J. Behav. Ther. & Exp. Psychiat. 46 (2015) 66–71

Contents lists available at ScienceDirect

Journal of Behavior Therapy and Experimental Psychiatry

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

Response inhibition in a subclinical obsessive-compulsive sample

Amitai Abramovitch [a, *], Noa Shaham [b], Lior Levin [b], Moran Bar-Hen [b], Avraham Schweiger [b]

[a] Department of Psychiatry, Massachusetts General Hospital & Harvard Medical School, Boston, MA, USA
[b] Department of Psychology, The Academic College of Tel Aviv, Tel Aviv, Israel

ARTICLE INFO

Article history:
Received 17 April 2014
Received in revised form
1 September 2014
Accepted 2 September 2014
Available online 10 September 2014

Keywords:
OCD
Obsessive-compulsive disorder
Executive function
Analogue sample
Endophenotype
Neuropsychology

ABSTRACT

Background and objectives: Inconsistent findings across studies challenge the viability of response inhibition (RI) as an endophenotype of obsessive-compulsive disorder (OCD). Contemporary conceptualization of endophenotypes in psychiatric disorders suggests that these markers vary continuously in the general population, highlighting the importance of analogue sample research. Although neuropsychological functions have been studied in subclinical obsessive-compulsive (OC) samples, no study to date had examined RI in the context of the go/no-go paradigm.
Methods: A subclinical OC sample (HOC; $n = 27$) and a low OC symptoms control sample (LOC; $n = 25$), as determined by the Obsessive-Compulsive Inventory-Revised, completed a go/no-go task and clinical questionnaires.
Results: The groups did not differ on age, gender, or state anxiety. Controlling for depressive severity, the HOC group made significantly more commission errors and exhibited larger response time variability on the go/no-go task. However, standardized scores produced using population norms revealed that the HOC group performed within normative range.
Limitations: This study used a non-clinical sample and no structured clinical screening was performed.
Conclusions: Compared to LOC participants, a psychometrically-defined subclinical OC sample exhibited deficient RI and sustained attention. However, when raw scores were converted to age and education adjusted standardized scores according to the test's population norms, the HOC group task performance was in the normative range. These results, are in line with findings in OCD samples, suggesting that moderate degree of RI deficiencies is associated with the presence of OC symptomatology regardless of clinical status. However, the conceptualization of RI underperformance as an OCD disorder-specific impairment, remains controversial.

1. Introduction

Obsessive-compulsive disorder (OCD) is a prevalent (2.5%; Ruscio, Stein, Chiu, & Kessler, 2010), and frequently debilitating disorder, characterized by obsessions and/or compulsions that are performed in order to reduce distress (American Psychiatric Association, 2013). Functional imaging studies have claimed to be consistent in their support of the cortico-striato-thalamo-cortical (CSTC) neurobiological model of OCD (Saxena & Rauch, 2000), highlighting aberrant frontostriatal functioning (for a review see Pauls, Abramovitch, Rauch, & Geller, 2014). In fact, whereas some changes to this model have been recently proposed (Milad & Rauch, 2012), this body of literature is considered by many to be amongst the most robust in psychiatric literature (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005). These models predict neuropsychological impairments in OCD, especially in the domain of executive functions, which the frontostriatal circuits are presumed to subserve. Indeed, research suggests that OCD may be associated with deficit in executive functions (Bannon, Gonsalvez, Croft, & Boyce, 2002; Chamberlain et al., 2005; Kuelz, Hohagen, & Voderholzer, 2004). However, in contrast to the consistent results seen across resting-state imaging studies, the large body of neuropsychological literature in OCD is characterized by inconsistent, and statistically heterogeneous results (Abramovitch, Abramowitz, & Mittelman, 2013; Kuelz et al., 2004).

* Corresponding author. Department of Psychiatry, Massachusetts General Hospital, 185 Cambridge Street, Suite 2000, Boston, MA 02114, USA. Tel.: +1 617 643 9934; fax: +1 617 643 3080.
E-mail addresses: aabramovitch@mgh.harvard.edu, amitai.mail@gmail.com (A. Abramovitch).

http://dx.doi.org/10.1016/j.jbtep.2014.09.001
0005-7916/

Several attempts to account for this inconsistency have been offered. Among them: 1) the use of different neuropsychological tests to examine similar constructs (Kuelz et al., 2004); 2) inconsistent application of corrections for multiple comparisons (Purcell, Maruff, Kyrios, & Pantelis, 1998); 3) the potential confounding effects of medication (Mataix-Cols, Alonso, Pifarre, Menchon, & Vallejo, 2002); 4) depressive severity (Basso, Bornstein, Carona, & Morton, 2001); 5) age of onset (Roth, Milovan, Baribeau, & O'Connor, 2005); 6) gender (Mataix-Cols et al., 2006); 7) comorbid conditions (Aycicegi, Dinn, Harris, & Erkmen, 2003); and 8) OCD symptom dimensions (Lawrence et al., 2006). However, no single factor, or combination of factors, was found to have a significant moderating effect that may account for this heterogeneity. In fact, in a recent meta-analysis examining neuropsychological performance in OCD, a comprehensive moderator analysis yielded no significant moderating effects of clinical or demographic factors, despite findings of statistically significant heterogeneity across neuropsychological domains (Abramovitch et al., 2013). The unexplained inconsistency among neuropsychological investigations in OCD hinders the identification of disorder-specific neurocognitive markers. These inconsistencies notwithstanding, the authors found that OCD is characterized by underperformance on several neuropsychological domains, including executive function, processing speed, and nonverbal memory. The overall magnitude of the differences found between OCD and control samples, however, was of moderate size, leading to the conclusion that individuals with OCD may underperform on neuropsychological tasks, but that these deficiencies may not fit the classic neuropsychological definition of clinically significant impairments in these domains (Abramovitch et al., 2013).

Response inhibition (RI), the ability to inhibit a pre-potent motor response, is a prominent executive function that is of particular interest to OCD researchers. Based on the phenotype of repetitive rituals and intrusive obsessions, it has been initially thought that OCD may be characterize by impairments in the ability to inhibit thoughts and behaviors (e.g., Penades et al., 2007). Consequently, RI has been proposed as a candidate endophenotypic marker of OCD (Chamberlain et al., 2005). This notion received support from a number of studies reporting deficient performance on tasks of RI, as well as from findings regarding familial RI deficits in OCD (Chamberlain et al., 2005). However, research into RI in OCD has produced contradictory results. Some studies reported reduced performance on tasks of RI in OCD (e.g., Abramovitch, Dar, Hermesh, & Schweiger, 2012; Martinot et al., 1990; Menzies et al., 2007; Penades et al., 2007), and yet others reported no performance differences between OCD patients to non-psychiatric controls (e.g., Bohne, Savage, Deckersbach, Keuthen, & Wilhelm, 2008; Boone et al., 1991; Krishna et al., 2011).

Several neuropsychological tasks have been used to examine RI in OCD. These include the Stop Signal Task, the Stroop test, continuous performance tests (CPT), and go/no-go tests. In the go/no-go paradigm, the index for RI is the number of commission errors (i.e., response to a no-go stimuli). Interestingly, across measures of response inhibition, Abramovitch et al. (2013) reported an overall medium effect size of .49 for RI, with a confidence interval of .61 to .04, and an overall Cohen's *d* effect size of .33 for differences between OCD and healthy controls on commission errors (Abramovitch et al., 2013). Notably, a recent meta-analysis of response inhibition across mental disorders found similar small-to-medium effect sizes across psychiatric disorders and concluded that response inhibition deficits are insufficiently sensitive or specific to be used as a biomarker in most mental disorders (Wright, Lipszyc, Dupuis, Thayapararajah, & Schachar, 2014).

In light of the overall unexplained inconsistency, we sought to examine the association between response inhibition and OCD phenomena using a go/no-go task, in a subclinical obsessive-compulsive (OC) sample (i.e., a samples of high and low OC symptoms). Previous research has indicated that examining OC phenomena in non-clinical samples is a viable means of investigation, that has been consistently contributing to our understanding of OCD (Burns, Formea, Keortge, & Sternberger, 1995; Gibbs, 1996). This notion received support from a recent comprehensive review of analogue sample research in OCD, suggesting that OCD symptoms are dimensional rather than categorical (i.e., they fall on a continuum from very mild to severe), and share similar qualitative characteristics across clinical and non-clinical populations (Abramowitz et al., 2014). Moreover, the use of analogue OCD samples may be particularly advantageous in examining cognitive functions, given the absence of potentially confounding factors such as medications or heterogeneous treatments (Mataix-Cols, 2003). Finally, as suggested by Cannon and Keller (2006), endophenotypes should vary continuously in the general population. The authors noted that, “Rather than binning all nonaffected individuals into a single category, continuous measures allow for the discernment of differences (i.e., scaling of liability) in the nonaffected population” (Cannon & Keller, 2006, p. 276). The authors further suggested that research into endophenotypes of psychiatric disorders should optimally include findings from different levels of analyses, and specifically, should comprise investigations of such markers in the general population (i.e., analogue samples).

A relatively limited body of research has been published on neuropsychological functioning in analogue OCD samples, especially those examining executive functions. In general, compared to individuals characterized by lower levels of OC symptom severity (LOC), individuals with higher levels of OC symptom severity (HOC) exhibit comparable performance on tasks of verbal and non-verbal memory (Kim, Jang, & Kim, 2009; Mataix-Cols, Junque, et al., 1999). However, consistent with neuropsychological studies of OCD, subclinical OC research concerning executive functions has yielded mixed results. Some studies found comparable performance between HOC and LOC on the Stroop test, Wisconsin Card Sorting test (WCST), verbal fluency test, and the Trail Making Test (i.e., TMT; Hajcak & Simons, 2002; Kim et al., 2009; Mataix-Cols, Barrios, Sanchez-Turet, Vallejo, & Junque, 1999; Mataix-Cols, Junque, et al., 1999). In contrast, relative to LOC samples, HOC samples were found to underperform on tasks assessing planning (Tower of Hanoi task), as well as on design fluency tasks, the Delayed Alteration tests, the WCST, and on the TMT (Kim et al., 2009; Mataix-Cols, Barrios, et al., 1999; Mataix-Cols, Junque, et al., 1999; Spitznagel & Suhr, 2002). To our knowledge, no study to date has utilized the go/no-go paradigm to directly assess response inhibition in a subclinical OC sample. One study (Mataix-Cols et al., 1997), however, utilized the Identical Pairs version of the Continuous Performance test (CPT-IP), and found a significant interaction effect between group and CPT-IP subscales (i.e., verbal and spatial), but no difference on commission errors between the groups.

To address this gap in the literature, the present study was designed to examine response inhibition among HOC and LOC college students, by comparing their performance on a go/no-go task while controlling for potential confounding factors. In order to aid in distinguishing between underperformance and impairment, we utilized the NeuroTrax computerized Expended Go/No-Go test. This test (described in more detail in Section 2.2.2) produces two scores automatically for every outcome measure: a raw score and a standard score, computed using the NeuroTrax normative data. These standard scores are similar to the ones produced by the Wechsler Intelligence Scale, in which standardized scaled scores have a mean of 100 and a standard deviation (SD) of 15. In accordance with these aims, and in light of the

neuropsychological literature on response inhibition reviewed above, we hypothesized that HOC participants will underperform compared to LOC participants on the go/no-go test, but that these differences would not constitute 'impaired performance' (more than 2 standard deviations below normative population means), as indicated by the standardized scaled scores assessed according to conventions in clinical neuropsychology (Lezak, Howieson, Bigler, & Tranel, 2012).

2. Methods

2.1. Participants

Participants were 52 undergraduate college students, recruited from a pool of 212 students on the basis of their scores on the Obsessive-Compulsive Inventory – Revised (OCI-R; Foa et al., 2002). Employing the definition used by Mataix-Coles et al. (e.g., Mataix-Cols, 2003; Mataix-Cols, Barrios, et al., 1999), an OCI-R cutoff score ≥1 SD was predetermined as the inclusion criteria for the subclinical OC group (HOC). Twenty-seven participants (OCI-R > 29; 28% males) comprised the HOC group and 25 participants who scored lower than one SD below the sample mean (OCIR ≤ 10; 44.4% males) comprised the LOC group. The OCI-R mean scores for the HOC and LOC groups (see Table 2) were similar to scores typically obtained by OCD patients and healthy controls, respectively (e.g., Abramovitch et al., 2012; Lee, Yost, & Telch, 2009). Participants received course credit for their participation in this study. The study was approved by the Institutional Review Board in accordance with the declaration of Helsinki.

2.2. Measures

2.2.1. Clinical measures

The *Obsessive-compulsive inventory - revised* (OCI-R; Foa et al., 2002) was used to assess the severity of obsessive-compulsive symptoms. The OCI-R consists of 18 OCD-related symptoms. Participants are asked to rate the extent to which they have been bothered by these symptoms over the past month on a four-point Likert scale, ranging from 0 (not at all) to 4 (extremely bothered). The OCI-R has been shown to possess very good psychometric properties, including test-retest reliability and internal consistency in clinical and non-clinical samples (Foa et al., 2002; Hajcak, Huppert, Simons, & Foa, 2004). We used the *Eysenck's Impulsiveness Venturesomeness Empathy scale* (IVE; Eysenck & Eysenck, 1978) to assess self-reported behavioral impulsivity. The Beck Depression Inventory II (BDI-II; Beck, Steer, & Brown, 1996) was used to assess severity of depressive symptoms, and the 6-item state scale of the State-Trait Anxiety Inventory (STAI; Marteau & Bekker, 1992) was administered for the assessment of state anxiety.

Table 1
Demographics and clinical characteristics of the high and low obsessive-compulsive samples.

	LOC ($N = 25$)		HOC ($N = 27$)		$F(1,50)$	*Sig*
	M	*SD*	*M*	*SD*		
Age	24.12	2.20	24.16	2.46	.00	.95
BDI-II	18.40	9.47	6.04	4.04	36.37	.00
STAI-State	14.44	1.96	13.88	1.53	1.31	.25
OCI-R Total Score	36.63	5.82	6.00	2.56	585.32	.00
IVE Impulsiveness	9.92	5.54	7.60	3.36	3.27	.07

HOC, high obsessive compulsive participants; LOC, low obsessive compulsive participants; BDI-II, beck depression inventory II; STAI-State, state-trait anxiety inventory - State; OCI-R, obsessive-compulsive inventory-revised; IVE, Eysenck's impulsiveness-venturesomeness-empathy scale.

2.2.2. Neuropsychological task

The *Expanded Go/No-Go task* is a subtest of the NeuroTrax computerized neuropsychological battery (Neurotrax, 2003). The NeuroTrax testing battery is a standardized, widely used computerized neuropsychological battery for cognitive assessment. Skills assessed are verbal and non-verbal memory, attention, processing speed, visual spatial abilities, verbal function, motor skill, and executive function. For the purpose of the current study, participants completed only the Expanded Go/No-Go subtest. The Expanded Go/No-Go test requires the participants to respond as quickly as possible to a sequence of individually presented colored squares (blue, green, and white; go stimuli) by clicking the mouse button. This rule has one exception, according to which participants are instructed to avoid responding to red squares (no-go stimulus). The battery's data output consists of two types of data: raw data, as well as age, and education-adjusted normalized scores ($M = 100$ and $SD = 15$) produced using the test's normative data. The automated test also produces raw and standardized scores for speed-accuracy tradeoff [(accuracy/reaction time)*100]. The Expanded Go/No-Go test demonstrates very good psychometric properties across normal control subjects and psychiatric or neurological populations, including OCD and ADHD (Abramovitch, Dar, Schweiger, & Hermesh, 2011; Schweiger, Abramovitch, Doniger, & Simon, 2007).

2.3. Procedure

A double-blind procedure was employed, according to which both the study's trained experimenters as well as the participants were not aware of the groups to which each participant had been assigned. Participants were tested individually in a quiet room. To avoid the effects of fatigue, after signing an informed consent, all participants first took the 14-min computerized go/no-go task on a desktop computer with a 19" flat screen, followed by completion of the STAI, BDI-II, and IVE questionnaires.

2.4. Data analysis

Statistical analyses were conducted using the IBM Statistical Package for the Social Sciences (SPSS) version 20.0 (IBM, 2011). Univariate analysis of variance (ANOVA) was used to analyze continuous demographic and clinical variables (i.e., age, OCI-R, BDI-II, STAI and IVE). Differences in gender proportions were analyzed using Pearson's χ^2 test. Significant group effects on the expanded go/no-go test's primary outcome measures (commission errors, omission errors, response time, response time standard deviation) were analyzed using multivariate analysis of covariance (MANCOVA; controlling for depressive severity), followed by individual univariate ANOVAs. Finally, we analyzed the association between behavioral impulsivity (IVE) and response inhibition (commission errors) using Pearson's correlation analysis.

3. Results

The groups did not differ significantly on gender distribution (LOC males = 28.0%, HOC males = 44.4%; $\chi^2(1) = 1.54$, $p = 0.22$), as well as age in years (Table 1). In addition, the HOC group scored significantly higher on the BDI-II and OCI-R than that LOC group (Table 1). The mean BDI-II score for the HOC group reflects mild severity of depressive symptoms and, as mentioned above, was consequently held constant in subsequent neuropsychological analyses. The groups did not differ significantly on the IVE-impulsivity and the STAI-state scores.

All four go/no-go outcome measures (i.e., response time, response time standard deviation, omission errors, and commission errors) were entered into the MANCOVA model, controlling for

Table 2
Comparison of raw and standardized scores for the go/no-go outcome measures controlling for depressive severity.

	HOC ($N = 27$)				LOC ($N = 25$)				$F(1,50)$[a]	*ES*
	Raw scores		Standardized scores		Raw scores		Standardized scores			
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*		
Response Time	379.44	48.14	104.04	15.27	371.84	33.20	105.34	9.28	1.86	0.18
Response Time SD	75.92	24.89	102.87	13.99	67.72	11.75	107.12	5.91	5.26*	0.42
Omission Errors	1.18	2.11	102.21	8.42	1.72	2.80	100.84	12.17	1.69	0.22
Commission Errors	5.66	3.90	95.61	17.71	4.24	2.53	101.10	10.56	5.02*	0.43

*$p < 0.05$; HOC, high obsessive-compulsive participants; LOC, low obsessive-compulsive participants; SD, standard deviation.
[a] Analyses of raw scores.

depressive severity. The results revealed an overall significant difference between the groups (Wilks' Lambda = .81, $p = 0.04$, partial $\eta^2 = .19$). Results of the subsequent univariate analyses (presented in Table 2) revealed that the HOC group made significantly more commission errors and exhibited greater response time variability (i.e., higher response time standard deviation scores). Standardized scores produced from the HOC group's raw scores (Table 2) were all within the normative range; spanning from 95.6 to 104. This span corresponds to a range between −.29 and .27 SD difference between the HOC group and the mean score of age and education adjusted controls from the test's normative data. Finally, a separate ANCOVA, aimed at examining group difference in speed-accuracy tradeoffs, revealed no significant difference between the HOC (raw $M = 25.38$, $SD = 2.70$; standardized $M = 103.46$, $SD = 16.31$) and LOC (raw $M = 25.99$, $SD = 2.17$; standardized $M = 105.06$, $SD = 11.35$; $F(1, 49) = 2.85$, $p = 0.10$).

Pearson's correlation analyses between the IVE impulsiveness score, the OCI-R total score, and mean commission errors yielded no significant correlations in the entire sample (all $p > 0.05$). These null results were similar when computed separately within each group, with the exception of a significant negative correlation between the IVE impulsiveness score and OCI-R scores within the HOC group ($r = -.39$, $p = 0.047$).

4. Discussion

The present study is the first to examine response inhibition in a psychometrically-defined subclinical OC sample. Our aim was to examine the association between response inhibition and OC phenomena over and above clinical status, especially in consideration of the inconsistency and controversy extant regarding response inhibition research in OCD. The utilization of psychometrically-defined subclinical OC, or 'analogue' samples, in exploring basic questions regarding the nature of this disorder, is considered a valid methodology that, has contributed substantially to our understanding of OCD (Abramowitz et al., 2014; Burns et al., 1995; Gibbs, 1996; Mataix-Cols, 2003). Furthermore, the current understanding of endophenotypes in psychiatry research (i.e., the measurable components between genotype and clinical phenotype; Gottesman & Gould, 2003), is that such markers lie on a continuum in the population, and consequently require complementary investigations in the general population (Cannon & Keller, 2006).

Our results demonstrate that compared to a LOC sample, HOC individuals underperform on a go/no-go task. Specifically, the HOC group made significantly more commission errors, suggestive of reduced response inhibition abilities, and exhibited significantly larger response time variability, suggestive of reduced sustained attention. These differences were not accounted for by depressive severity, state anxiety, gender or age. The groups did not differ on omission errors, response time, or speed/accuracy tradeoff. These results are in accord with studies reporting reduced performance on tests of response inhibition in general, and with a few studies reporting elevated number of commission errors in clinical OCD samples (Abramovitch et al., 2011; Ghisi, Bottesi, Sica, Sanavio, & Freeston, 2013; Penades et al., 2007; da Rocha, Alvarenga, Malloy-Diniz, & Correa, 2011). Our results regarding deficient sustained attention are in line with findings reported in a subclinical OC sample (Mataix-Cols et al., 1997). They are also consistent with findings of elevated RT SD on a go/no-go test in a sample of OCD patients (Abramovitch et al., 2011). The latter findings seem to be countervailed by most studies which did not find deficient sustained attention in OCD samples (Lee, Chiu, Chiu, Chang, & Tang, 2009). However, these studies did not assess RT SD, a well validated measure for sustained attention, within the CPT or the go/no-go paradigms.

These findings are in accordance with the predictions of the prevailing CSTC neurobiological model of OCD (Saxena & Rauch, 2000), which predicts deficient neuropsychological task performance - predominantly in executive functions — that the frontostriatal system is thought to subserve (Pauls et al., 2014). Our results, evidenced cognitive underperformance in a subclinical OC sample, may be in line with other studies showing reduced cognitive test performance in remitted OCD patients (Bannon, Gonsalvez, Croft, & Boyce, 2006). Although such results may be construed as supporting contemporary understanding of endophenotypes — inherently perceived as a state-independent trait features of mental disorders — it is well-nigh impossible to exclude a state-related mechanism. For example, some authors suggested that neuropsychological deficits in OCD are an epiphenomenon (Abramovitch et al., 2011; Moritz, Hottenrott, Jelinek, Brooks, & Scheurich, 2012), resulting from OC symptoms. Specifically, the Executive Overload Model of OCD (Abramovitch et al., 2012) suggests that the overflow of obsessive thoughts overloads the executive system, hindering the ability of individuals with OCD from exhibiting their full cognitive capacity, contingent upon the intensity of obsessive thoughts at the time of testing. Thus, it is plausible that HOC individual's underperformance on the go/no-go task is due to increased obsessive thoughts compared to the LOC sample. It is also hypothetically conceivable that a trait-like tendency towards somewhat lower cognitive abilities could be further affected negatively by state-dependent levels of obsessive thoughts. Ultimately, the state versus trait controversy (including the possibility of a dual state-trait mechanism) underlying neuropsychological underperformance, is underresearched and further investigations in this area is sorely needed.

The secondary aim of the present investigation was to examine whether reduced performance on the go/no-go task is of sufficient magnitude to be interpreted as an impairment in this domain. Utilizing a well-validated, computerized go/no-go task, we were able to examine both raw scores as well as standardized scores. Examination of the HOC standardized scores (benchmark $M = 100$,

SD = 15) revealed that test performance was within the normative range (range 96–104), when compared to the test's normative data. In the realm of neuropsychological testing, test performance may be considered impaired when test scores fall at least two standard deviations below the age-adjusted population's mean (Lezak et al., 2012, p. 163). For example, the Wechsler intelligence scale defines a difference of 1–2 SD as 'low average', and 2 SD and higher, as 'borderline' performance (Wechsler, 2008). Thus, although exhibiting statistically significant lower scores than the LOC group, the HOC group's standardized scores cannot be interpreted as reflective of impairment in response inhibition as defined in the realm of clinical neuropsychology.

Our results are commensurate with research findings in OCD samples, where reduced performance on tests of response inhibition in general is a common finding. In addition, the effect size for response inhibition in the present study was $d = .43$. This small-to-medium effect size corresponds with the results of a recent meta-analysis of neuropsychological functions in OCD (Abramovitch et al., 2013), in which the authors report an effect size of .49 for differences in response inhibition, and specifically an effect size of .33 for commission errors. Furthermore, in the present study we found no association between neuropsychological test performance and depressive or OC symptom severity across the entire sample, as well as within each group—a finding that has been repeatedly reported in OCD clinical samples (e.g., Bedard, Joyal, Godbout, & Chantal, 2009; Bucci et al., 2007; Simpson et al., 2006), as well as in analogue OCD samples (Kim et al., 2009; Mataix-Cols, Junque, et al., 1999; e.g., Mataix-Cols et al., 1997).

Our results support the association between the presence of OCD symptomatology and reduced neuropsychological performance that may be viewed as dimensional. That is, this association may be present in highly functioning students with elevated OC symptoms. This notion is supported by an overall lack of association between neuropsychological test performance and functional impairments in clinical OCD samples (Abramovitch et al., 2013).

The strength of the present study lies in that it is the first examination of RI—a suggested OCD endophenotype— in a psychometrically-defined subclinical OC sample, and in which potential confounds were controlled (i.e., depressive severity, state anxiety, age, and education). In addition, we were able to examine both raw and standardized tests scores. However, a limitation of the present study is the lack of a semi-structured screening interview. Thus, it is possible that some participants may have had OCD or other disorders. Nevertheless, our HOC group had high OCI-R scores that are comparable to scores reported in carefully diagnosed OCD patients, whereas our LOC group had OCI-R scores observed in carefully screened healthy controls (Abramovitch et al., 2012; Foa et al., 2002).

5. Conclusion

In conclusion, the present study demonstrates that HOC individuals underperformed on a go/no-go test when compared to LOC individuals, suggesting reduced response inhibition abilities (i.e., elevated commission errors), as well as reduced sustained attention (i.e., RT SD). However, HOC performance on these measures was found to lie within the normative range when compared to the general population norms. Thus, these results indicate that OCD symptomatology may be associated with a moderate degree of deficient performance, yet this may not constitute an inherent clinical impairment. This notion is supported by findings from a recent comprehensive meta-analytical examination of neuropsychological test performance in OCD. This notion may also account for the unusual variability among studies and the lack of significant association between response inhibition and OCD symptom severity. Future studies should investigate the association between RI and OCD symptomatology using tests of different paradigms (i.e., go-no/go, CPT, SST and Stroop), as well as attend to the rarely studied association with OCD symptom dimensions.

Acknowledgment

We thank the participants and research staff for their time and efforts. We also like to thank Amelia P. Tankersley for her help with the preparation of this manuscript.

References

Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive-compulsive disorder: a meta-analysis. *Clinical Psychology Review, 33*(8), 1163–1171. http://dx.doi.org/10.1016/j.cpr.2013.09.004.

Abramovitch, A., Dar, R., Hermesh, H., & Schweiger, A. (2012). Comparative neuropsychology of adult obsessive-compulsive disorder and attention deficit/hyperactivity disorder: implications for a novel executive overload model of OCD. *Journal of Neuropsychology, 6*(2), 161–191. http://dx.doi.org/10.1111/j.1748-6653.2011.02021.x.

Abramovitch, A., Dar, R., Schweiger, A., & Hermesh, H. (2011). Neuropsychological impairments and their association with obsessive-compulsive symptom severity in obsessive-compulsive disorder. *Archives of Clinical Neuropsychology, 26*(4), 364–376.

Abramowitz, J. S., Fabricant, L. E., Taylor, S., Deacon, B. J., McKay, D., & Storch, E. A. (2014). The relevance of analogue studies for understanding obsessions and compulsions. *Clinical Psychology Review, 34*(3), 206–217. http://dx.doi.org/10.1016/j.cpr.2014.01.004.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5* (5th ed.). Arlington, VA: American Psychiatric Association.

Aycicegi, A., Dinn, W. M., Harris, C. L., & Erkmen, H. (2003). Neuropsychological function in obsessive-compulsive disorder: effects of comorbid conditions on task performance. *European Psychiatry, 18*(5), 241–248.

Bannon, S., Gonsalvez, C. J., Croft, R. J., & Boyce, P. M. (2002). Response inhibition deficits in obsessive-compulsive disorder. *Psychiatry Research, 110*(2), 165–174.

Bannon, S., Gonsalvez, C. J., Croft, R. J., & Boyce, P. M. (2006). Executive functions in obsessive-compulsive disorder: state or trait deficits? *Australian and New Zealand Journal of Psychiatry, 40*(11–12), 1031–1038.

Basso, M. R., Bornstein, R. A., Carona, F., & Morton, R. (2001). Depression accounts for executive function deficits in obsessive-compulsive disorder. *Neuropsychiatry Neuropsychology and Behavioral Neurology, 14*(4), 241–245.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck depression inventory - Second edition manual*. San Antonio: The Psychological Corporation.

Bedard, M. J., Joyal, C. C., Godbout, L., & Chantal, S. (2009). Executive functions and the obsessive-compulsive disorder: on the importance of subclinical symptoms and other concomitant factors. *Archives of Clinical Neuropsychology, 24*(6), 585–598.

Bohne, A., Savage, C. R., Deckersbach, T., Keuthen, N. J., & Wilhelm, S. (2008). Motor inhibition in trichotillomania and obsessive-compulsive disorder. *Journal of Psychiatric Research, 42*(2), 141–150.

Boone, K. B., Ananth, J., Philpott, L., Kaur, A., et al. (1991). Neuropsychological characteristics of nondepressed adults with obsessive-compulsive disorder. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 4*(2), 96–109.

Bucci, P., Galderisi, S., Catapano, F., Di Benedetto, R., Piegari, G., Mucci, A., et al. (2007). Neurocognitive indices of executive hypercontrol in obsessive-compulsive disorder. *Acta Psychiatrica Scandinavica, 115*(5), 380–387.

Burns, G. L., Formea, G. M., Keortge, S., & Sternberger, L. G. (1995). The utilization of nonpatient samples in the study of obsessive compulsive disorder. *Behaviour Research and Therapy, 33*(2), 133–144. http://dx.doi.org/10.1016/0005-7967(94)00039-M.

Cannon, T. D., & Keller, M. C. (2006). Endophenotypes in the genetic analyses of mental disorders. *Annual Review of Clinical Psychology, 2*(1), 267–290. http://dx.doi.org/10.1146/annurev.clinpsy.2.022305.095232.

Chamberlain, S. R., Blackwell, A. D., Fineberg, N. A., Robbins, T. W., & Sahakian, B. J. (2005). The neuropsychology of obsessive compulsive disorder: the importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neuroscience and Biobehavioral Reviews, 29*(3), 399–419.

Eysenck, S. B., & Eysenck, H. J. (1978). Impulsiveness and venturesomeness: their position in a dimensional system of personality description. *Psychological Reports, 43*(3 Pt 2), 1247–1255.

Foa, E. B., Huppert, J. D., Leiberg, S., Langner, R., Kichic, R., Hajcak, G., et al. (2002). The obsessive-compulsive inventory: development and validation of a short version. *Psychological Assessment, 14*(4), 485–496.

Ghisi, M., Bottesi, G., Sica, C., Sanavio, E., & Freeston, M. H. (2013). Is performance on the go/nogo task related to not just right experiences in patients with obsessive compulsive disorder? *Cognitive Therapy and Research, 37*(6), 1121–1131. http://dx.doi.org/10.1007/s10608-013-9560-1.

Gibbs, N. A. (1996). Nonclinical populations in research on obsessive-compulsive disorder: a critical review. *Clinical Psychology Review, 16*(8), 729–773. http://dx.doi.org/10.1016/S0272-7358(96)00043-8.

Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: etymology and strategic intentions. *American Journal of Psychiatry, 160*(4), 636–645.

Hajcak, G., Huppert, J. D., Simons, R. F., & Foa, E. B. (2004). Psychometric properties of the OCI-R in a college sample. *Behaviour Research and Therapy, 42*(1), 115–123.

Hajcak, G., & Simons, R. F. (2002). Error-related brain activity in obsessive-compulsive undergraduates. *Psychiatry Research, 110*(1), 63–72.

IBM. (2011). *IBM SPSS statistics for windows (Version 20.0)*. Armonk, NY: IBM Corp.

Kim, M. S., Jang, K. M., & Kim, B. N. (2009). The neuropsychological profile of a subclinical obsessive-compulsive sample. *Journal of the International Neuropsychological Society, 15*(2), 286–290. http://dx.doi.org/10.1017/S1355617709090213.

Krishna, R., Udupa, S., George, C. M., Kumar, K. J., Viswanath, B., Kandavel, T., et al. (2011). Neuropsychological performance in OCD: a study in medication-naive patients. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 35*(8), 1969–1976. http://dx.doi.org/10.1016/j.pnpbp.2011.09.009.

Kuelz, A., Hohagen, F., & Voderholzer, U. (2004). Neuropsychological performance in obsessive-compulsive disorder: a critical review. *Biological Psychology, 65*(3), 185–236.

Lawrence, N. S., Wooderson, S., Mataix-Cols, D., David, R., Speckens, A., & Phillips, M. L. (2006). Decision making and set shifting impairments are associated with distinct symptom dimensions in obsessive-compulsive disorder. *Neuropsychology, 20*(4), 409–419. http://dx.doi.org/10.1037/0894-4105.20.4.409.

Lee, Chiu, C. C., Chiu, C. H., Chang, C. J., & Tang, H. S. (2009). Continuous performance test in drug-naive patients with obsessive-compulsive disorder: a case-controlled study. *Psychiatry Research, 169*(2), 183–185. http://dx.doi.org/10.1016/j.psychres.2008.10.017.

Lee, Yost, B. P., & Telch, M. J. (2009). Differential performance on the go/no-go task as a function of the autogenous-reactive taxonomy of obsessions: findings from a non-treatment seeking sample. *Behaviour Research and Therapy, 47*(4), 294–300. http://dx.doi.org/10.1016/j.brat.2009.01.002.

Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (2012). *Neuropsychological assessment* (5th ed.). Oxford ;New York: Oxford University Press.

Marteau, T. M., & Bekker, H. (1992). The development of a six-item short-form of the state scale of the Spielberger State-Trait Anxiety Inventory (STAI). *British Journal of Clinical Psychology, 31*(Pt 3), 301–306.

Martinot, J. L., Allilaire, J. F., Mazoyer, B. M., Hantouche, E., Huret, J. D., Legaut-Demare, F., et al. (1990). Obsessive-compulsive disorder: a clinical, neuropsychological and positron emission tomography study. *Acta Psychiatrica Scandinavica, 82*(3), 233–242.

Mataix-Cols, D. (2003). Declarative and procedural learning in individuals with subclinical obsessive-compulsive symptoms. *Journal of Clinical and Experimental Neuropsychology, 25*(6), 830–841. http://dx.doi.org/10.1076/jcen.25.6.830.16477.

Mataix-Cols, D., Alonso, P., Pifarre, J., Menchon, J. M., & Vallejo, J. (2002). Neuropsychological performance in medicated vs. unmedicated patients with obsessive-compulsive disorder. *Psychiatry Research, 109*(3), 255–264.

Mataix-Cols, D., Barrios, M., Sanchez-Turet, M., Vallejo, J., & Junque, C. (1999). Reduced design fluency in subclinical obsessive-compulsive subjects. *Journal of Neuropsychiatry and Clinical Neurosciences, 11*(3), 395–397.

Mataix-Cols, D., Junque, C., Sanchez-Turet, M., Vallejo, J., Verger, K., & Barrios, M. (1999). Neuropsychological functioning in a subclinical obsessive-compulsive sample. *Biological Psychiatry, 45*(7), 898–904.

Mataix-Cols, D., Junque, C., Vallejo, J., Sanchez-Turet, M., Verger, K., & Barrios, M. (1997). Hemispheric functional imbalance in a sub-clinical obsessive-compulsive sample assessed by the continuous performance test, identical pairs version. *Psychiatry Research, 72*(2), 115–126.

Mataix-Cols, D., Rahman, Q., Spiller, M., Alonso, M. P., Pifarre, J., Menchon, J. M., et al. (2006). Are there sex differences in neuropsychological functions among patients with obsessive-compulsive disorder? *Applied Neuropsychology, 13*(1), 42–50. http://dx.doi.org/10.1207/s15324826an1301_6.

Menzies, L., Achard, S., Chamberlain, S. R., Fineberg, N., Chen, C. H., del Campo, N., et al. (2007). Neurocognitive endophenotypes of obsessive-compulsive disorder. *Brain, 130*(Pt 12), 3223–3236. http://dx.doi.org/10.1093/brain/awm205.

Milad, M. R., & Rauch, S. L. (2012). Obsessive-compulsive disorder: beyond segregated cortico-striatal pathways. *Trends in Cognitive Sciences, 16*(1), 43–51. http://dx.doi.org/10.1016/j.tics.2011.11.003.

Moritz, S., Hottenrott, B., Jelinek, L., Brooks, A. M., & Scheurich, A. (2012). Effects of obsessive-compulsive symptoms on neuropsychological test performance: complicating an already complicated story. *Clinical Neuropsychologist, 26*(1), 31–44. http://dx.doi.org/10.1080/13854046.2011.639311.

Neurotrax, Corporation. (2003). *Neruotrax computerized cognitive tests*. Bellaire, TX: NeuroTrax Corporation. Retrieved from http://www.neurotrax.com/.

Pauls, D. L., Abramovitch, A., Rauch, S. L., & Geller, D. A. (2014). Obsessive-compulsive disorder: an integrative genetic and neurobiological perspective. *Nature Reviews: Neuroscience, 15*(6), 410–424. http://dx.doi.org/10.1038/nrn3746.

Penades, R., Catalan, R., Rubia, K., Andres, S., Salamero, M., & Gasto, C. (2007). Impaired response inhibition in obsessive compulsive disorder. *European Psychiatry, 22*(6), 404–410.

Purcell, R., Maruff, P., Kyrios, M., & Pantelis, C. (1998). Neuropsychological deficits in obsessive-compulsive disorder: a comparison with unipolar depression, panic disorder, and normal controls. *Archives of General Psychiatry, 55*(5), 415–423.

da Rocha, F. F., Alvarenga, N. B., Malloy-Diniz, L., & Correa, H. (2011). Decision-making impairment in obsessive-compulsive disorder as measured by the Iowa Gambling Task. *Arquivos de Neuro-Psiquiatria, 69*(4), 642–647.

Roth, R. M., Milovan, D., Baribeau, J., & O'Connor, K. (2005). Neuropsychological functioning in early- and late-onset obsessive-compulsive disorder. *Journal of Neuropsychiatry and Clinical Neurosciences, 17*(2), 208–213. http://dx.doi.org/10.1176/appi.neuropsych.17.2.208.

Ruscio, A., Stein, D., Chiu, W., & Kessler, R. (2010). The epidemiology of obsessive-compulsive disorder in the national comorbidity survey replication. *Molecular Psychiatry, 15*(1), 53–63.

Saxena, S., & Rauch, S. L. (2000). Functional neuroimaging and the neuroanatomy of obsessive-compulsive disorder. *Psychiatric Clinics of North America, 23*(3), 563–586.

Schweiger, A., Abramovitch, A., Doniger, G. M., & Simon, E. S. (2007). A clinical construct validity study of a novel computerized battery for the diagnosis of ADHD in young adults. *Journal of Clinical and Experimental Neuropsychology, 29*(1), 100–111.

Simpson, H. B., Rosen, W., Huppert, J. D., Lin, S. H., Foa, E. B., & Liebowitz, M. R. (2006). Are there reliable neuropsychological deficits in obsessive-compulsive disorder? *Journal of Psychiatric Research, 40*(3), 247–257. http://dx.doi.org/10.1016/j.jpsychires.2005.04.004.

Spitznagel, M. B., & Suhr, J. A. (2002). Executive function deficits associated with symptoms of schizotypy and obsessive-compulsive disorder. *Psychiatry Research, 110*(2), 151–163.

Wechsler, D. (2008). *Wechsler adult intelligence scale* (4th ed.). San Antonio, TX: Harcourt Assessment, Inc.

Wright, L., Lipszyc, J., Dupuis, A., Thayapararajah, S. W., & Schachar, R. (2014). Response inhibition and psychopathology: a meta-analysis of go/no-go task performance. *Journal of Abnormal Psychology*. http://dx.doi.org/10.1037/a0036295.