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Self-reported and neurocognitive impulsivity in obsessive-compulsive disorder

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ABSTRACT

Background: Although a behavioural addiction model of obsessive-compulsive disorder (OCD) has been proposed, it is still unclear if and how self-report and neurocognitive measures of impulsivity (such as risk-taking-, reflection- and motor-impulsivities) are impaired and/or inter-related in this particular clinical population.
Methods: Seventeen OCD patients and 17 age-, gender-, education- and IQ-matched controls completed the Barratt Impulsivity Scale, the Obsessive-Compulsive Inventory-Revised, and the Beck Depression Inventory and were evaluated with the Yale-Brown Obsessive-Compulsive Scale and three computerized paradigms including reward (the Cambridge Gambling Task), reflection (the Information Sampling Task) and motor impulsivity (Stop Signal Task).
Results: Despite not differing from healthy controls in any neurocognitive impulsivity domain, OCD patients demonstrated increased impulsivity in a self-report measure (particularly attentional impulsivity). Further, attentional impulsivity was predicted by severity of obsessive-compulsive symptoms.
Conclusions: Our findings suggest that OCD is characterized by a subjective (rather than objective) impulsivity; in addition, self-reported impulsivity was largely determined by severity of OCD symptoms.

1. Introduction

Compulsivity has been defined as the "tendency to perform repetitive acts in a habitual/stereotyped manner to attempt to prevent adverse consequences" [1], whereas impulsivity is considered "a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individuals or to others" [2]. Compulsivity and impulsivity are traditionally considered opposite ends of a risk aversive vs. seeking spectrum [3]. However, this model has been criticized by many researchers, who argue that compulsivity and impulsivity actually seem to be orthogonally [4] or positively [5] related. Accordingly, many clinicians have noted they may actually coexist in increased levels in individual patients with obsessive-compulsive and related disorders, substance and behavioural addictions, or disruptive, impulse-control and conduct disorders [6].

Evidence of impulsive traits in obsessive-compulsive disorder (OCD) and of compulsive features in addictions and impulse control disorders has contributed to the hypothesis that impulsivity and compulsivity may share common neurobiological mechanisms. For instance, neurocognitive [7], imaging [8], and neurosurgical [9] studies suggest that OCD participants exhibit dysfunctional reward processes thought to be important to impulse control and addictive disorders [10]. Consequently, a behavioural addiction model for OCD has been recently proposed [11]. There is evidence suggesting that impulsivity in OCD tend to become more prominent with the progression and severity of the disorder [12]. According to this model, long-term OCD participants could display decreased resistance, control and insight in relation to their compulsive (particularly motor) behaviours, thus characterizing what some have termed 'impulsive compulsions' for representing compulsive behaviours that are performed in an automatic/unplanned fashion [6,12–15].

Although several studies have found increased neurocognitive [16–18] and self-reported impulsivity [16,18–21] in OCD patients, there is still debate on which types and/or domains of impulsivity are predominantly affected among these individuals. For instance, some studies were unable to find differences in terms of motor and/or risk taking impulsivity

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between OCD patients and controls [22,23]. Perhaps because of different methodologies, impairments in OCD samples have been reported in some [24,25] but not all meta-analysis [26,27]. Similarly, reflection impulsivity (tendency to gather and evaluate information before making a decision) [28] has been only sparsely assessed in adult [29] and juvenile [30] OCD participants, with inconsistent findings. For instance, although reflection impulsivity levels did not differ between adult OCD patients and controls [29], juvenile OCD samples have shown an increased decision threshold (the opposite of an impulsive decision) in a different version of the task using computational modeling [30].

Despite all the empirical evidence suggesting that compulsivity and impulsivity can share some characteristics, it is important to study how they relate both at the self-reported and at the neurocognitive levels, which may also not necessarily correlate with each other [31]. For instance, research has shown increased rates of impulse control disorders [including motorically-focused ("low order") disorders such as trichotillomania (hair pulling disorder) and excoriation (skin-picking) disorders] [32–35] and medium effect sizes for response inhibition deficits according to Stroop Interference in OCD [26]; but no evidence of greater commission errors on the go non-go task in OCD patients as compared to healthy controls [36]. Further, some have argued that OCD is in fact associated with reduced behavioural impulsivity, and that the BIS attentional impulsivity (lack of sense of control of thoughts) causes an artificial inflation of the BIS total score [37].

In this study, our aim was to comprehensively access all the components of self-reported and neurocognitive impulsivity in OCD participants and matched controls and to investigate whether they were differentially impaired. Although lack of correlations between self-report scales and behavioural tasks of impulsivity have been reported in different contexts, some authors suggest that these correlations would be greater if neurocognitive tasks measured more general impulsive tendencies (rather than single cognitive abilities) or if self-report questionnaires assessed specific processes identified by the neurocognitive tasks. [38] Thus, we attempted to address this issue by employing a broad neurocognitive battery assessing different aspects of impulsivity. Based on the behavioural addiction model of OCD and meta-analytic findings, we predicted that both self-reported and neurocognitive impulsivity levels would be significantly elevated in OCD.

2. Material and methods

2.1. Participants

Seventeen OCD patients and 17 age-, gender-, education-, and IQ-matched controls were included in our study. Patients were selected from individuals being treated in the OCD and Anxiety Disorders clinic of the Institute of Psychiatry of the Federal University of Rio de Janeiro (IPUB/UFRJ) and a few local private clinics. Controls have been recruited from D'Or Institute for Research and Education (IDOR) and IPUB/UFRJ administrative and support staff (e.g. cleaners, doorkeepers, and handypersons). The Ethics Committee of the Federal University of Rio de Janeiro approved this research protocol (CAAE # 05089412.2.1001.5263), which complied with the Declaration of Helsinki. Accordingly, written informed consent was obtained from all participants after the procedures involved were fully explained.

All research participants were submitted to an initial general assessment performed by a trained psychiatrist (IF), which included the Structured Interview for Disorders of Axis I (SCID) [39]; the Global Assessment of Functioning Scale (GAF) [40]; and the Yale-Brown Obsessive-Compulsive Symptom Scale (YBOCS) [41,42]. The participants also answered the Beck Depression Inventory (BDI) [43] and the Obsessive-Compulsive Inventory – Revised (OCI-R) [44,45]. Patients' OCD symptoms in descending order of severity were obsessions, neutralization, ordering, washing, checking, and hoarding.

The inclusion criteria comprised being aged between 18 and 65 years, having at least high school education, and, for OCD patients, scoring at least 16 on the YBOCS. Controls had a minimum score of 60 on the GAF, indicating satisfactory everyday functioning. Exclusion criteria for both groups comprised severe mental illness (e.g. psychosis), conditions associated with impulsivity/suicidality (such as comorbid borderline or antisocial personality disorders), alcohol or other substances use disorders, and the use of antipsychotics or benzodiazepines (due to their effects on reaction times). Almost all OCD patients were under a serotonin reuptake inhibitor (SRI), with the exception of one patient taking a serotonin norepinephrine reuptake inhibitor (venlafaxine). One control was on a SRI due to major depression that has been fully remitted for more than a year.

Generally, neuropsychological assessments took place when OCD patients were already prescribed a SRIs and less anxious. However, they were in different stages of follow-up. For instance, many patients who were recruited and tested within the first weeks of treatment were still symptomatic and under lower doses of SRIs. Other subjects were under chronic SRIs prescribed by their previous clinicians when they reached our clinic. They were not necessarily resistant, but required some sort of medication adjustment. Finally, additional patients, who were in later stages of treatment and under higher doses of SRIs displayed different symptom severities and degrees of therapeutic resistance. That said, our sample can be described as heterogeneous in terms of duration of follow up, SRIs doses and treatment response, and cannot be characterized as resistant to treatment. For a summary on SRIs and doses prescribed, see Table 1.

To quantify the effects of SRIs on OCD patients' cognitive performance, scores were attributed to equivalent SRI doses (i.e., zero to no medication; one to 20 mg of fluoxetine, paroxetine or citalopram, 50 mg of sertraline, 100 mg of fluvoxamine, or 75 mg of clomipramine; two to the double of these doses; three to the triple, and so on) based on the minimum dose required to occupy at least 80% of the brain serotonin transporters in the striatum [46]. A similar rationale was used to rate escitalopram (1 to 10 mg), and venlafaxine (1 to 75 mg) equivalent doses. For a description of this SRI dose quantification strategy, see a previous paper by our group [47]. Although there are different approaches to calculate equivalent doses of antidepressants (including SRIs) [48], we felt the present one to be the closest to the clinical reality in OCD patients, which generally require higher doses of SRIs than major depression patients to be optimally treated.

2.2. Instruments

2.2.1. Self-reported impulsivity

2.2.1.1. Barratt Impulsivity Scale. The BIS-11 is the most often used self-report instrument to access personality/behavioural construct of

Table 1
Antidepressants prescribed for the present sample and their corresponding dose.

Patient	Drug	Dose
1	Fluoxetine	60 mg
2	Fluoxetine	40 mg
3	Fluoxetine	40 mg
4	Fluoxetine	20 mg
5	Fluoxetine	20 mg
6	Sertraline	200 mg
7	Sertraline	200 mg
8	Sertraline	150 mg
9	Sertraline	100 mg
10	Sertraline	50 mg
11	Paroxetine	60 mg
12	Paroxetine	50 mg
13	Escitalopram	15 mg
14	Escitalopram	10 mg
15	Clomipramine	225 mg
16	Clomipramine	75 mg
17	Venlafaxine	375 mg

impulsiveness worldwide. It comprises 30 items scored on a Likert scale (ranging from never = 1 point to very frequently = 4 points). The BIS-11 measures impulsivity on its attentional (e.g. "I don't "pay attention"), motor (e.g. "I do things without thinking"), and non-planning (e.g. "I am more interested in the present than the future") aspects [49]. In this study, we used the validated Brazilian Portuguese version of the BIS-11 [50].

2.2.2. Neurocognitive impulsivity

We employed three different tasks from the *Cambridge Neuropsychological Test Automated Battery* (CANTAB) in order to access risk taking, motor and reflection impulsivity [51].

2.2.2.1. Cambridge gambling task. The Cambridge Gambling Task (CGT) was developed to access decision-making and risk-taking behaviour under uncertainty [51]. In this task, the examinee attempts to accumulate as many points as possible. For each trial, a computer screen exhibits a row with a variable number of ten red and blue boxes (10 in total). The participant must choose the box colour (red or blue) they feel contains a yellow token underneath. After making this decision, they then gamble a proportion of points on whether or not they have made the correct colour choice. By sampling colour and bet choices across a range of box ratios, the task decomposes different aspects of decision-making. The outcome measures of interest were: overall proportion bet (i.e. mean percentage of points gambled), quality of decision-making (the proportion of trials when the logical colour choice was made), and risk adjustment (i.e. the extent to which the bet amount varied with the likelihood of winning) [52].

After each trial, the subject receives a feedback if he won or lost. The CGT differs from other gambling tasks by distinguish risk-taking from impulsivity as the participant who wants to make a risky bet must wait patiently for it to appear. We have compared three key outputs of the CGT between OCD and controls, namely: overall proportion bet (i.e. mean percentage of points gambled), quality of decision making [i.e. the fraction of time that the participant chose the most likely outcome (e.g. betting on red when seven red squares and three blue squares are displayed on the screen)], and risk adjustment (i.e. the extent to which the bet amount varies with the likelihood of winning) [52].

2.2.2.2. Stop signal task. The SST was employed to assess motor impulsivity [51]. Performance in the Stop Signal Task (SST) is modelled as a horse race between a "go process", triggered by the presentation of the "go" stimulus (e.g. an arrow pointing to the left or right), and a "stop process", triggered by the presentation of the stop signal (in our case, an auditory tone) [53]. When the stop process finishes before the go process, the response is inhibited; when the go processes finishes before the stop process, the response is emitted [53].

The SST is divided in two parts. In the first one, involving 16 practice trials, there is an arrow pointing either to the left or to the right and the subject must press a correspondent button according to the direction of the arrow. In the second part, comprising of five blocks of 64 trials with 16 stop trials per block, the subject must refrain from pressing any button if he or she hears an auditory stimulus after the visual one. [51] The outcome measure from the task was the stop-signal reaction time (SSRT), which is an estimate of the time taken by an individual's brain to suppress a response that would normally be made [53].

2.2.2.3. Information sampling task. The IST was employed to assess reflection impulsivity. It comprises the exhibition of a 5 × 5 matrix of grey boxes hiding a random distribution of blue or yellow squares; these two colours are also displayed at the bottom of a computer screen [51]. The participants must touch a grey box, which then revels its hidden colour. Participants should choose the colour that predominates on that specific trial at the bottom of the computer screen [54]. To this end, the participant is allowed to touch and reveal as many boxes as he or she wants to make his or her decision [54]. The boxes that were opened by the participants remained visible during the whole duration of the trial to minimize the demands on working memory.

The IST comprises two conditions, one fixed and one decreasing win (FW and DW, respectively). While in the FW the participant is awarded 100 points for a correct colour decision regardless of the number of boxes opened, the number of available points decreased by 10 with every box opened in the DW condition. Thus, in the DW, there is a points' cost for higher levels of information sampling [54]. The outcome measures of interest were the mean number of boxes opened for each of the two task conditions (FW and DW) [55].

2.3. Statistical analysis

All analyses were conducted with the Statistical Package for Social Sciences (SPSS) version 21 for Mac (Chicago, SPSS Inc.). Groups (OCD patients and controls) had their sociodemographic and clinical features compared by means of Student's t or Mann-Whitney tests (according to the normality of distribution) and Chi-square tests. As the IST had two conditions a general linear model repeated measures with condition (fixed vs. decreasing) as within-subjects factor and diagnostic status (OCD vs. controls) as between-subjects factor was performed using the IST outcome. Performances on the SST and CGT were also compared with of Student's t or Mann-Whitney tests. The adopted level of significance was 0.05 uncorrected. The study had an 80% power to detect an effect size of Cohen's d = 1.0 or higher at $p < .05$ uncorrected given the present sample size ($n = 17$ in each group).

3. Results

The socio-demographic and clinical features of OCD patients and controls are described in Table 2. The sample was age-, gender-, education-, and IQ-matched and all OCD patients were symptomatic, with Y-BOCS mean total score of 24.05. As expected, the OCD group had higher scores in the OCI-R and BDI when compared to controls. Regarding self-reported impulsivity, we found that OCD patients had statistically significant higher scores in the BIS Attention and Total subscale.

Since BIS attention was normally distributed (Shapiro-Wilk test = 0.95; df-34; p=. 19), an exploratory linear regression model was performed to investigate which factors, other than diagnostic group, were able to predict BIS attention scores. Accordingly, total OCI-R and the BDI scores were entered in the model for differing significantly between the OCD and controls groups. Eventually, a model that included OCI-R ($\beta = 0.54$; $p = .01$), but not BDI scores ($\beta = 0.28$; $p = .15$) or diagnostic status ($\beta = -0.11$; $p = .58$), was found to predict BIS attention scores (Overall model fit was R2 = 0.46).

Comparisons between OCD and controls on neurocognitive tests are portrayed in Table 3. No significant group differences in the performance of CGT, SST and IST by OCD and controls were found. In terms of the IST, there was the expected significant effect of condition for mean number of box opened [Wilks' Lambda = 0.39, F (1, 32) = 49.56; $p < .0001$). However, no interaction was found between the later variable and participants' diagnostic status, i.e. OCD or controls [Wilks' Lambda = 0.99, F (1,32) = 0.13, $p = .71$)]. Relationships between test performance and both the BDI and the SRI scores in the OCD group are depicted in Table 4. Although correlations were found between the mean number of box opened in the decreasing winning condition and BDI scores ($r = 0.48$, $p = .05$), no significant correlations between performance in neurocognitive tests and SRI doses was noted.

4. Discussion

In this study, we have performed a comprehensive assessment of impulsivity in OCD patients, both in terms of their self-reported (attentional, motor and non-planning impulsivity) and neurocognitive (reward/risk-taking, motor and reflection impulsivity) aspects, which

Table 2
Socio-demographic, clinical features and cognitive impulsivity of obsessive-compulsive disorder vs. healthy control sample.

	OCD patients (SD); n = 17	Control participants (SD); *n* = 17	Statistical tests
Socio-demographic features			
Age	35.88 (13.13)	35.29 (10.84)	$t = 0.14$; df = 32.00; $p = .88$
Gender (male)	14 (82.4%)	14 (82.4%)	Fisher's Exact test = 1.0
Education	14.47 (2.15)	14.76 (2.27)	$t = -3.87$; df = 32.00; $p = .70$
IQ	99.26 (10.52)	93.67 (9.88)	$t = 1.59$; df = 32.00; $p = .12$
GAF	46.76 (8.46)	93.00 (6.60)	$t = -17.75$; df = 32.00; p < .0001
Severity of symptoms			
OCI-R total	24.23 (11.33)	6.64 (6.33)	$t = 5.58$; df = 25.10; p < .0001
Checking	3.23 (3.23)	1.11 (1.49)	Z = -2.27; $p = .02$
Hoarding	2.29 (2.59)	1.11 (1.57)	Z = -1.90; p = .05
Neutralization	4.76 (4.56)	0.29 (0.77)	Z = -3.36; $p = .001$
Obsessing	6.05 (3.61)	1.47 (2.12)	Z = -3.66; $p = .0002$
Ordering	4.35 (2.62)	2.17 (1.77)	Z = -2.55; p = .01
Washing	3.52 (4.20)	0.47 (0.87)	Z = -2.43; p = .01
BDI	14.29 (7.42)	4.18 (4.88)	$t = 4.58$; df = 31.00; p < .0001
BIS			
Attention	18.82 (4.40)	15.29 (3.35)	$t = 2.63$; df = 32; p = .01*
Motor	17.41 (2.73)	16.58 (2.23)	$t = 0.96$; df = 32; $p = .34$
Non-planning	25.35 (3.63)	23.64 (3.51)	$t = 1.39$; df=; $p = .17$
Total	61.58 (8.52)	55.52 (6.32)	$t = 2.35$; df = 32; p = .02*
YBOCS			
Obsessions	11.70 (3.23)	–	
Compulsions	12.35 (2.69)	–	
Total	24.05 (5.43)	–	

OCD = Obsessive-Compulsive Disorder; GAF = Global Assessment of Functioning Scale; IQ = Intelligence Quotient; OCI-R = Obsessive-Compulsive Inventory – Revised; BDI = Beck Depression Inventory; BIS = Barratt Impulsivity Scale; Y-BOCS = Yale-Brown Obsessive-Compulsive Symptom Scale.

were then compared to those of age-, gender, education, and IQ matched controls. Despite predicting that adult OCD patients would present increased levels of impulsivity [11], we found only partial support for this hypothesis. In fact, OCD patients exhibited significant heightened impulsivity that was restricted to the self-reported (particularly attentional) domain and was largely determined by the severity of OCD symptoms. Thus, our findings suggest that, despite describing themselves as impulsive, adult OCD patients do not show objective neurocognitive evidence of such abnormalities.

While the finding of increased BIS scores seems intuitive on the basis of phenomenological descriptions of OCD patients [who frequently report not being "in control" despite any objective evidence [56]], the fact that increased self-reported impulsivity in our OCD sample could be credited mostly to greater attentional impulsivity has already been described in previous studies with other OCD samples [16,19–21]. Actually, it is interesting that, similarly to OCD obsessions (described as recurrent and persistent thoughts, urges, or images experienced as intrusive and unwanted in DSM-5) [57], increased attentional impulsivity has been related to the inability of "deleting no-longer-relevant information" from working memory [58].

Dissociation between self-reported and neurocognitive impulsivity in our OCD sample dovetails with studies showing that self-report and laboratory behavioural assessments of impulsivity are often unrelated to each other [38,59]. We can only speculate on the reasons for this dissociation within our sample. Firstly, as previously suggested, they may actually relate to different constructs [38] regardless of the nature of the population under study. Secondly, in light of previous studies showing increased rates of childhood attention deficit hyperactivity disorder (ADHD) symptoms in OCD patients [60], and several findings showing etiological and pathophysiological links between the two conditions [61], longitudinal studies could investigate whether increased self-reported impulsivity in OCD adults can be ascribed to previous ADHD symptoms (even subsyndromal) or to an early neurocognitive impulsivity that vanishes later due to the progressive maturation of fronto-subcortical circuits. [62]

Our negative findings regarding decision-making are consistent with a substantial part of the literature. For instance, previous studies with the CGT did not find evidence of an impaired CGT performance in OCD [22,29,63,64]. In fact, several studies that reported impaired "decision-making" in OCD used the Iowa Gambling Task [65–67], which has also been criticized for being unable to isolate risk preference from working memory abilities due to its emphasis on learning that the task demands [54]. Thus, although OCD patients may occasionally show "risky" symptoms under conditions of uncertainty (such as, for

Table 3
Neurocognitive performances of obsessive-compulsive disorder patients vs. healthy control participants.

	OCD patients ($n = 17$)	Control participants (n = 17)	Statistical tests
Cambridge gambling task			
Overall proportion bet	0.44 (0.13)	0.51 (0.16)	$t = -1.21$; df = 32; $p = .23$
Quality of decision	0.85 (0.19)	0.91 (0.10)	$t = -1.13$; df = 32; $p = .26$
Risk adjustment	1.49 (1.42)	1.36 (1.57)	Z = −0.19; $p = .85$
Stop signal task			
Mean correct reaction time on go trials	512.64 (120.90)	517.11 (145.52)	$t = -0.09$; df = 32; $p = .92$
Information sampling task			
Mean number of box opened/trial			
W/ fixed winning	15.52 (6.11)	16.14 (6.32)	$t = -0.29$; df = 32; $p = .77$
W/ decreasing winning	9.23 (3.70)	10.47 (4.66)	$t = -0.85$; df = 32; $p = .39$

OCD = Obsessive-Compulsive Disorder.

Table 4
Correlations between neuropsychological performance, depressive symptoms and doses of serotonin reuptake inhibitors.

	BDI scores	SRI scores
Cambridge gambling task		
Overall proportion bet	$r = -0.41$	$r = -0.16$
	$p = .10$	$p = .53$
Quality of decision	$r = 0.26$	$r = 0.27$
	$p = .30$	p = .30
Risk adjustment	$r = 0.25$	r = 0.26
	$p = .32$	$p = .31$
Stop signal task		
Mean correct reaction time on go trials	$r = -0.01$	r = 0.27
	$p = .97$	$p = .28$
Information sampling task		
Mean number of box opened, fixed winning	$r = 0.07$	$r = 0.10$
	p = .77	p = .71
Mean number of box opened, decreasing winning	r = 0.48	$r = 0.17$
	p = .05	$p = .49$

BDI: Beck Depression Inventory; SRI: Serotonin reuptake inhibitors.

instance, avoiding drinking water as a way of reducing urinary elimination because of the fear of contamination in toilets [68]), we were unable to identify neurocognitive evidence of increased risk taking under uncertainty.

Contrary to expectation, we did not find differences in the SST performance between OCD and controls. Although studies report impaired motor response inhibition in OCD samples [17,18,29,69], a finding confirmed by one meta-analysis [24], SST performance in OCD is still a controversial matter [26]. It is tempting to speculate that differences between studies may depend on the particular OC symptom domains; i.e. some domains may be more strongly associated with this deficit than others. For example, predominance of overt (motor) vs. covert (mental) ritualistic behaviours may be important. Interestingly, our patients' scores on cognitive (obsessing and neutralization) symptoms were higher than on behavioural symptoms (checking and washing). Although we are not aware of specific instruments available to measure overt vs. covert rituals in a consistent manner, future studies could consider comparing SST performance between OCD subjects with predominant motor vs. mental rituals.

Finally, a lack of group differences in the number of boxes opened for the IST confirms previous findings on this CANTAB task in OCD [29]. Although in the present study we were more interested in testing impulsive responses in the IST, lack of any difference between OCD and controls on the IST is surprising, as the IST has also been argued by some to measure intolerance of uncertainty (the "incapacity to endure the aversive response triggered by the perceived absence of salient, key, or sufficient information") [70], a construct that had been proposed to be shared by OCD and other related disorders, including generalized anxiety disorder [71]. In this regard, a recent study found increased decision thresholds on a modified reflection-impulsivity task in adolescent OCD [30]. This would be more in keeping with intolerance of uncertainty and we suspect that the latter task, which also uses computational modeling, may be more behaviorally sensitive to cognitive changes in OCD [30].

Our study has some limitations. Firstly, it included few participants. Thus, confirmation of its negative findings using a larger sample is advisable. In fact, the study was only powered to detect group differences of large effect sizes; and might have missed subtler group differences (i.e. those with small-medium effect sizes). Similarly, also due to the sample size, no correction for multiple comparisons was performed. Further, despite being substantially symptomatic [mean YBOCS = 24.05 (5.43)], most patients were receiving SRIs, which could theoretically affect at least some aspects of cognition. Nevertheless, no correlation between SRIs doses and cognition was found, suggesting that our results are not accounted by this possibility. Accordingly, other studies have shown that both motor [72] and reward [73] impulsivities are not reliably impacted by SRIs. However, as SRIs may interfere with reflection impulsivity [74], we feel that a follow up study assessing drug free patients both before and after pharmacotherapy would be the best way to investigate the impact of SRI on the neurocognition of OCD patients.

Some relevant phenotypic features, such as age at OCD onset and comorbidity profile (e.g. current or past ADHD), were not assessed in our sample. Bearing in mind theories (such as the behavioural addiction model of OCD) suggesting that increased impulsivity may be restricted to patients with longer duration of illness [12], it would have been interesting to clarify whether our sample was characterized by recent onset OCD. Although we are unable to rule out this possibility, it seems unlikely. Our sample included mostly adult patients [mean age at assessment = 35.88 (13.13) years]. As OCD has been suggested to start in child and adolescent years in up to 80% of cases [75], it is reasonable to speculate that our patients had a long history of OCD. Lastly, we did not rule out a history of some mental disorders (e.g. major depression) in the control sample, a methodological aspect that differs from some previous cognitive studies, which may have diminished the ability to detect deficits in patients. Despite these methodological caveats, our findings suggest that the impulsivity features reported in OCD subjects are more subjective (rather than objective) and largely determined by severity of obsessive-compulsive symptoms.

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Declaration of competing interest

None.

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