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RESEARCH ARTICLE

Inhibition-related differences between tic-free and tic-related obsessive–compulsive disorder: evidence from the N2 and P3

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Abstract

Tic-related OCD (obsessive–compulsive disorder) was introduced as an OCD subtype in the DSM-5 based mainly on family and clinical data that showed differences between OCD in dependence of accompanying tics. Little is known, however, regarding neurocognitive differences between subtypes. We used the stop-signal task to examine whether differences exist in response inhibition between OCD patients without tics ($n = 21$), patients with tic-related OCD ($n = 12$), and 21 healthy controls. The groups were carefully matched for gender, age and level of education. The stop-signal reaction time (SSRT) and inhibition-related N2 and P3 were used to examine behavioral and neural correlates of response inhibition and inhibition-related processes. In the SSRT, no difference was found between groups. P3 amplitude was larger in tic-free compared to tic-related OCD and healthy controls. No group differences were found in the N2 amplitude. For tic-related OCD, SSRT data indicate intact response inhibition, and P3 data indicate intact neural aftereffects of inhibition like the evaluation of the outcome. This is similar to what is found in patients with TD and may, thus, be interpreted as a support for shared mechanisms in relation to TD. In OCD, alterations in P3 amplitude indicate hyperactivity in the evaluation of the outcome of the inhibition process. This is in line with hyperactivity generally found in performance monitoring in OCD.

Keywords Obsessive–compulsive disorder · Tic disorders · EEG · Electroencephalography · N2 · P3 · Response inhibition

Introduction

One approach in the unmasking of the pathopsychology of obsessive–compulsive disorder (OCD) is the identification of subtypes, i.e., the dissection of the phenotype into more homogenous groups based on the presence of mutually exclusive features (Miguel et al. 2005). One candidate subtype is tic-related OCD which is defined by the lifetime presence of tic disorders (TD) in OCD. Pauls et al. proposed that the tic-related OCD subtype is influenced by the genetic factors associated with TD and that it may represent a variant expression of genetic TD vulnerability (Pauls et al. 1986). This should lead to shared psychopathological mechanisms between tic-related OCD and TD. In fact, tic-related OCD differs from tic-free OCD clinically (e.g., early-onset, more males, reduced responses to selective serotonin response inhibitors (SSRIs) (Kloft et al. 2018; Leckman et al. 2010), and genetically (Yu et al. 2015). In line with this evidence, tic-related OCD was introduced as a specifier in the DSM-5.

In contrast to the large number of studies that examined the clinical features of tic-related OCD, little is known about potential differences in neurocognition, although studies point to differences in sensory–motor integration (Ahmari et al. 2012), and performance monitoring (Hanna et al. 2012). A central neurocognitive dysfunction in OCD is deficient response inhibition which is an important facet of performance monitoring. Dysfunctional response inhibition has been related to difficulties of OCD patients to stop their obsessions and inhibit their compulsions. It is often

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measured in the stop-signal task which requires subjects to respond with a button press towards an imperative stimulus and to inhibit their response once a stop signal occurs (Logan and Cowan 1984). The main outcome measure is the stop-signal reaction time (SSRT) which measures the time that is needed to stop an already initiated response. The SSRT has been repeatedly found to be longer in patients with OCD (see Lipszyc and Schachar 2010) and also their unaffected first-degree relatives (Chamberlain et al. 2007; Menzies et al. 2007). However, several studies did not find altered response inhibition as well (e.g., Kalanthroff et al. 2017). One reason for inconsistencies between studies could be heterogeneity of OCD patients in terms of underlying brain mechanisms which could be linked to phenotypical differences (e.g., subtypes). Contrary to what is seen in OCD, tics are associated with normal or only marginally longer SSRTs (Johannes et al. 2001; Li et al. 2006; Roessner et al. 2008). This was also corroborated in a recent study showing that response inhibition is only impaired in children with OCD but intact in children with TD or comorbid OCD + TD (Mancini et al. 2018). In addition, the neurobiological structures underlying intact response inhibition (cingulo-opercular network in addition to supplementary motor areas, inferior frontal gyrus and parietal regions) and those associated with OCD, overlap (de Wit et al. 2012; Menzies et al. 2007; van Velzen et al. 2014). In contrast, the neural alterations found during response inhibition in patients with TD are limited to the motor cortices (Ganos et al. 2014; Thomalla et al. 2014).

Event-related brain potentials (ERPs) are very fruitful to gain a deeper understanding of the brain processes related to behavioral response inhibition and their temporal dynamics. The stop-signal task evokes two ERPs of main interest: the N2, a pronounced fronto-central negativity occurring around 200–300 ms after onset of the stop stimulus, which is followed by the P3 a fronto-central to centro-parietal positivity peaking around 300–400 ms after the stop stimulus. The stop-related N2 has been related to conflict monitoring, whereas the stop-related P3 seems to be more directly linked response-related evaluative processing stages, specifically the evaluation of the outcome of the response inhibition (Huster et al. 2013).

Compared to the large number of studies that examined SSRT in OCD, inhibition-related N2 and P3 have rarely been examined in these patients. Moreover, only some studies used the stop-signal task (Fan et al. 2016; Johannes et al. 2001; Lei et al. 2015); whereas, others used the go/nogo task (Herrmann et al. 2003; Keskin-Ergen et al. 2014; Kim et al. 2007). During go/nogo tasks, subjects are required to respond in go trials and withhold a response in nogo trials. That is, the inhibition of a response in the nogo condition occurs before a response is initiated; whereas, inhibition in the stop-signal task requires the inhibition of an already initiated response. The latter is more similar to the psychopathology of patients which shows difficulties in stopping an ongoing response. This methodological heterogeneity may be one reason for inconsistent results observed in ERP studies on response inhibition. For the N2, tic-free OCD patients either showed increased (Johannes et al. 2001; Lei et al. 2015), smaller (Kim et al. 2007) or normal amplitudes (Fan et al. 2016; Herrmann et al. 2003; Keskin-Ergen et al. 2014). Increased amplitude is in line with the hypothesis of general hyperactivity of the conflict monitoring system in OCD (Endrass and Ullsperger 2014). The amplitude of the P3 was normal in most studies (Herrmann et al. 2003; Johannes et al. 2001; Kim et al. 2007; Lei et al. 2015) but alterations are suggested by Fan et al. (2016) who found reduced P3 amplitudes for OCD. Importantly, if the P3 is linked to inhibition and if inhibition is assumed to be dysfunctional in OCD, one would expect altered P3 amplitudes. Regarding the association of N2 and P3 with tics, most studies revealed normal amplitudes (Brandt et al. 2017; Shephard et al. 2016; but Johannes et al. 2001). Note that no study to date compared the electrophysiological indicators related to response inhibition between OCD with or without TD. We expect that in patients with tic-related OCD, given the etiological proximity with the psychopathology of tics, we would see a pattern similar to that in other patients with tics, i.e., normal SSRTs and normal N2 and P3 amplitudes. In contrast, in tic-free OCD, prior evidence and theoretical assumptions lead us to expect increased SSRTs and altered N2 and P3 amplitudes.

Methods

Subjects

Twenty-one tic-free OCD patients, twelve tic-related OCD patients and twenty-one healthy comparison subjects participated in the present study. All participants were examined by trained clinicians using the Structured Clinical Interview for DSM-IV (SCID-I, First et al. 1996), had normal or corrected-to-normal vision, reported no history of head trauma or neurological disease, and were aged between 18 and 65 years. Participants were matched regarding age, gender, handedness, and years of education (Table 1).

All OCD patients had to fulfill DSM-IV criteria for OCD which was established through the SCID-I. The presence (tic-related OCD group) or absence (tic-free OCD group) of lifetime TD was established with the Diagnostic Confidence Interview (Robertson et al. 1999). Patients in the tic-related OCD group also had to fulfill lifetime criteria for persistent motor or vocal tic disorder or Tourette´s disorder. Given the complex genetic relationship between OCD and TD, in case of tic-free OCD patients not only patients but also their first-degree relatives had to be free

Table 1 Demographic and clinical data by group

Measure	Tic-free OCD ($n=21$)		Tic-related OCD ($n=12$)		Healthy controls ($n=21$)		Analysis
	Mean	SD	Mean	SD	Mean	SD	
Age, years	34.0	8.3	33.6	8.8	30.9	7.8	$F(2,53)=0.812$, $p=0.450$
Verbal IQ	28.6	6.6	31.0	5.8	29.8	5.3	$F(2,53)=0.598$, $p=0.554$
BDI-II	16.8	10.3	20.5	8.8	3.8	4.2	$F(2, 53)=20.679$, $p=0.000$
OCI-R	22.2	9.1	29.9	13.0	7.5	9.2	$F(2, 53)=21.399$, $p=0.000$
Y-BOCS	18.6	5.8	21.5	5.5	–	–	$F(1, 32)=2.796$, $p=0.105$
YGTSS	–	–	10.8	5.9	–	–	–

OCD obsessive–compulsive disorder, *SD* standard deviation, *IQ* intelligence quotient, *BDI* Beck Depressions Inventory—Revised, *OCI-R* obsessive–compulsive inventory—revised, *Y-BOCS* Yale-Brown Obsessive–Compulsive Scale, *YGTSS* Yale Global Tic Severity Scale

of tic symptoms. The Yale-Brown Obsessive–Compulsive Scale (Y-BOCS, Goodman et al. 1989) was used to measure severity of OCD symptoms and the Yale Global Tic Severity Scale (Leckman et al. 1989) was used to measure severity of tic symptoms. Comorbidities in the tic-free OCD group were social phobia ($n=1$), specific phobia ($n=1$), depression ($n=4$, one remitted) and panic disorder ($n=2$), depression ($n=4$, one remitted), and specific phobias ($n=2$) in the tic-related OCD group. Of the tic-free OCD patients, five were medicated with selective serotonin reuptake inhibitors (SSRIs), and of the tic-related OCD group five were medicated with SSRISs. Patients were excluded if they fulfilled lifetime criteria for substance dependency, schizophrenic spectrum disorders, or delusional disorders.

Recruitment and instruments used

OCD patients were recruited from the outpatient unit of the Humboldt-Universität zu Berlin and through advertisement of the German Tourette Society and German Society for OCD. Healthy comparison subjects were recruited through local advertisement. They reported no family history of OCD and no past or present signs of psychiatric disease.

All participants completed the Beck Depression Inventory (BDI) II (Steer et al. 1997), the Obsessive–Compulsive Inventory—Revised (Foa et al. 2002), the University of Sao Pauli—Sensory Phenomena Scale (USP-SPS) (Rosario et al. 2009) and a German vocabulary test measuring verbal intelligence (Wechsler 2008). Participants received verbal and written information and informed consent was obtained from all individual participants included in the study. All procedures performed were in accordance with the ethical standards of the local institutional and/or national research committee (local ethics committee) and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Task

The stop-signal task was used to assess response inhibition and performance monitoring. In this task, participants were required to respond rapidly to left- or right-facing arrows presented on a computer screen (for 1000 ms) with corresponding button presses and to attempt to inhibit responses when an auditory "stop signal" sounded (25% of trials, 1000 Hz, 50 ms, and 85 dB). The total number of trials was 480, the time between trials varied between 900 and 1400 ms. A tracking algorithm adjusted stop-signal delay (SSD) for each hand separately in 50-ms increments, increasing after Stops and decreasing after Stop failures to iterate a 50% inhibition rate (Aron et al. 2004). The starting value of the SSD was 250 ms. Subjects performed one practice block with 32 trials. After that subjects continued with the test trials which were run in six blocks of 80 trials each. After each block subjects received performance feedback (e.g., "You made 51% errors.") and the instruction to "continue performing as before" if error rates were between 48 and 52%, to perform "more accurate" if error rates were between 53 and 57%, to "much more accurate" if error rates were larger than 57%, "faster" if error rates were between 43 and 47%, and "much faster" if error rates were smaller than 43%. There was no upper limit for stop-signal delay but the lower limit was 0 ms. Subjects were seated in a dimly lit room. A chin rest was used to minimize head movements. Stimuli were generated by a personal computer using Presentation software 12.2 (Neurobehavioral Systems, Inc., Albany, NY) and were presented on a 19-inch monitor (distance from eyes: 65 cm).

EEG recording and analysis

EEG signals were recorded from 65 electrodes, including Cz as a recording reference. Electrodes were mounted on an electrode cap with equidistant electrode positions (EASY-CAP GmbH, Herrsching, Germany). Additional electrodes

were placed at the following four locations: IO1, IO2, nasion, and neck. The ground electrode was located below T1. All electrode impedances were < 5 kW. EEG activity was recorded with a sampling rate of 500 Hz and filtered with a band pass of 0.01–100 Hz. Off-line, the data were re-referenced to the average of all scalp electrodes corrected for eye-movement artifacts using the Independent Component Analysis implemented in the Brain Vision Analyzer 2 (Brain Products GmbH, Gilching, Germany). The continuous EEG signals were digitally filtered with a low-pass filter of 40 Hz and a notch filter of 50 Hz. Stimulus-locked epochs with a duration of 1200 ms, including a 200-msc preresponse interval, were extracted. Epochs containing voltages exceeding a standard deviation of 100 mV, or voltage steps > 40 mV, between consecutive data points were excluded from further analysis.

For analysis of the behavioral performance, we first calculated the mean successful stop rate. The average SSD was then identified for each participant to determine the point at which the inhibitory process begins according to the horse race model. To determine the point at which the inhibitory process must finish to yield successful stops, reaction times to "go" stimuli were rank-ordered. The reaction time on this distribution that reflected the participant's successful stop rate was selected as the point that separated "go" responses into "fast go" and "slow go" categories. For example, if participant A had a successful stop rate of 60%, then this reaction time would be defined as the point that separates the bottom 60% of this participant's "go" reaction times from the upper 40%. In this way, the calculation process takes into account each participant's individual task performance (i.e., their unique distribution of "go" reaction times). The SSRT was then calculated by subtracting the average individual SSD from that midpoint reaction time, yielding a value that indicates the amount of time that it took for participants to complete the inhibitory process amid an already initiated excitatory "go" process (Logan and Cowan 1984; Verbruggen et al. 2013). In line with the recommendations by Verbruggen et al. (2019), SSRT was not calculated if the probability to respond on a stop trial was lower than 0.25 or higher than 0.75. Trials were excluded if reaction times were smaller than 100 ms or larger than 1000 ms or if a go response was omitted.

For each participant, ERPs were averaged locked to stop-signal onset during successful and unsuccessful stop trials and we then used two procedures to gain stop-related ERPs. We first computed "corrected" ERPs because stop-related ERPs overlap with ERP activity locked to the preceding "go" stimulus, ERPs for these "go" stimuli were also derived. Following the procedure described by Kok et al. (2004) or Palmwood et al. (2017) subtracted activity related to the preceding "go"-stimuli from activity related to the "stop" -stimuli to account for overlapping activity associated with the go-trial. According to the race model, slower reaction times to "go" stimuli are associated with successful stopping, and faster reaction times are associated with unsuccessful stopping. Thus, "go"-locked ERPs were categorized as "fast go" or "slow go" by taking the midpoint reaction time used in obtaining the SSRT. "Go" trials associated with a reaction time that fell below this midpoint are categorized as "fast go" and those that fell above as "slow go." Before subtracting "fast go" ERPs from "unsuccessful stop" ERPs and "slow go" ERPs from "successful stop" ERPs, we controlled for differences in the SSD, i.e., the time of the onset of the stop stimulus. Because the SSD for successful trials is shorter than the SSD for unsuccessful trials, these two categories of stop trial ERPs overlap with different segments of the preceding "go" ERPs. To derive adjusted "successful stop" ERPs, an earlier segment of the "slow go" ERP starting 1 ms (and lasting 1000 ms) before the average SSD for "successful stop" trials was subtracted from averaged "successful stop" trials. Similarly, for calculation of adjusted "unsuccessful stop" ERPs, a later segment of the "fast go" ERPs starting 1 ms (and lasting 1000 ms) before the average SSD for "unsuccessful stop" ERPs was subtracted from averaged "unsuccessful stop" trials. In summary, subtraction led to two stop ERPs that were corrected for differences in overlap to the proceeding "go" stimuli: adjusted "successful stop" trial (SST; i.e., averaged "successful stop" ERP minus averaged "slow go" ERPs) and "unsuccessful stop" trial (USST; i.e., averaged "unsuccessful stop" ERPs minus averaged "fast go"). All ERPs were baseline corrected prior to the subtraction process using activity in the preceding 200 ms (de Jong et al. 1990; Kok et al. 2004; Palmwood et al. 2017 for additional detail regarding these calculations). N2 and P3 amplitude and latency were quantified as peak amplitudes by computing the local (negative or positive) maximum within the time windows from 130 to 250 for the N2 and 250 to 410 ms for the P3. In line with the literature, peak measures were derived from FCz and Cz electrodes for N2 and P3, respectively (e.g., Kok et al. 2004). We also computed N2 and P3 amplitudes using the same spatial and temporal parameters without the correction method that was described above. These are termed "uncorrected" data in the results section.

Statistical analysis

One-way analyses of variance (ANOVAs) were used to examine symptom severity scores and behavioral parameters. Repeated-measures ANOVAs were used for statistical analyses of ERP measures using group (tic-free OCD patients, tic-related OCD patients, and healthy comparison subjects) as a between-subject factor and inhibition (StC = stop correct, StF = stop fail) as within-subject factors. All statistical tests were two-tailed. Post hoc comparisons

were corrected using the Bonferroni procedure, and only corrected p values are reported. Reported effect sizes are partial eta squared (η^2) for ANOVAs and Cohen's d for pairwise comparisons between groups. Power ($1-\beta$) was calculated for group comparisons. Correlation coefficients across groups (Pearson's r) were used to examine associations between symptom severity and outcome measures. To evaluate medication effects, the behavioral parameters were also analyzed comparing only unmedicated tic-free and tic-related OCD patients that were matched according to age, gender, and education. For analyses of the ERP data, medication was used as a covariate in the repeated-measures ANOVA, instead, as this has greater power. Statistical analyses were conducted using IBM SPSS Statistics, Version 24.0 (IBM, Armonk, United States of America).

Results

Sociodemographic and clinical data

Table 1 presents demographic and clinical data as well as information for statistical analysis. Groups did not differ with regard to age, verbal intelligence and gender. The percentage of female participants was 41.7% in the tic-related OCD group, and 45.5% in the tic-free OCD and normal group (Chi-squared = 0.055, $p = 0.973$). Groups differed in clinical measures: as expected, healthy controls had smaller BDI and OCI-R scores compared to both groups of OCD patients (tic-related OCD $p < 0.01$, tic-free OCD $p < 0.01$). There was a trend for larger OCI-R in the tic-related OCD group ($p = 0.053$). Patients with tic-related OCD had higher scores in two subscales of the USP-SPS measuring sensory phenomena compared to tic-free OCD, namely in the touching-[mean = 1.9, standard deviation (SD) = 1,16 vs. mean = 0.7, SD = 0.9, $p < 0.01$] and auditory-related just-right experiences (mean = 1.1, SD = 0.8 vs. mean 0.3, SD = 0.8, $p < 0.01$) domains. No other group differences were significant.

The seven unmedicated tic-related OCD patients were compared to seven tic-free OCD patients that were matched according to age, gender, and education. The mean age was 32.71 (7.63) compared to 35.29 (5.47) [$t(12) = 2.57$, $p = 0.483$], and the mean verbal IQ was 33.00 (6.86) compared to 30.28 (6.10) [$t(12) = 2.71$, $p = 0.449$], for the tic-related OCD and tic-free OCD group, respectively. The gender ration (female:male) was 3:4 and 2:5. The median SSRT for the tic-related OCD group was 205 (75) vs. 207 (44), $t(12) = 1.29$, $p = 0.970$.

Behavioral data

Groups did not differ in any of the behavioral parameters (Table 2).

ERP data

Corrected data

The amplitude of the P3 showed a main effect of inhibition, $F(1, 51) = 15.088$, $p < 0.01$, $\eta^2 = 0.228$, due to larger amplitudes for correct stop trials compared to failed stop trials. The main effect for group was also significant, $F(2, 51) = 6.512$, $p < 0.01$, $\eta^2 = 0.203$, power = 0.966. Post hoc analyses showed that tic-free OCD patients had, independent of condition, larger amplitudes compared to tic-related OCD $F(1, 31) = 4.879$, $p > 0.05$, Cohen's $d = 0.825$, power = 0.707), and healthy controls, $F(1, 31) = 11.057$, $p > 0.01$, Cohen's $d = 1.052$, power = 0.967 (Figs. 1 and 2). No difference was found between tic-related OCD and healthy controls [$F(1,52) = 0.235$, $p = 0.631$], Cohen's $d = 0.181$, power = 0.089. The interaction group × inhibition was not significant ($F(2,52) = 0.388$, $p = 0.680$), $\eta^2 = 0.015$,

Table 2 Median and standard deviations for behavioral stop data by group

	Tic-free OCD		Tic-related OCD		Healthy controls		Analysis
	Median	SD	Median	SD	Median	SD	
Go RT	632	151	532	135	610	151	$F(2,53) = 1.789$, $p = 0.117$, $\eta^2 = 0.066$
SSRT	162	76	201	58	182	39	$F(2, 53) = 1.629$, $p = 0.206$, $\eta^2 = 0.600$
SSD	470	152	356	138	387	136	$F(2,53) = 2.947$, $p = 0.061$, $\eta^2 = 0.104$
Failed stop RT	574	150	479	139	518	124	$F(2,53) = 2.001$, $p = 0.145$, $\eta^2 = 0.071$
% No response	0.6	1.0	1.0	1.38	1.7	3.3	$F(2,53) = 1.205$, $p = 0.308$, $\eta^2 = 0.009$
% Choice errors	0.5	0.9	0.9	0.9	0.6	0.8	$F(2,53) = 1.204$, $p = 0.308$, $\eta^2 = 0.045$
Probability of responding on stop trials	51.3	2.4	50.8	2.9	48.9	1.6	$F(2,53) = 2.326$, $p = 0.108$ $\eta^2 = 0.084$

OCD obsessive–compulsive disorder, *SD* standard deviation, *RT* reaction time, *SSD* stop-signal delay, *SSRT* stop-signal reaction time

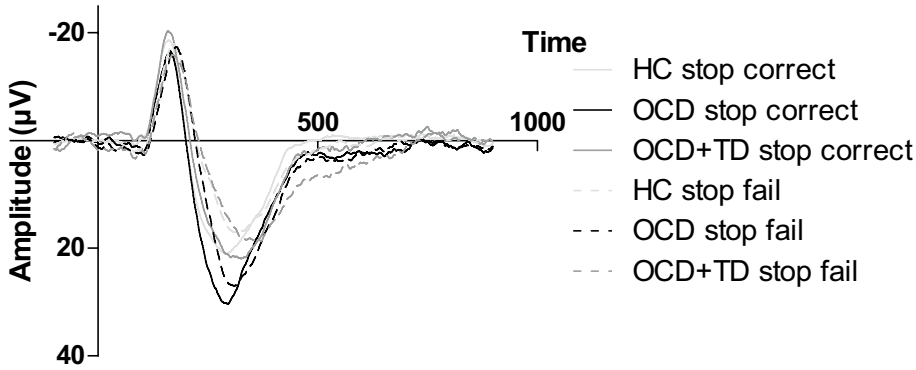

Fig. 1 Grand average waveforms at electrode Cz for correct and failed stop trials for patients with tic-free obsessive–compulsive disorder (OCD), healthy controls (HC), and tic-related OCD (OCD + TD)

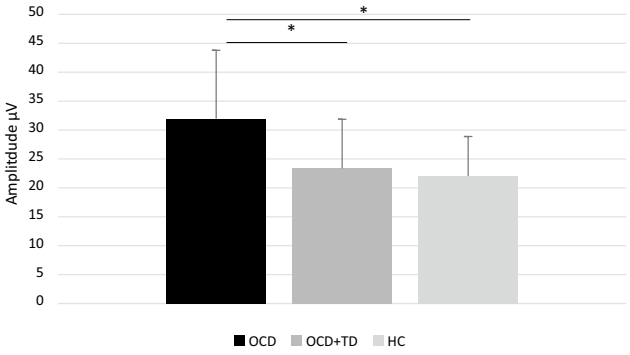

Fig. 2 Mean amplitude values of P3 at FCz electrode for tic-free obsessive–compulsive disorder (OCD) patients, for tic-related OCD patients, and healthy controls (HC). Differences marked by asterisk are significant at $p < 0.05$*

power = 0.329. Regarding P3 latencies, there was also a main effect of inhibition, $F(1, 51) = 13.894$, $p < 0.01$, $\eta^2 = 0.214$, due to shorter latencies for correct stop trials compared to failed stop trials. Although latencies were numerically longer for tic-related OCD patients (see Table 3), the main effect of group was not significant, $F(2, 51) = 1.450$, $p = 0.244$, $\eta^2 = 0.054$, power 0.377 neither was the interaction $F(2, 51) = 0.415$, $p = 0.663$, $\eta^2 = 0.016$, power = 0.353.

When medication was used as a covariate, no significant interaction with inhibition occurred and though the F value for the group effect was reduced, the main effect of group was still significant for P3 amplitudes, $F(1, 30) = 4.267$, $p < 0.05$, $\eta^2 = 0.125$, power = 0.800.

For the N2, no main or interaction effects were found [main effect inhibition: $F(1,51) = 0.048$, $p = 0.828$, $\eta^2 = 0.001$,; main effect group: $F(2, 51) = 0.062$, $p = 0.940$, $\eta^2 = 0.002$, power = 0.058; group × inhibition interaction: $F(2, 51) = 0.917$, $p = 0.460$, $\eta^2 = 0.035$, power = 0.678]. No significant effects were found when medication was entered as a covariate (all $p > 0.265$).

Since there were significant group differences in BDI and OCI-R scores, we re-ran the analysis of ERP measures and included these scores as a covariate. This analysis affected results only marginally and the P3 group effect remained significant for BDI [$F(2, 49) = 6.991$, $p = 0.002$, $\eta^2 = 0.222$] and OCI-R [$F(2,50) = 6.825$, $p = 0.002$, $\eta^2 = 0.214$].

Uncorrected data

When the P3 was calculated without correcting for activity of the go stimulus, amplitudes showed a main effect of inhibition that was largely reduced in size, $F(1, 51) = 6.681$, $p < 0.05$, $\eta^2 = 0.116$, due to larger amplitudes for correct stop trials compared to failed stop trials (see Table 4). Similarly, the group effect was significant $F(2, 51) = 4.646$, $p < 0.05$, $\eta^2 = 0.154$, power = 0.249, but smaller in size. Regarding P3 latencies, there was also a reduced main effect of inhibition, $F(1, 51) = 9.930$, $p < 0.01$, $\eta^2 = 0.163$, due to shorter latencies for correct stop trials compared to failed stop trials.

Table 3 Median and standard deviations for P3 latencies by group

	Tic-free OCD		Tic-related OCD		Healthy controls		Analysis
	Mean	SD	Mean	SD	Mean	SD	
P3 StC latency	304	35	314	32	302	337	$F(2,53) = 0.459$, $p = 0.636$, $\eta^2 = 0.018$
P3 StF latency	318	31	341	42	322	33	$F(2,53) = 1.861$, $p = 0.166$, $\eta^2 = 0.068$

OCD obsessive–compulsive disorder, *SD* standard deviation, *RT* reaction time, *SSD* stop-signal delay, *SSRT* stop-signal reaction time

Table 4 Uncorrected P3 amplitudes and latencies by group

	Tic-free OCD		Tic-Related OCD		Healthy controls		Analysis
	Mean	SD	Mean	SD	Mean	SD	
P3 StC amplitudes	28.9	11.3	22.2	9.2	22	6.7	$F(2,53)=3.359$, $p=0.043$, $\eta^2=0.116$
P3 StF amplitudes	28.1	10.9	19.8	8.5	19.4	8.4	$F(2,53)=1.498$, $p=0.233$, $\eta^2=0.170$
P3 StC latencies	302	32	326	29.0	306	40	$F(2,53)=3.359$, $p=0.043$, $\eta^2=0.068$
P3 StF latencies	322	31	340	48	321	24	$F(2,53)=5.241$, $p=0.009$, $\eta^2=0.055$

OCD obsessive–compulsive disorder, *SD* standard deviation, *RT* reaction time, *SSD* stop-signal delay, *SSRT* stop-signal reaction time

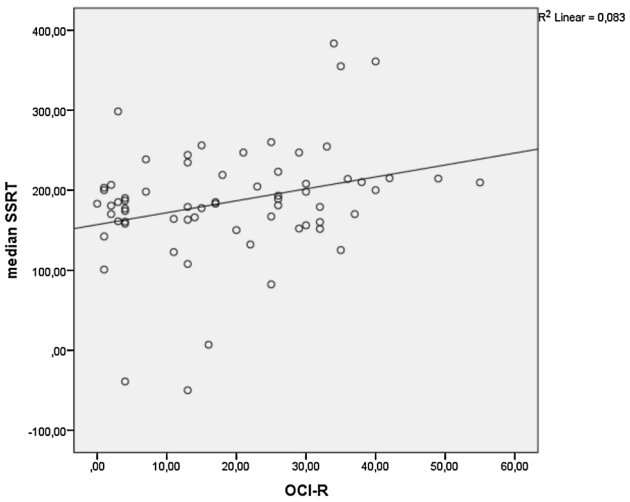

Fig. 3 Scatterplot illustrating the relationship between median stop-signal reaction time (SSRT) and scores in the Obsessive–Compulsive Inventory—Revised (OCI-R)

Correlational analysis

The only significant correlation was a positive correlation between OCI-R scores and SSRT ($r=0.288$, $p<0.05$), see Fig. 3.

Discussion

In the present study, behavioral and neural correlates of response inhibition and inhibition related processes were compared between tic-free OCD, tic-related OCD, and normal controls. The SSRT was normal in both OCD groups. In the ERPs, however, differences were found since tic-free OCD patients had significantly increased P3 amplitudes compared to tic-related OCD patients and normal controls, which did not differ from each other. The N2 did not differ between groups.

We expected to find normal SSRT in tic-related OCD and slower SSRT in tic-free OCD but only found partial support for this. The normal SSRT we observed in tic-related OCD match the normal SSRTs in TD patients (Fan et al. 2018; Johannes et al. 2001; Lei et al. 2015) and the intact response inhibition in pediatric patients with comorbid OCD and TD (Mancini et al. 2018). Intact behavioral response inhibition in association with tics is further corroborated by a study that showed intact performance in a go/nogo task for TS patients comorbid with OCD compared to normal controls (Muller et al. 2003). In TD, intact functioning in tasks requiring response inhibition is often interpreted in two ways. One is the assumption of compensatory neural activity which then leads to intact behavioral performance. This compensatory activity, however, is only observed during the execution of a movement (Ganos et al. 2014; Thomalla et al. 2014), whereas activity during stopping is mostly normal compared to healthy controls (Fan et al. 2018). Importantly, in our tic-related OCD sample, intact response inhibition was not accompanied by dysfunctional ERPs which renders this explanation less convincing. Another interpretation of intact behavioral response inhibition is that tics are a voluntary response to reduce discomfort elicited by sensory phenomena (e.g., not-just-right experience) rather than a failure of response inhibition (Li et al. 2006). Patients with tic-related OCD report more of these sensory phenomena compared to tic-free OCD (de Alvarenga et al. 2012; Kloft et al. 2018; Miguel et al. 1997) which was also replicated in the present study.

In contrast to our expectation, we found normal SSRT in tic-free OCD. A large number of studies exist that found slower SSRT for these patients (e.g., Chamberlain et al. 2006; Fan et al. 2016; Lei et al. 2015; Lipszyc and Schachar 2010; McLaughlin et al. 2016; Menzies et al. 2007). However, there are also several studies that report normal SSRTs in OCD (Dayan et al. 2017; Johannes et al. 2001; Kalanthroff et al. 2017; Kim et al. 2017; Martoni et al. 2018). Inconsistency between studies may result from differences in methods although an examination of the so far conducted studies does not suggest influence of factors like modality of the stop signal, proportion of go vs. stop stimuli, or number of trials. Differences between studies may also result from sample differences although SSRT seems to be independent of medication (Kalanthroff et al. 2017), age of onset

(Lei et al. 2017) and symptom dimension (Lei et al. 2015). Kalanthroff et al. argued that the failure to find behavioral dysfunction of response inhibition in some studies and the generally small effect size indicate that not all OCD patients show this deficit. They assume a "second-hit" model with a so far undetermined factor mediating behavioral response inhibition. Further studies are needed to elucidate this.

In the absence of behavioral differences, we observed differences in the P3. We found normal amplitudes of the P3 in tic-related OCD and increased amplitudes of the P3 in tic-free OCD. The P3 has been considered as a neural correlate of behavioral response inhibition through studies that found larger P3 amplitudes under increased inhibitory demand (e.g., Smith et al. 2007) and for correct vs failed stop trials (for a review Huster et al. 2013). Larger amplitudes in correct trials were also replicated in the present study. Yet, it is often argued that the P3 occurs too late to reflect the actual inhibition process. In the present study, the SSRT was 179 ms; whereas, the latency of the P3 in correct stop trials was 306 which further supports this idea. It is rather assumed that the P3 reflects an aftereffect of inhibition that could be the evaluation of the outcome of the inhibition (Bruin et al. 2001; Huster et al. 2013). Normal P3 amplitudes in our sample of tic-related OCD, thus, further corroborate intact response inhibition by showing that the neural processes related to behavioral response inhibition (i.e., evaluation of the response outcome) are intact. As in behavioral studies, this finding is similar to what is found in TD patients, e.g., normal P3 amplitudes were found in a stimulus–response compatibility task (Thibault et al. 2008). Importantly, this was independent of co-morbid OCD further indicating similarity between TD and tic-related OCD. In TD, normal P3 amplitudes were also found in other tasks measuring performance monitoring (e.g., Brandt et al. 2017; Eichele et al. 2016; Shephard et al. 2016). Hence, performance of patients with tic-related OCD mirrors performance of patients with TD not only on a behavioral but also on a neural level.

Regarding tic-free OCD, the increased P3 amplitude supports dysfunctional evaluation of the inhibition. In accordance with this, increased P3 amplitudes in tic-free OCD were also found in tasks measuring attentional inhibition (Fan et al. 2014; Gohle et al. 2008; Mavrogiorgou et al. 2002). Further support for altered evaluation of the inhibition stems from the overlap in structures associated with inhibition-related P3 and alterations found in OCD. These include the middle frontal gyrus including the inferior frontal cortex, inferior parietal cortex, preSMA, temporal regions, and the basal ganglia (de Wit et al. 2012; Huster et al. 2013; Rubia et al. 2010; Woolley et al. 2008). However, in a study that directly compared neural activity in the SSRT between patients with TD and OCD, no inhibition-related differences between these groups were found (Fan et al. 2018). As a limitation to this result, the study failed to replicate its own prior finding of hyperactive activity of the preSMA and hypoactivity of the inferior parietal cortex. The authors assume that this is at least in part due to learning effect as the same patients were included (de Wit et al. 2012). Hence, further examination is warranted.

Increased P3 amplitudes indicating hyperactivity of the evaluation of the outcome of the inhibition are in line with the broader assumption during performance monitoring as a core deficit in OCD (Endrass and Ullsperger 2014), and potential biomarker (Riesel et al. 2011; 2015). So far, dysfunctional performance monitoring in OCD has mainly be examined in response-related potentials like the error-related negativity (ERN) and correct-related negativity (CRN). The ERN has consistently been found to be increased in OCD and some other studies also found an enhancement of the CRN (see Endrass and Ullsperger 2014). Our data together with results from Hanna et al. (2012) who reported increased ERN amplitudes for pediatric tic-free OCD patients and normal ERN amplitudes for tic-related OCD patients indicate that this process is only dysfunctional in patients with tic-free OCD but is intact in tic-related OCD. The present data also suggest that hyperactive performance monitoring may also occur in situations that require response inhibition and that during these situations overactive evaluation of the outcome seems to occur in tic-free OCD. This can be translated into the compulsions of patients, as from a patient's perspective, the inhibition of a response requires a particularly heightened evaluation of the outcome as the stopping of a compulsion is associated with a feared catastrophe.

Together, these further support the idea that different psychobiological mechanisms underlie the compulsions in tic-free and tic-related OCD. In tic-free OCD, our P3 data indicate that the compulsions are related to dysfunctional outcome evaluation in relation to response inhibition. In tic-related OCD, the higher number of sensory phenomena and normal P3 amplitudes—a pattern that is similar to TD—indicate that their compulsions are more tic-like (Holzer et al. 1994).

A potential limitation of the interpretation of the P3 data is that other studies did not find increased amplitudes. One reason to explain this difference lies in the procedure to calculate the P3. In the SSRT, the go stimulus and the stop stimulus follow in close temporal proximity. That is, activity of processing the go stimuli may overlap with the activity of interest, i.e., processing of the stop signal. To create a (potentially) less confounded measure of correct and failed stop activity, it is a prerequisite to isolate these from the processing of the go stimulus. Different procedures including the one used here have been successfully used for this (e.g., Bekker et al. 2005; Palmwood et al. 2017; Wessel and Aron 2015) and doing so appears state-of-the-art. However, none of the studies that examined the ERPs in the

stop-signal task in OCD did use any isolation procedure. When we re-analyzed our data by using the unadjusted P3 amplitudes, the effect size was reduced from 1.03 (Cohen's *d*) to 0.85. Hence, it cannot be excluded that the differences between the present and prior studies are due to differences in analytical methods and the processes that are captured by these. Importantly, in our study significantly increased P3 amplitudes were found with both methods.

The N2 did not show a condition effect which appears at odds with its role in conflict monitoring or altered conflict monitoring in OCD, respectively. In general, results regarding the N2 in response inhibition paradigms appear less consistent than, e.g., for the P3. While increased amplitudes for failed compared to stop trials are often found and thus support a role in conflict monitoring (e.g., Galdo-Alvarez et al. 2016; Palmwood et al. 2017; Ramautar et al. 2004), other studies failed to find this condition effect (Gonzalez-Villar et al. 2016; Johnstone et al. 2007). Moreover, in some studies, the N2 was only elicited for failed trials (Greenhouse and Wessel 2013; Kok et al. 2004); while in others; the N1 was elicited instead of the N2 (Bekker et al. 2005; Dimoska and Johnstone 2007). The N2 and its underlying process, thus, appear susceptible to methodological and/or sample effects. In addition, Larson et al. (Larson et al. 2014) who reviewed the role of the N2 (and the ERN) in conflict tasks concluded that depending on the paradigm that is used, the N2 may reflect different processes such as response inhibition, mismatch, conflict monitoring or others, and that not all N2s necessarily index conflict monitoring. In accordance with this, the N2 effects in OCD vary across studies such that enhanced (e.g., Riesel et al. 2017; Ruchsow et al. 2007), reduced (Kim et al. 2007; Morault et al. 1997) or normal (Herrmann et al. 2003; Keskin-Ergen et al. 2014) N2 amplitudes have been found. It is interestingly, though, that the process that is reflected in the N2 appears similar between tic-free and tic-related OCD. Hence, while the two OCD groups appear to be affected differentially in terms of the response inhibition process reflected in the P3, they respond equally in terms of the N2.

The present study is not without limitation. We only included a small number of patients with tic-related OCD. It was extremely difficult to find OCD patients with comorbid TD despite the fact that recruitment was done in a large outpatient unit. To overcome the problem of small samples, it might be necessary to conduct multicenter studies. However, to our knowledge, this is the first study that compared a priori and carefully matched patients with tic-free and tic-related OCD. An important avenue for future research would be the direct examination of TD patients without comorbid OCD. Showing that tic-related OCD is more similar to TD than OCD through a direct examination would support the hypothesis of tic-related OCD being a variant expression of TD vulnerability. In addition, the present study included a mixture of medicated and non-medicated patients. The effect of SSRIs on response inhibition is controversial as studies showed that desipramine can improve the ability to inhibit responses (Overtoom et al. 2003; Skandali et al. 2018) whereas other studies did not find altered stopping performance in relation to SSRIs such (Drueke et al. 2010; Nandam et al. 2011). Therefore, it cannot be excluded that medication had an impact on response inhibition. Importantly, however, the direction and significance of results was unchanged when we controlled for the effect of medication. Future studies should try to replicate our findings in a sample of only unmedicated patients.

In our study design, feedback was only given on stop performance but not go performance. Verbruggen et al. (2013) showed that focusing on the stop performance can lead to severely skewed RT distributions and invalid SSRT computations even when the integration method is used, as it was done in the present study. The authors suggest to give clear advance instructions (e.g., by stressing speed in the go task and explaining the staircase-tracking procedure) and by providing feedback after every trial. Future studies could investigate whether this affects study results with OCD samples.

Taken together, SSRT data indicate intact response inhibition and P3 data indicate intact outcome evaluation in relation to response inhibition in tic-related OCD. This finding resembles results in TD and may be interpreted as a support for the idea that tic-related OCD may be etiologically related to TD and differs from OCD. In OCD, alterations in P3 indicate hyperactivity in the evaluation of the outcome of the inhibition process. This is in line with hyperactivity generally found in performance monitoring in OCD.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest

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