in asymptomatic siblings (Carrasco et al., 2013) and first degree relatives (Riesel et al., 2011) of OCD patients, suggests that ERN might be a potential candidate endophenotype for OCD. Additionally, other ERPs such as, P3 and N2 were found to have significant changes in OCD patients, compared to healthy controls. Enhanced P3 and greater negativity of N2, indicating cortical hyper-arousal and impaired response inhibition respectively, were some of these findings. Additionally, shorter N2 and P3 latencies were observed in OCD patients, which demonstrate speeding of cognitive processing, potentially due to cortical hyper-arousal (Beech et al., 1983).

7.1. Concepts

The diverse nature of EEG findings in OCD could be conceptualized in terms of defective structure and function of the frontal-striatal-thalamic (FST) circuitry (Gan et al., 2017), resulting in poor functional connectivity between different brain regions. Functional connectivity, which entails integration of information to link brain regions, is achieved via synchronization of neuronal oscillations. In the FST circuit, theta and alpha oscillations play an important role in maintaining functional connectivity (Buzsáki & Draguhn, 2004). Therefore, it can be hypothesized, subject to verification, that oscillatory abnormalities in these frequencies, as seen in OCD, may disrupt coordinated network activity, and therefore underpin the clinical symptoms of the disorder. Serotonergic antidepressants are thought to alter this oscillatory activity in some individuals by changing the activity of thalamic 'pacemaker' cells (both theta and alpha oscillations are produced by the cerebral cortex under the strong influence of thalamic pacemakers) (Leuchter et al., 2017).

An alternative approach that might explain the excess of low frequency oscillations in OCD, particularly in the theta band, is the concept of thalamo-cortical dysrhythmia (Llinás, Ribary, Jeanmonod, Kronberg, & Mitra, 1999). Hyperpolarization of thalamic cells leads to disrupted dynamic between the thalamus and cortex, resulting in pathological theta oscillations in the cortex. The areas that are anticipated to be mostly affected by thalamo-cortical dysrhythmia, leading to maximal low frequency activity are the medial prefrontal and cingulate cortices, which corresponds with the QEEG findings.

Furthermore, increased REM density, which was the most prominent sleep EEG finding in OCD patients, may be explained as the body's way of compensating for the distress associated with OCD (I. S. Fontenelle et al., 2010). Research has shown that REM sleep has the unique capacity for overnight modulation of affective networks and emotional experiences (Walker & van Der Helm, 2009).

Moreover, overactive performance monitoring in OCD, as depicted by enhanced ERN amplitude, was found to be present in OCD patients regardless of symptom manifestation. This supports the assumption that increased performance monitoring indicates the vulnerability for the disorder and a common neural correlate, rather than an expression of the disease state itself (Riesel et al., 2014). Furthermore, evoked alpha and theta oscillations during encoding and recognition phases of a stimulus are involved in generating the early components of ERPs. Therefore, altered band powers in these frequencies at the stimulus onset, as seen in OCD sufferers, may cause alterations in the resultant ERPs (Klimesch, Sauseng, Hanslmayr, Gruber, & Freunberger, 2007).

TMS-EEG holds the potential to broaden our understanding of OCD by either using EEG to monitor the changes caused by stimulation or using stimulation to specifically change EEG biomarkers of OCD (ERN, N2, P3). Some studies suggest that ERN, which is a consistently altered biomarker, is a candidate endophenotype for OCD, thereby making its use for TMS-EEG questionable. However, if stimulation causes ERN alterations, there is a possibility of changing the OCD phenotype, as behavioural changes in OCD may be driven by the same underlying processes that lead to ERN alterations. Alternatively, if changing ERN via stimulation does not improve OCD symptoms, it can be assumed that ERN changes are isolated and does not remove the associated vulnerability.

7.2. Future directions

Future research should focus on resolving the inconsistencies identified in the different aspects of EEG in OCD. The various theories hypothesizing the underlying pathophysiology of OCD should be further explored to develop novel OCD treatments. For example, brain stimulation approaches other than TMS could be explored in OCD patients in order to modulate ERPs and thereby, correct the underlying oscillatory abnormalities. TMS-EEG techniques have been helpful in analysing ERP alterations during brain stimulation in various psychiatric disorders. As OCD exhibits ERP alterations, future studies should utilize TMS-EEG to understand the underlying pathophysiology. Furthermore, the majority of the QEEG studies in OCD were conducted while the participants were resting with eyes closed. Future work should focus on analysing the EEG variations during cognitive tasks, as task-related EEG changes provide useful information on brain functionality.

7.3. Summary

OCD is a chronic, disabling mental illness causing significant impairment in the quality of life of those affected. Poor understanding of the underlying pathophysiology of the disease has caused successful treatment of OCD to remain a challenge to date. Individuals with OCD have demonstrated numerous differences in their EEG activity, compared to healthy controls. Dysfunctional frontal circuitry, impaired REM sleep and overactive performance monitoring were some of the major underlying defects identified in the literature. Furthermore, ERN findings of OCD studies point towards its potential usefulness as a candidate endophenotype. Several EEG abnormalities appear to be consistently present in OCD sufferers; namely, increased resting theta and decreased resting alpha activity in the frontotemporal region, both of which are potentially attributed to dysfunctional FST circuitry. Future work should focus on studying EEG activity in OCD sufferers during cognitive tasks to analyse potential functional deficits. Additionally, future research could focus on using TMS-EEG to further

our knowledge on the underlying brain pathologies of OCD. Continuing to study the electrophysiology of OCD will undoubtedly aid in broadening our understanding of the underlying pathophysiology of the disease.

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