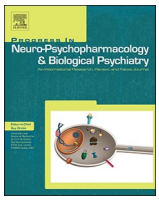
Progress in Neuropsychopharmacology & Biological Psychiatry 104 (2021) 110061

Contents lists available at ScienceDirect

Progress in Neuropsychopharmacology & Biological Psychiatry

journal homepage: www.elsevier.com/locate/pnp



Lateralized readiness potentials and sensorimotor activity in adults with obsessive-compulsive disorder

Simon Morand-Beaulieu[a,b,c,*], Frederick Aardema[a,d], Kieron P. O'Connor[a,d,1], Marc E. Lavoie[a,b,d,*]

[a] Centre de recherche de l'Institut universitaire en santé mentale de Montréal, Montréal, QC, Canada
[b] Département de neurosciences, Université de Montréal, Montréal, QC, Canada
[c] Child Study Center, Yale University School of Medicine, New Haven, CT, USA
[d] Département de psychiatrie et d'addictologie, Université de Montréal, Montréal, QC, Canada

ARTICLE INFO

Keywords:
Obsessive-compulsive disorder
Electrophysiology
Lateralized readiness potentials
Response preparation
Movement preparation

ABSTRACT

Obsessive-compulsive disorder (OCD) patients are known to have various functional abnormalities in prefrontal and motor areas. Given the presence of compulsions in many OCD patients, impaired response preparation processes could be a core feature of OCD. Yet, these processes remain understudied from a neurophysiological standpoint. Nineteen OCD patients were matched on age and sex to 19 healthy controls. Continuous EEG was recorded in all participants during a stimulus-response compatibility task. EEG from electrodes C3 and C4 was then averaged into stimulus- and response-locked LRPs. We compared both groups on various LRP measures, such as the LRP onset, the Gratton dip, and the maximum LRP peak. OCD patients showed significantly larger LRP peak than healthy controls, as well as larger Gratton dip. However, there was no group difference regarding LRP onset. Among OCD patients, it seems that motor regions are overactive during response preparation. Such overactivity was found for both incorrect responses that are aborted before execution and responses that are truly executed. These results suggest that regulation of sensorimotor activity should be addressed in the treatment of OCD.

1. Introduction

Obsessive-compulsive disorder (OCD) is a condition involving recurrent obsessive and intrusive thoughts, as well as repetitive compulsions performed in response to these thoughts (American Psychiatric Association, 2013). OCD affects between 1 and 3% of the population and is therefore among the most prevalent mental health disorders (Kessler et al., 2005; Ruscio et al., 2010). OCD affects multiple aspects of patients' lives. Notably, impairments in executive functions and other cognitive domains have been widely reported (Abramovitch et al., 2013; Snyder et al., 2014).

Given the presence of repetitive movements such as compulsions in OCD patients, many researchers have investigated action initiation processes and goal-directed behavior in OCD. It has been suggested that compulsions might be associated with deficits in goal-directed behavior as well as an overreliance on habits (Gillan et al., 2011; Gillan and Robbins, 2014; Voon et al., 2015). Such habits are thought to be mediated by stimulus-response (S-R) associations (Gottwald et al., 2018).

Along with this, a large amount of research has focused on what happens in the OCD brain when a response is inhibited. A narrative review by van Velzen et al. (2014) reported that functional abnormalities are commonly found among OCD patients in interference control and motor inhibition tasks. Despite some inconsistencies, these abnormalities mostly consist of hyperactivations of the prefrontal areas and of some structures of the cortico-striato-thalamo-cortical (CSTC) circuits. Motor regions of the brain, such as the supplementary motor area (SMA), also constitute an important correlate of response inhibition in OCD, as overactivation of both the pre-SMA (de Wit et al., 2012) and SMA (Yücel et al., 2007) has been reported during inhibitory control tasks. The pre-SMA and SMA are also overactivated in OCD patients during error processing (Norman et al., 2019). Hyperactive error processing mechanisms have been widely documented in OCD. For instance, enhanced error-related negativity has been reported quite often in the literature (Riesel, 2019).

While much research focused on the neural correlates of inhibition and error processing in OCD, only few studies have targeted the neural processes involved in response preparation. These processes are highly relevant to the study of OCD, given the recurrent urges to perform an action that affect many patients. Electroencephalography (EEG) and event-related potentials (ERP) constitute excellent options to study motor preparation processes given their high temporal precision. However,

* Corresponding authors.
E-mail addresses: simon.morand-beaulieu@yale.edu (S. Morand-Beaulieu), marc.lavoie@umontreal.ca (M.E. Lavoie).
[1] Deceased August 27, 2019

https://doi.org/10.1016/j.pnpbp.2020.110061
Received 19 December 2019; Received in revised form 13 June 2020; Accepted 3 August 2020
Available online 08 August 2020

these techniques have not been used extensively to understand how obsessive-compulsive symptomatology relates to motor preparation processes. Notably, Leocani et al. (2001) reported a delayed onset of the mu event-related EEG desynchronization prior to voluntary movements, which is thought to reflect an anomaly in motor preparation.

With the ERP technique, one can assess the readiness potential, which occurs prior to a voluntary movement and is maximal over the contralateral precentral region (Kornhuber and Deecke, 2016). In an initial investigation of this feature, Khanna et al. (1987) reported a larger readiness potential among adults with OCD, which they interpreted as a dysfunction in complex motor programming. Dayan et al. (2014) tested the readiness potential in 14 undergraduates with either low or high obsessive-compulsive symptoms. Among participants with high obsessive-compulsive symptoms, they found a larger readiness potential slope gradient, which represents the component's maximal amplitude relative to its duration. In a later study, they assessed the readiness potential in patients with clinically diagnosed OCD and replicated their initial finding regarding the gradient slope of the readiness potential, but also reported a larger amplitude of that component in OCD patients (Dayan et al., 2017). Despite these interesting results, the readiness potential might only partly reflect motor aspects of sensorimotor processing and could also involve non motor-related EEG activity. In a recent study, the same group used lateralized readiness potentials (LRP) to assess motor preparation in OCD patients (Dayan-Riva et al., 2020). LRPs are calculated as the difference in amplitude between electrodes contralateral and ipsilateral to the responding hand (Slobounov, 2010). By doing so, it is possible to eliminate cortical activity that is unrelated to motor processes. Furthermore, one of the generators of the LRP is the SMA (Deecke et al., 1984; Rektor, 2002), which appears to be overactivated in OCD patients during inhibition and error processing. In a stimulus-response compatibility (SRC) task, LRPs are used to evaluate the motor preparation processes when the compatibility between a stimulus and the expected response varies. In their study, Dayan-Riva et al. (2020) reported larger stimulus-locked LRP (sLRP) amplitude among OCD patients, only in the incompatible condition of an SRC task. However, they did not assess the Gratton dip (activation of the incompatible response) nor the response-locked LRP (rLRP). Consistent with these findings and our previous studies on Tourette syndrome patients (Morand-Beaulieu et al., 2018; 2015), we hypothesized that OCD patients would show larger Gratton dip and sLRP and rLRP peak, as well as a delayed sLRP onset.

2. Methods

2.1. Participants

Nineteen OCD patients were included in the current study. They were initially recruited as part of a larger project on an inference-based treatment for OCD (Aardema et al., 2017). Criteria for inclusion in the current study were to (i) be aged between 18 and 65 years old and to (ii) have a primary diagnosis of OCD as defined by the DSM-IV-TR. Criteria for exclusion were: (i) current or past diagnosis of schizophrenia, bipolar disorder, or organic mental disorder; (ii) presence of a neurological disorder; (iii) head injury in the last year; (iv) have changed antidepressant medication type or dose within the last 12 weeks; and to (v) have changed anxiolytics medication type or dose within the last four weeks. Eleven patients were under psychiatric medication at the time of the study (selective serotonin reuptake inhibitors (n = 8), antipsychotics[2] (n = 5), benzodiazepines (n = 3), serotonin-norepinephrine reuptake inhibitors (n = 2), serotonin antagonist and reuptake inhibitors (n = 1), norepinephrine-dopamine reuptake inhibitors (n = 1), and alpha-2 receptors agonists (n = 1)). Fourteen patients had comorbid disorders (generalized anxiety disorder (n = 6), motor tics (n = 3), vocal tics (n = 1), major depressive disorder (n = 2), social anxiety (n = 1), body-focused repetitive behaviors (n = 1), adjustment disorder (n = 1), anorexia (n = 1), and substance use disorder (n = 1)).

OCD patients were matched on age and sex with 19 healthy controls. Inclusion criterion for healthy controls was to be aged between 18 and 65 years old, while exclusion criteria were to (i) have an history of neurological or psychiatric disorder; (ii) head injury in the last year; (iii) psychiatric medication intake; and (iv) misuse of drugs or alcohol. Socio-demographic and clinical characteristics for all participants can be found in Table 1. This study was approved by the review board of the *Institut universitaire en santé mentale de Montréal* and conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from all participants before taking part in the study.

2.2. Procedures

2.2.1. Clinical assessment

We assessed anxiety and depressive symptoms in both OCD patients and healthy controls, with the Beck Anxiety Inventory (BAI; Beck et al., 1988) and the Beck Depression Inventory (BDI; Beck et al., 1961), respectively. We also assessed impulsivity symptoms with the Barratt Impulsiveness Scale (BIS-10; Patton et al., 1995). To ensure correct basic perceptual performance during the stimulus-response compatibility (SRC) task, visual acuity (Snellen) and color perception (Ishihara) were assessed prior to testing. In OCD patients, symptoms' severity was assessed with the Padua Inventory-Revised (Van Oppen et al., 1995).

2.2.2. Experimental procedure

The SRC task consisted in left- and right-pointing colored arrows that were presented for 200 ms with a pseudo-randomized inter-stimulus interval ranging from 1500 to 1800 ms. Participants were asked to press the keyboard key corresponding to the direction of the arrow when blue arrows were presented (compatible condition), and the key opposed to the direction of the arrow when black arrows were presented (incompatible condition). Red arrows were No-Go stimuli (not used in LRP analyses). Participants were instructed to give no response to these stimuli (see Fig. 1). There were 100 compatible, 100 incompatible, and 50 No-Go stimuli. Stimuli were presented in a pseudo-random order during a single block and left- and right-pointing arrows were equally distributed in each condition. The task was monitored with the Presentation software (Neurobehavioral Systems, Albany, CA, USA). In both groups. behavioral data, such as accuracy, reaction times (RT), and intra-individual variability in reaction times (RT variability) were extracted. RT variability was measured as the mean standard deviation in RT for each participant.

2.3. Electrophysiological recordings

During the SRC task, EEG signal was recorded continuously at a sampling rate of 500 Hz from 62 Ag/AgCl electrodes mounted in a lycra cap (Electrode Arrays, El Paso, TX, USA). Electrodes position was consistent with standard EEG guidelines (American EEG Society, 1994). All electrodes were reference to the nose. The signal was digitally amplified (Sensorium Inc., Charlotte, VT, USA) and recorded with IWave software (InstEP Systems, Montréal, QC, Canada). Online filtering involved a 0.01 Hz high-pass filter and a 100 Hz low-pass filter. An electrolyte gel (JNetDirect Biosciences, Herndon, VA) was used to keep impedance below 5KΩ. Additional electrodes were placed below and above the left eye and at the outer canthus of each eye to correct ocular artifacts with the Gratton algorithm (Gratton et al., 1983).

2.4. Averaging procedure

The continuous EEG signal was averaged offline and time-locked to stimulus and response onset. Offline filtering a 0.3 Hz high-pass filter and a 30 Hz low-pass filter. Clippings due to amplifiers saturation and

[2] Antipsychotics included quetiapine (n = 3) and risperidone (n = 2).

Table 1
Socio-demographic and clinical characteristics.

	OCD patients		Healthy controls				
	Mean	SD	Mean	SD	t	p	d
Age	44	15.4	43	13.9	0.40	0.69	0.13
Sex (M:W)	9:10	N/A	9:10	N/A	0.00[a]	1.00[a]	N/A
Handedness (R:L:A)	17:2:0	N/A	18:0:1	N/A	2.59[b]	.49[b]	N/A
Anxiety (BAI)	15	11.6	5	5.3	**3.39**	**0.002**	1.10
Depression (BDI)	18	7.6	4	4.3	**7.10**	**< 0.001**	2.29
Impulsivity (BIS-10)	71	7.1	67	7.8	1.48	0.15	0.48
Y-BOCS total	27	6.2	–	–	–	–	–
Y-BOCS obsessions	14	3.3	–	–	–	–	–
Y-BOCS compulsions	13	3.9	–	–	–	–	–
PI-R total	74	36.5	–	–	–	–	–
PI-R impulses	7	6.7	–	–	–	–	–
PI-R washing	15	12.8	–	–	–	–	–
PI-R checking	16	10.8	–	–	–	–	–
PI-R rumination	31	17.0	–	–	–	–	–
PI-R precision	6	5.2	–	–	–	–	–
Comorbid disorders	14/19	–	–	–	–	–	–
Psychiatric medication	11/19	–	–	–	–	–	–

A, ambidextrous; BDI, Beck Depression Inventory; BAI, Beck Anxiety Inventory; L, left-handed; PI-R, Padua Inventory-Revised; R, right-handed; SD, Standard deviation; Y-BOCS, Yale-Brown Obsessive Compulsive Scale.
Significant results are highlighted in bold.
[a] Chi-squared test.
[b] Fisher's exact test.

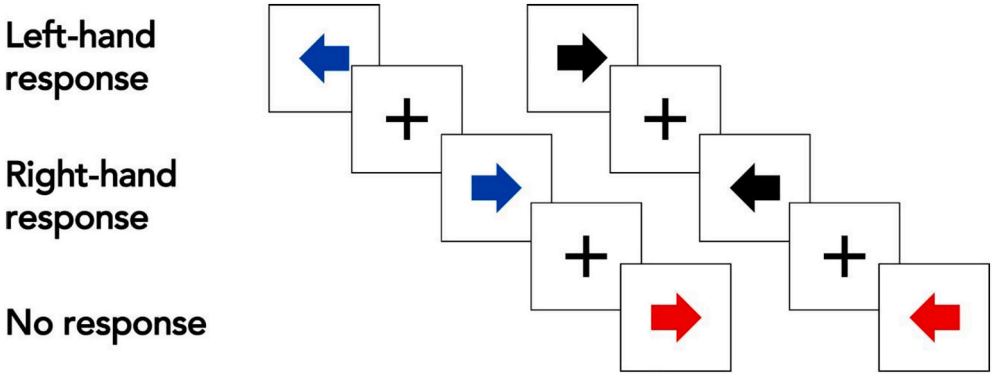


Fig. 1. Stimulus-response compatibility task. Blue, black, and red arrows were presented on a computer screen. For blue arrows, participants had to press the keyboard key corresponding to the direction of the arrow. For black arrows, participants had to press the keyboard key opposed to the direction of the arrow. For red arrows, participants had to refrain from pressing a key. This figure was adapted from Morand-Beaulieu et al. (2015). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

remaining epochs exceeding 100 μV were removed. For LRP analyses, we specifically targeted the C3 and C4 electrodes. LRPs were computed with the averaging method: LRP = $\frac{[\text{Mean}(C4-C3)^{\text{left hand}} + \text{Mean}(C3-C4)^{\text{right hand}}]}{2}$ (Coles, 1989). sLRPs were baseline corrected from −100 ms to stimulus onset, while rLRPs were baseline corrected from −750 to −500 ms before motor response. The sLRP peak and onset were measured from 150 to 900 ms following stimulus onset, while the rLRP peak and onset were measured from 500 to 0 ms before the motor response was given. To assess the sLRP onset, a relative criterion method was used (Mordkoff and Gianaros, 2000), which was set at 20% of the maximum peak. The Gratton dip was scored as the positive area under the curve between 150 and 350 ms following incompatible stimulus onset.

2.5. Statistical analysis

Between-group comparisons of socio-demographic and clinical data were performed with independent-samples *t*-tests. Behavioral data were analyzed with mixed ANOVAs, with the between-subjects factor Group (OCD/HC), and the within-subjects factor Condition (compatible/incompatible/No-Go (only for accuracy analyses)). Both sLRP and rLRP peaks and onsets were analyzed with mixed ANOVAs, with the between-subjects factor Group (OCD/HC), and the within-subjects factor Condition (compatible/incompatible). The Gratton dip was analyzed with an independent-samples t-test. Post-hoc tests were performed with the Bonferroni method. Effect sizes were reported with Cohen's d for pairwise comparisons and partial eta-squared (ηp^2) for interactions and variables with more than two levels. We also performed Spearman's rank-order correlations between LRP measures and clinical scales.

3. Results

3.1. Behavioral performance

There was a condition by group interaction regarding reaction times [$F(1,36) = 4.66$, $p = .04$, $\eta p^2 = 0.12$]. Healthy controls showed a typical condition effect, with faster reaction times for compatible than incompatible stimuli [$F(1,18) = 17.63$, $p = .001$, $d = 0.50$]. This effect was however absent in OCD patients [$F(1,18 = 2.53$, $p = .13$, $d = 0.14$].

RT variability was larger in the compatible than in the incompatible condition [$F(1,35) = 6.66$, $p = .01$, $d = 0.20$].[3] There was also a trend toward a group by condition interaction [$F(1,35) = 3.37$, $p = .08$, $\eta p^2 = 0.09$], suggesting larger RT variability for compatible than incompatible stimuli among OCD patients [$F(1,17) = 9.51$, $p < .01$, $d = 0.38$]. No such difference was found in healthy controls [$F(1,18) = 0.28$, $p = .60$, $d = 0.06$].

[3] RT variability data was missing for one OCD patient.

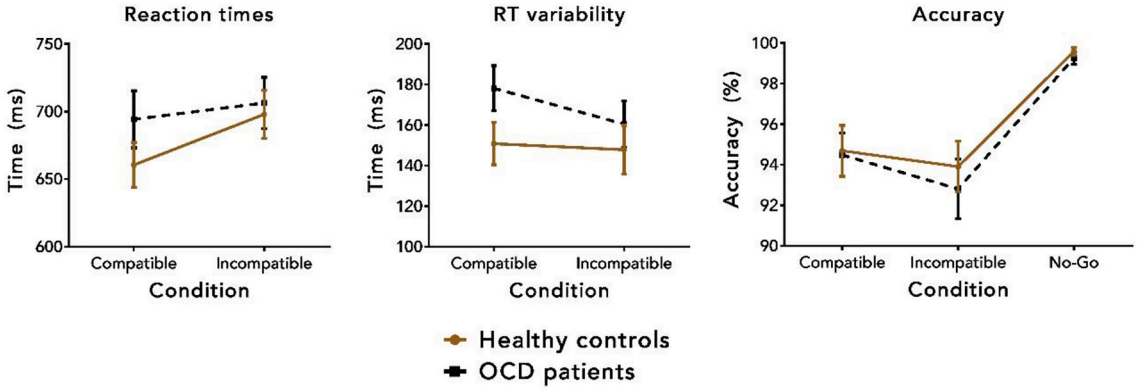


Fig. 2. Behavioral data. In healthy controls (solid gold line), reaction times were faster for compatible than incompatible stimuli. This condition effect was not found in OCD (dashed black line) patients. However, there was a condition effect regarding RT variability in OCD patients, with larger variability for compatible than incompatible stimuli. Such condition effect was not present in healthy controls. Both groups had similar task accuracy, with less errors in the No-Go than the compatible and incompatible conditions. Error bars represent the standard error of the mean (SEM). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

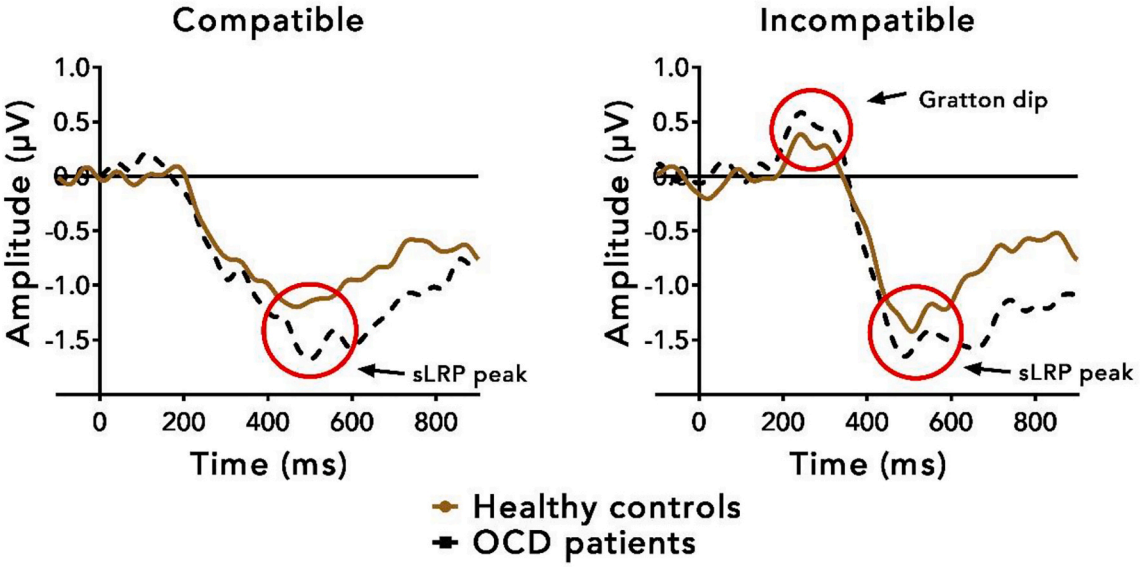


Fig. 3. Stimulus-locked LRP (sLRP) waveforms. sLRPs were obtained from electrodes C3 and C4. Data were filtered with a 12 Hz low-pass filter for display purposes only. In the incompatible condition, the sLRP onset is preceded by a positive deflection in amplitude toward the incorrect response, which is called the Gratton dip.

The analysis on task accuracy revealed a condition effect [$F(2,72) = 30.85, p < .001, \eta p^2 = 0.46$], with better accuracy for No-Go than compatible [$p < .001$, $d = 1.35$] and incompatible [$p < .001$, $d = 1.34$] stimuli. However, there was no main effect or interaction involving the Group factor [all *p*-values > .6] (see Fig. 2).

3.2. Electrophysiological results

3.2.1. Stimulus-locked lateralized readiness potentials (sLRP)

sLRP waveforms are presented in Fig. 3. The Gratton dip (incorrect activation between 150 and 350 ms), which precedes the incompatible sLRP onset, was larger in OCD patients than in healthy controls [$t(27.90) = 2.08, p = .047, d = 0.68$]. The compatible sLRP onset occurred at around 249 ms. Given the presence of a Gratton dip, the incompatible sLRP onset occurred later (366 ms) than the compatible sLRP onset [$F(1,36) = 54.38, p < .001, d = 1.68$]. However, there was no main effect or interaction involving the Group factor [all *p*-values > .4]. Finally, the sLRP peak (correct activation peaking around 541 ms) was larger in OCD patients [$F(1,36) = 8.60, p < .01, d = 0.95$]. There was no condition main effect or interaction regarding the sLRP peak [all p-values > .7] (see Fig. 5A).

3.2.2. Response-locked lateralized readiness potentials (rLRP)

rLRP waveforms are presented in Fig. 4. The compatible and incompatible rLRP onsets occurred at 242 ms and 247 ms before the response, respectively. There was no difference between both conditions and there were no main effect or interaction involving the Group factor either [all p-values > .2]. The rLRP peak (correct activation peaking around 130 ms before the response) was larger in OCD patients [$F(1,36) = 4.70, p = .04, d = 0.70$]. There was no condition main effect or interaction regarding the rLRP peak [all p-values > .2] (see Fig. 5B).

3.3. Correlational analyses

Among all participants, more depressive symptoms as assessed by the BDI were associated to a larger compatible [$r_s(38) = -0.37$, $p = .02$] and incompatible [$r_s(38) = -0.38$, $p = .02$] sLRP peak. It is unlikely that depression alone may explain group difference regarding sLRP peak, since this result tended to remain significant even when the BDI was added as a covariate [$F(1,35) = 4.02, p = .053$]. However, the BDI was not associated to the rLRP peak. The BDI was also correlated to the incompatible rLRP onset [$r_s(38) = -0.32$, $p = .05$], suggesting that delayed rLRP onset is associated to more depressive symptoms.

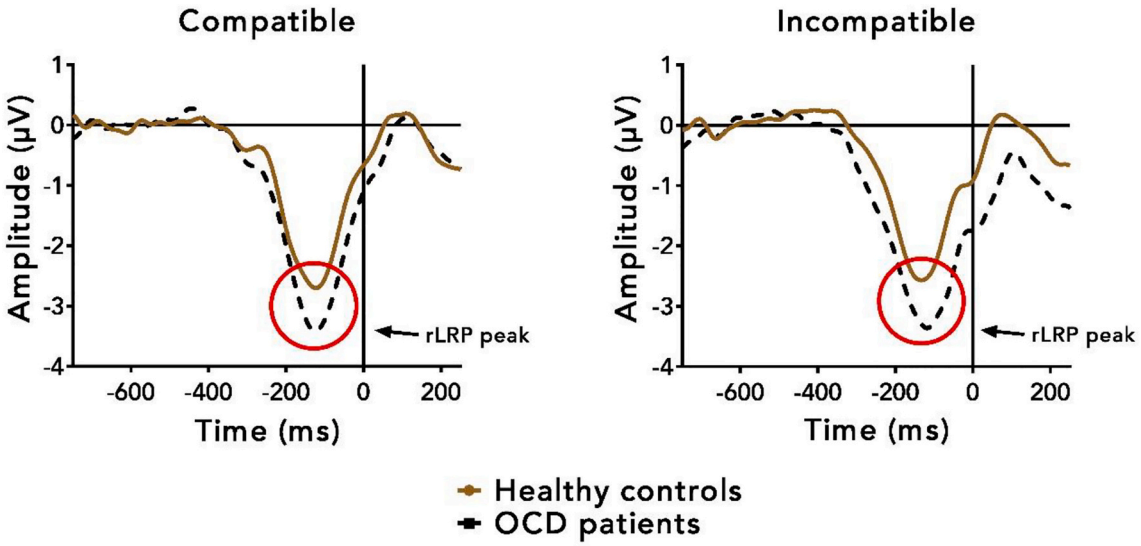


Fig. 4. Response-locked LRP (rLRP) waveforms. rLRPs were obtained from electrodes C3 and C4. The rLRP peak was larger in OCD patients. Data were filtered with a 12 Hz low-pass filter for display purposes only.

However, none of the LRP measures were significantly correlated to BAI and BIS-10 scores. Among OCD patients, we did not find a significant correlation between LRP measures and obsessive and compulsive symptoms as measured by the PI-R global score.

3.4. Supplementary analyses

In order to control the possible impact of psychiatric medication on LRP measures, we compared the sLRP and rLRP peaks and onsets, as

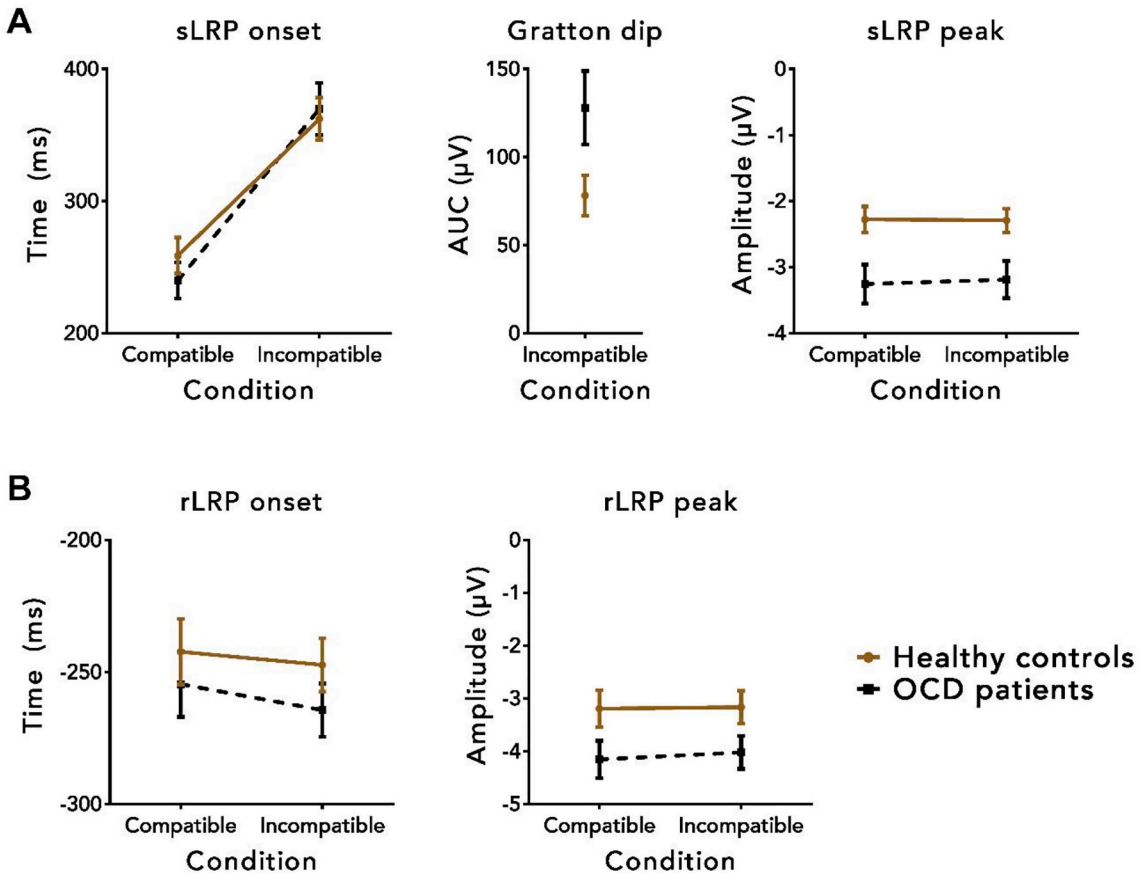


Fig. 5. Differences in LRP measures. (A) There was no between-group difference regarding the sLRP onset. The Gratton dip, which was assessed as the positive area under the curve (AUC) between 150 and 350 ms after incompatible stimuli presentation, was larger among OCD patients (dashed black line) than healthy controls (solid gold line). The sLRP peak was also larger among OCD patients, no matter the condition. (B) There was no between-group difference regarding the rLRP onset. The rLRP peak was larger among OCD patients than healthy controls. Error bars represent the standard error of the mean (SEM). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

well as the Gratton dip, between medicated and non-medicated OCD patients using mixed ANOVAs and independent samples *t*-test. However, there were no between-group differences [all *p*-values > .11]. Given the possible impact of antipsychotics on the motor system, we also conducted mixed ANOVAs and independent samples t-test to test for differences in LRP measures between patients using antipsychotics medication and those who were not. Here again, there were no between-group differences [all p-values > .14].

4. Discussion

Both groups performed the task with similar accuracy, but OCD patients did not show the typical stimulus-response congruency effect in reaction times that was observed in healthy controls. Previous studies that used a Simon task in OCD patients have found reaction times that were similar to that of healthy controls (Marsh et al., 2014; Wolff et al., 2019). In stimulus-response compatibility paradigms, the addition of a No-Go component increases task complexity (Schapkin et al., 2007). In OCD patients, deficits in inhibitory performance typically occur at a lower task complexity than in healthy controls (van Velzen et al., 2014). Therefore, the presence of a No-Go component in our experiment could explain why OCD patients did not show the expected compatibility effect. The absence of this stimulus-response compatibility effect could also be explained by larger RT variability for compatible stimuli among OCD patients, which might negatively impact reaction times in this condition.

The main finding of the current study is that OCD patients exhibited larger electrocortical activity during movement preparation. This was true for incorrect activations that are aborted before execution (Gratton dip) and responses that are fully executed (LRP peak). This replicates earlier findings (Dayan et al., 2017; Dayan-Riva et al., 2020) reporting increased readiness potential/LRP in OCD patients. In their most recent study, Dayan-Riva et al. (2020) reported increased sLRP amplitude among OCD patients only in the incompatible condition, which slightly differs from our own findings. However, they conducted follow-up analyses in the absence of a group by condition interaction. It is therefore possible that both conditions contribute to the observed group difference, as in our study. Also, while they studied LRPs with an SRC task, they did not assess the Gratton dip. Therefore, our study is the first evidence of an enhanced Gratton dip among OCD patients. It is unlikely that this enhanced component stem from larger competition between responses, since high conflict between responses typically leads to a reduced LRP (Frame et al., 2018; Kappenman et al., 2012). Larger amplitudes of both the Gratton dip and the LRP amplitude rather suggests overactivation of both incorrect and correct responses which could involve a relatively specific motor function anomaly. Motor functions might be an important part of the OCD pathophysiology. The meta-analysis of Snyder et al. (2014) revealed a moderate effect size regarding the performance on the Trail Making Test Part A and other measures of motor performance, which suggests slightly impaired global motor speed. Another meta-analysis reported abnormal grey matter volume and reduced activations in the cerebellum during motor inhibition tasks, which suggests abnormal sensorimotor processing among OCD patients (Eng et al., 2015). Also, poor performances in a motor task in childhood has previously been associated to the persistence of OCD symptoms into adulthood (Bloch et al., 2011). Another study also revealed that individuals who develop OCD in adulthood had worse performance in a motor task than those who did not develop OCD (Grisham et al., 2009).

In our sample, premotor processes did not correlate with OCD symptomatology as measured by the PI-R. With a limited sample size, it is hazardous to make hard conclusion regarding the non-association of premotor processes and obsessive or compulsive symptoms. However, the absence of such correlations suggests that abnormalities in action initiation might not be directly associated to the severity of symptoms but to the presence of OCD *per se*. We invite researchers to replicate our findings with larger samples and by evaluating the OC symptoms in sub-clinical populations as well, which would also allow to characterize the determinants of LRP amplitude in a wider spectrum of OC disorders and behaviors.

The regulation of sensorimotor activity in OCD patients could constitute an interesting target for treatment. Prior studies have shown that repetitive transcranial magnetic stimulation (rTMS) applied to the SMA (especially low-frequency rTMS) induced a decrease in OCD symptoms (Mantovani et al., 2010; Rehn et al., 2018). In Tourette syndrome, which shares many clinical and neurobiological features with OCD, a CBT with a particular aim on reducing muscular tension has shown its efficacy to normalize excessive brain activity and sensorimotor activation (Morand-Beaulieu et al., 2018). Therefore, including such physiological dimensions in the psychological treatment of OCD might further increase symptoms improvement.

5. Limitations

One of the main limitations of the current study is the limited sample size. Larger samples would allow a better understanding of the factors involved in the motor overactivation that we reported. Yet, our sample was larger than those included in the initial studies of Dayan-Riva et al. (2014, 2017) and the effects we reported were fairly robust. Another limitation of this study is that 11 of the 19 patients were under medication, and 5 of them were using antipsychotics. We conducted supplementary analyses to assess differences in LRP measures between medicated and non-medicated patients, which did not reveal differences between these subgroups. However, these subgroups have a limited size and further studies are warranted to fully understand the impact of psychiatric medication on motor preparation in OCD.

6. Conclusion

In conclusion, we found larger Gratton dip and LRP peak in OCD patients, in comparison with healthy controls. Such results suggest an overactivation of motor regions of the brain – notably the SMA – during action preparation. This motor overactivation might constitute an interesting treatment target.

Declaration of Competing Interest

The authors have no conflict of interests to report.

Acknowledgements

This study was supported by grants from the Canadian Institutes of Health Research (CIHR; RCT244288 and MOP84573) awarded to KPO, MEL, and FA. SMB was supported by a doctoral scholarship from the Université de Montréal and a postdoctoral fellowship award from the CIHR (415541). We wish to thank Martine Germain for electrophysiological testing and Karine Bergeron for clinical coordination. We also would like to thank all the patients and healthy controls who participated in this study.

References

Aardema, F., O'Connor, K.P., Delorme, M.-E., Audet, J.-S., 2017. The inference-based approach (IBA) to the treatment of obsessive–compulsive disorder: an open trial across symptom subtypes and treatment-resistant cases. Clin. Psychol. Psychother. 24 (2), 289–301.

Abramovitch, A., Abramowitz, J.S., Mittelman, A., 2013. The neuropsychology of adult obsessive-compulsive disorder: a meta-analysis. Clin. Psychol. Rev. 33 (8), 1163–1171.

American EEG Society, 1994. Guideline thirteen: guidelines for standard electrode position nomenclature. J. Clin. Neurophysiol. 11 (1), 111–113.

American Psychiatric Association, 2013. Diagnostic and Statistic Manual of Mental Disorders, 5th ed. Author, Arlington, VA.

Beck, A.T., Ward, C.H., Mendelson, M., Mock, J., Erbaugh, J., 1961. An inventory for measuring depression. Arch. Gen. Psychiatry 4, 561–571.

Beck, A.T., Epstein, N., Brown, G., Steer, R.A., 1988. An inventory for measuring clinical anxiety: psychometric properties. J. Consult. Clin. Psychol. 56 (6), 893–897.

Bloch, M.H., Sukhodolsky, D.G., Dombrowski, P.A., Panza, K.E., Craiglow, B.G., Landeros-Weisenberger, A., Leckman, J.F., Peterson, B.S., Schultz, R.T., 2011. Poor fine-motor and visuospatial skills predict persistence of pediatric-onset obsessive-compulsive disorder into adulthood. J. Child Psychol. Psychiatry 52 (9), 974–983.
Coles, M.G., 1989. Modern mind-brain reading: psychophysiology, physiology, and cognition. Psychophysiology 26 (3), 251–269.
Dayan, A., Berger, A., Anholt, G.E., 2014. Enhanced action tendencies in high versus low obsessive-compulsive symptoms: an event-related potential study. Psychiatry Res. Neuroimaging 224 (2), 133–138.
Dayan, A., Berger, A., Anholt, G.E., 2017. Enhanced action tendencies in obsessive-compulsive disorder: an ERP study. Behav. Res. Ther. 93, 13–21.
Dayan-Riva, A., Berger, A., Anholt, G.E., 2020. Affordances, response conflict, and enhanced-action tendencies in obsessive-compulsive disorder: an ERP study. Psychol. Med. 1–16.
Deecke, L., Bashore, T., Brunia, C.H., Grunewald-Zuberbier, E., Grunewald, G., Kristeva, R., 1984. Movement-associated potentials and motor control. Report of the EPIC VI Motor Panel. Ann. New York Acad. Sci. 425, 398–428.
Eng, G.K., Sim, K., Chen, S.H., 2015. Meta-analytic investigations of structural grey matter, executive domain-related functional activations, and white matter diffusivity in obsessive compulsive disorder: an integrative review. Neurosci. Biobehav. Rev. 52, 233–257.
Frame, M.E., Johnson, J.G., Thomas, R.D., 2018. A neural indicator of response competition in preferential choice. Decision 5 (4), 272–286.
Gillan, C.M., Robbins, T.W., 2014. Goal-directed learning and obsessive-compulsive disorder. Philos. Trans. Roy. Soc. Lond. 369, 1655.
Gillan, C.M., Papmeyer, M., Morein-Zamir, S., Sahakian, B.J., Fineberg, N.A., Robbins, T.W., de Wit, S., 2011. Disruption in the balance between goal-directed behavior and habit learning in obsessive-compulsive disorder. Am. J. Psychiatry 168 (7), 718–726.
Gottwald, J., de Wit, S., Apergis-Schoute, A.M., Morein-Zamir, S., Kaser, M., Cormack, F., Sule, A., Limmer, W., Morris, A.C., Robbins, T.W., Sahakian, B.J., 2018. Impaired cognitive plasticity and goal-directed control in adolescent obsessive-compulsive disorder. Psychol. Med. 48 (11), 1900–1908.
Gratton, G., Coles, M.G., Donchin, E., 1983. A new method for off-line removal of ocular artifact. Electroencephalogr. Clin. Neurophysiol. 55 (4), 468–484.
Grisham, J.R., Anderson, T.M., Poulton, R., Moffitt, T.E., Andrews, G., 2009. Childhood neuropsychological deficits associated with adult obsessive-compulsive disorder. Br. J. Psychiatry 195 (2), 138–141.
Kappenman, E.S., Kaiser, S.T., Robinson, B.M., Morris, S.E., Hahn, B., Beck, V.M., Leonard, C.J., Gold, J.M., Luck, S.J., 2012. Response activation impairments in schizophrenia: evidence from the lateralized readiness potential. Psychophysiology 49 (1), 73–84.
Kessler, R.C., Chiu, W.T., Demler, O., Merikangas, K.R., Walters, E.E., 2005. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Arch. Gen. Psychiatry 62 (6), 617–627.
Khanna, S., Mukundan, C.R., Channabasavanna, S.M., 1987. Obsessive compulsive disorder: is it a problem of complex motor programming? Indian J. Psychiatry 29 (1), 41–47.
Kornhuber, H.H., Deecke, L., 2016. Brain potential changes in voluntary and passive movements in humans: readiness potential and reafferent potentials. Pflugers Arch. - Eur. J. Physiol. 468 (7), 1115–1124.
Leocani, L., Locatelli, M., Bellodi, L., Fornara, C., Henin, M., Magnani, G., Mennea, S., Comi, G., 2001. Abnormal pattern of cortical activation associated with voluntary movement in obsessive-compulsive disorder: an EEG study. Am. J. Psychiatry 158 (1), 140–142.
Mantovani, A., Simpson, H.B., Fallon, B.A., Rossi, S., Lisanby, S.H., 2010. Randomized sham-controlled trial of repetitive transcranial magnetic stimulation in treatment-resistant obsessive-compulsive disorder. Int. J. Neuropsychopharmacol. 13 (2), 217–227.
Marsh, R., Horga, G., Parashar, N., Wang, Z., Peterson, B.S., Simpson, H.B., 2014. Altered activation in fronto-striatal circuits during sequential processing of conflict in unmedicated adults with obsessive-compulsive disorder. Biol. Psychiatry 75 (8), 615–622.
Morand-Beaulieu, S., O'Connor, K.P., Sauvé, G., Blanchet, P.J., Lavoie, M.E., 2015. Cognitive-behavioral therapy induces sensorimotor and specific electrocortical changes in chronic tic and Tourette's disorder. Neuropsychologia 79 (Part B), 310–321.
Morand-Beaulieu, S., O'Connor, K.P., Blanchet, P.J., Lavoie, M.E., 2018. Electrophysiological predictors of cognitive-behavioral therapy outcome in tic disorders. J. Psychiatr. Res. 105, 113–122.
Mordkoff, J.T., Gianaros, P.J., 2000. Detecting the onset of the lateralized readiness potential: a comparison of available methods and procedures. Psychophysiology 37 (3), 347–360.
Norman, L.J., Taylor, S.F., Liu, Y., Radua, J., Chye, Y., De Wit, S.J., Huyser, C., Karahanoglu, F.I., Luks, T., Manoach, D., Mathews, C., Rubia, K., Suo, C., van den Heuvel, O.A., Yücel, M., Fitzgerald, K., 2019. Error processing and inhibitory control in obsessive-compulsive disorder: a meta-analysis using statistical parametric maps. Biol. Psychiatry 85 (9), 713–725.
Patton, J.H., Stanford, M.S., Barratt, E.S., 1995. Factor structure of the Barratt impulsiveness scale. J. Clin. Psychol. 51 (6), 768–774.
Rehn, S., Eslick, G.D., Brakoulias, V., 2018. A meta-analysis of the effectiveness of different cortical targets used in repetitive transcranial magnetic stimulation (rTMS) for the treatment of obsessive-compulsive disorder (OCD). Psychiatr. Quart. 89 (3), 645–665.
Rektor, I., 2002. Scalp-recorded Bereitschaftspotential is the result of the activity of cortical and subcortical generators–a hypothesis. Clin. Neurophysiol. 113 (12), 1998–2005.
Riesel, A., 2019. The erring brain: error-related negativity as an endophenotype for OCD-A review and meta-analysis. Psychophysiology 56 (4), e13348.
Ruscio, A.M., Stein, D.J., Chiu, W.T., Kessler, R.C., 2010. The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. Mol. Psychiatry 15 (1), 53–63.
Schapkin, S.A., Falkenstein, M., Marks, A., Griefahn, B., 2007. Practice-related effects in a go-Nogo task. Perceptual Motor skills 105 (3_suppl), 1275–1288.
Slobounov, S.M., 2010. Event-related potentials: Slow potentials. In: Kompoliti, K., Metman, L.V. (Eds.), Encyclopedia of Movement Disorders. Academic Press, Oxford, pp. 456–458.
Snyder, H.R., Kaiser, R.H., Warren, S.L., Heller, W., 2014. Obsessive-compulsive disorder is associated with broad impairments in executive function: a meta-analysis. Clin. Psychol. Sci. 3 (2), 301–330.
Van Oppen, P., Hoekstra, R.J., Emmelkamp, P.M., 1995. The structure of obsessive-compulsive symptoms. Behav. Res. Ther. 33 (1), 15–23.
van Velzen, L.S., Vriend, C., de Wit, S.J., van den Heuvel, O.A., 2014. Response inhibition and interference control in obsessive-compulsive spectrum disorders. Front. Hum. Neurosci. 8, 419.
Voon, V., Derbyshire, K., Ruck, C., Irvine, M.A., Worbe, Y., Enander, J., Schreiber, L.R., Gillan, C., Fineberg, N.A., Sahakian, B.J., Robbins, T.W., Harrison, N.A., Wood, J., Daw, N.D., Dayan, P., Grant, J.E., Bullmore, E.T., 2015. Disorders of compulsivity: a common bias towards learning habits. Mol. Psychiatry 20 (3), 345–352.
de Wit, S.J., de Vries, F.E., van der Werf, Y.D., Cath, D.C., Heslenfeld, D.J., Veltman, E.M., van Balkom, A.J., Veltman, D.J., van den Heuvel, O.A., 2012. Presupplementary motor area hyperactivity during response inhibition: a candidate endophenotype of obsessive-compulsive disorder. Am. J. Psychiatry 169 (10), 1100–1108.
Wolff, N., Chmielewski, W., Buse, J., Roessner, V., Beste, C., 2019. Paradoxical response inhibition advantages in adolescent obsessive compulsive disorder result from the interplay of automatic and controlled processes. NeuroImage 23, 101893.
Yücel, M., Harrison, B.J., Wood, S.J., Fornito, A., Wellard, R.M., Pujol, J., Clarke, K., Phillips, M.L., Kyrios, M., Velakoulis, D., Pantelis, C., 2007. Functional and biochemical alterations of the medial frontal cortex in obsessive-compulsive disorder. Arch. Gen. Psychiatry 64 (8), 946–955.