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Comparative neuropsychology of adult obsessive-compulsive disorder and attention deficit/hyperactivity disorder: Implications for a novel executive overload model of OCD

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Research implicates frontostriatal pathophysiology in both attention deficit/hyperactivity disorder (ADHD) and obsessive-compulsive disorder (OCD). Nevertheless, ADHD is characterized with frontostriatal hypoactivity and OCD with hyperactivity. Furthermore, both disorders seem to lie on opposite ends of a clinical impulsive-compulsive continuum. While never having directly been compared, and despite these differences, OCD and ADHD appear to share similar neuropsychological impairments especially in executive functions. This study aimed at comparing adults with OCD and adults with ADHD on neuropsychological measures and behavioural impulsivity and OC measures. Thirty OCD, 30 ADHD, and 30 matched healthy control (HC) participants were administered a comprehensive neuropsychological battery and completed several questionnaires. The groups were compared on all neuropsychological and clinical measures and correlations between neuropsychological and clinical symptoms were computed. The ADHD and OCD groups performed more poorly than HC on all neuropsychological domains and most domain subtests. The ADHD group reported significantly higher impulsivity than the OCD group. OCD patients did not differ from HC on behavioural impulsivity. A unique dissociation was found between impulsivity and response inhibition where both clinical groups showed similar response inhibition deficit, but differed significantly on impulsivity. Moreover, a negative association between OC symptoms and response inhibition and a bias in self-perception of impulsivity was

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found only in the OCD group. We propose an executive overload model of OCD that views neuropsychological impairments in OCD as an epiphenomenon, according to which continuous attempts to control automatic processes are associated with obsessive thoughts overflow that causes an overload on the executive system.

Obsessive-compulsive disorder (OCD) and attention deficit/hyperactivity disorder (ADHD) are common and debilitating neuropsychiatric disorders with a prevalence rate of 2.3% and 2.5–4%, respectively (Kessler *et al.*, 2006; Ruscio, Stein, Chiu, & Kessler, 2010; Simon, Czobor, Balint, Meszaros, & Bitter, 2009). Extensive aetiological research has implicated a major role for heredity in both ADHD (Biederman, 2005; Faraone *et al.*, 2005) and OCD (Nicolini, Arnold, Nestadt, Lanzagorta, & Kennedy, 2009). Nevertheless, the relationship between genotype, endophenotypes (e.g., neurobiological and neuropsychological markers), and clinical manifestations in OCD appears to be more complex and in a sense somewhat paradoxical as compared to ADHD. In the present study, we compared neuropsychological functioning, disorder-specific clinical symptoms, and their interactions in OCD versus ADHD. We begin by reviewing the neurobiological and neuropsychological literature in ADHD and OCD, as well as the association between response inhibition and behavioural impulsivity in the two disorders. We then outline a theoretical model of executive overload in OCD.

Neurobiology of ADHD and OCD

Functional imaging studies consistently implicate deficiencies in frontostriatal circuits and structures (e.g., lateral prefrontal cortex, orbitofrontal cortex [OFC], anterior cingulated cortex [ACC], thalamus, and the basal ganglia) in OCD (Baxter, 1992; Harrison *et al.*, 2009; Maltby, Tolin, Worhunsky, O'Keefe, & Kiehl, 2005; Saxena & Rauch, 2000; Whiteside, Port, & Abramowitz, 2004) and in ADHD (Bush, Valera, & Seidman, 2005; Dickstein, Bannon, Xavier Castellanos, & Milham, 2006; Schneider, Retz, Coogan, Thome, & Rosler, 2006). While frontostriatal deficiencies are implicated in both ADHD and OCD, the nature of the dysfunction is fundamentally different if not antithetical between the two disorders. Functional neuroimaging studies consistently report *hypoactivity* of the frontostriatal network in ADHD in comparison to control participants, and frontostriatal *hyperactivity* in OCD.

Hyperactivation of the OFC, ACC, thalamus, and caudate nucleus in OCD was found in resting state (Baxter *et al.*, 1988; Harrison *et al.*, 2009; Saxena *et al.*, 2001) and in symptom provocation studies (Breiter *et al.*, 1996; Mataix-Cols *et al.*, 2004; Rauch *et al.*, 1994). In contrast, functional imaging studies in ADHD adults examining brain activity during task performance or resting state reveal hypoactivation in the thalamus and prefrontal and striatal regions (Bush *et al.*, 2005; Dickstein *et al.*, 2006). Several functional imaging studies examining frontostriatal functional connectivity found hypoactive functional connectivity in ADHD (Cubillo *et al.*, 2010; Konrad & Eickhoff, 2010; Wolf *et al.*, 2009) and hyperactive connectivity in OCD (Harrison *et al.*, 2009). Finally, most recently, a study directly comparing adolescent diagnosed with ADHD with adolescents diagnosed with OCD revealed opposite frontostriatal brain activity, in which OCD showed hyperactivation and ADHD exhibited underactivity (Rubia, Cubillo, Woolley, Brammer, & Smith, 2011).

Neuropsychology of ADHD and OCD

Naturally, neurobiological processes and neuropsychological test performance are strongly linked. For example, underactivity in, or insult to the prefrontal cortex, results in underperformance in executive functions tests (Stuss, 2007). Accordingly, it is expected that frontostriatal hyperactivity in OCD and hypoactivity in ADHD might be expected to be reflected in different neuropsychological performance of patients with the two disorders. Although no research to date directly compared the neuropsychological performance of ADHD and OCD, it seems that contrary to this rationale, neuropsychological impairments in OCD and ADHD appear to be analogous, especially in the executive functions domain.

Impaired performance in tests of executive functions (e.g., Trail Making B [TMB] test, the Wisconsin Card Sorting Test [Lucey *et al.*, 1997], and the Tower of London test [Strous *et al.*, 2007]) were found in OCD (Aycicegi, Dinn, Harris, & Erkmen, 2003; Lacerda *et al.*, 2003b; Lucey *et al.*, 1997; Penades, Catalan, Andres, Salamero, & Gasto, 2005) and in ADHD (Frazier, Demaree, & Youngstrom, 2004; Johnson *et al.*, 2001; Muller *et al.*, 2007). Impaired performance was also found in working memory and general attention tasks in OCD (Penades *et al.*, 2005) and in ADHD (Frazier *et al.*, 2004). Finally, both disorders are associated with underperformance in tasks that assess response inhibition, one of the principal domains of the executive system. Research indicates impaired performance on two prominent response inhibition tasks: Stroop and Go-NoGo, in OCD (Aycicegi *et al.*, 2003; Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005; Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; Penades *et al.*, 2007) and in ADHD (Frazier *et al.*, 2004; Gallagher & Blader, 2001; Hervey, Epstein, & Curry, 2004). Notably, neuropsychological research examining the memory domain is highly inconsistent in adult ADHD (Hervey *et al.*, 2004; Schoechlin & Engel, 2005). Similarly, some studies reveal memory impairments in OCD (Muller & Roberts, 2005; Segalas *et al.*, 2008), while others suggested that memory impairments in OCD stem from deficient encoding strategies (Greisberg & McKay, 2003; Savage *et al.*, 1999). Finally, it has recently been suggested that there are no memory impairments in OCD (Moritz, Kloss, von Eckstaedt, & Jelinek, 2009).

This review of the literature appears to lead to a puzzling conclusion, namely that OCD and ADHD display similar executive function deficits but opposite underlying neurobiological processes of the frontostriatal system. Moreover, in contrast to the relative consistency of findings in neuropsychological studies of ADHD, neuropsychological studies in OCD are more divergent in term of the types of impairments (Kuelz, Hohagen, & Voderholzer, 2004). In addition, whereas neuropsychological impairments in ADHD may directly reflect frontostriatal underactivity, the relationship between neuropsychological findings and their underlying brain abnormality (i.e., frontostriatal hyperactivation) in OCD has not been elucidated (Aycicegi *et al.*, 2003; Kuelz *et al.*, 2004; Lacerda *et al.*, 2003b).

Response inhibition and impulsivity in ADHD and OCD

In the phenomenological domain, comparison between ADHD and OCD raises particularly interesting questions with regard to behavioural impulsivity. Impulsivity is a highly complex construct (Evenden, 1999), but in general terms, 'The behavioural universe thought to reflect impulsivity encompasses actions that appear poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation and that often result in undesirable consequences' (Daruna & Barnes, 1993). Whereas behavioural impulsivity

is one of the hallmark symptoms of ADHD (American Psychiatric Association, 1994), its relevance to OCD is unclear. Individuals with OCD engage in repetitive rituals (American Psychiatric Association, 1994) that may be considered impulsive when regarded as expressing deficient ability to inhibit ongoing behaviour. Nevertheless, in a review of the OC spectrum disorders across the lifespan, Hollander (2005) argued that impulsivity and compulsivity are phenomenologically different in that compulsive individuals are harm avoidant and risk aversive, whereas impulsive individuals underestimate harm and engage in risky behaviours. Hollander supports his view by pointing out that neurobiologically, impulsivity is associated with OFC underactivity, while in compulsivity, the OFC is hyperactive (Hollander, 2005).

In line with Hollander's assertion, several studies found that individuals with OCD are in fact significantly less impulsive than non-patient controls (Alonso *et al.*, 2008; Shoval, Zalsman, Sher, Apter, & Weizman, 2006; Wu, Clark, & Watson, 2006; Fullana *et al.*, 2004). In non-patients, Li and Chen (2007) found that OC symptoms were negatively correlated with behavioural impulsivity. Moreover, adult individuals with OCD are characterized with a behaviourally inhibited temperamental style (e.g., high levels of restraint, withdrawal, and avoidance of novel stimuli) (Van Ameringen, Mancini, & Oakman, 1998). This temperamental style was also found to be a childhood predictor of OCD symptoms (Muris, Meesters, & Spinder, 2003). This association was also found in a large sample of healthy controls (HCs), where OC symptoms were significantly correlated with retrospective self-report of childhood behavioural inhibition (Coll, Kagan, & Reznick, 1984). Summerfeldt, Hood, Antony, Richter, and Swinson (2004) compared OCD and control participants' scores on the Barratt Impulsiveness Scale (BIS) (Patton, Stanford, & Barratt, 1995) and found that the OCD group had significantly higher overall ratings of impulsivity on the BIS total scale. However, this difference originated *only* from a significant difference in the 'cognitive impulsivity' subscale score, which assesses difficulty in inhibiting thoughts and not behaviour (Summerfeldt *et al.*, 2004). The authors pointed out that the 'cognitive impulsivity' scale represents distractibility and awareness of uncontrollable intrusive activity, a common symptom experienced by individuals with OCD. Summerfeldt *et al.* (2004) note that the lack of difference in other impulsivity domains is consistent with the finding that OCD individuals are harm avoidant and do not display any deficit in planning and organizing behaviour before acting. In sum, clinically, ADHD and OCD seem to be very different disorders, and the hallmark symptoms of behavioural impulsivity in ADHD and OC symptoms in OCD seem to lie at opposite ends of a continuum (Hollander, 2005; Carlsson, 2001).

Response inhibition is frequently defined as the ability to inhibit an already activated motor response (Aron & Poldrack, 2005). Commission errors performed in Go-NoGo tests of response inhibition are defined as 'inappropriate motor response to no-go stimuli' and are considered a common marker for general response inhibition deficit (Chamberlain & Sahakian, 2007). While in contrast to ADHD, OCD is not associated with behavioural impulsivity, both disorders appear to display response inhibition deficit. Specifically, numerous studies suggest that both OCD and ADHD participants make more commission errors and generally perform more poorly than controls on Go-NoGo tests (Aycicegi *et al.*, 2003; Bannon, Gonsalvez, Croft, & Boyce, 2002; Frazier *et al.*, 2004; Penades *et al.*, 2007; Schachar, Mota, Logan, Tannock, & Klim, 2000; Watkins *et al.*, 2005). This finding is intriguing, as response inhibition deficits typically characterize psychiatric disorders associated with behavioural impulsivity (Logan, Schachar, & Tannock, 1997; Mortensen, Rasmussen, & Haberg, 2010; Schachar *et al.*, 2000). In fact, the number of commission errors is considered a strong predictor of behavioural impulsivity within the

realm of neuropsychological assessment (Keilp, Sackeim, & Mann, 2005). This prediction is particularly evident in ADHD, where behavioural impulsivity is considered a hallmark symptom (American Psychiatric Association, 1994; Barkley, 2002; Biederman, 2005), and response inhibition deficit, a prominent ADHD endophenotype (Aron & Poldrack, 2005; Barkley, 1997; Crosbie, Perusse, Barr, & Schachar, 2008; Doyle *et al.*, 2005; Nigg, 2001). The case of OCD, therefore, appears to present an anomaly, as it is associated with impairment in response inhibition but not with behavioural impulsivity.

Neuropsychological impairments and disorder-specific symptomatology

Underactivity or damage to frontal brain regions is associated with behavioural impulsivity, increased risk taking, and neuropsychological impairments, especially in tests of executive functions. These behavioural symptoms and neuropsychological deficits are associated with ADHD as well as with brain damage, which explains why ADHD was formerly called 'Minimal Brain Damage' (Castellanos & Tannock, 2002). In contrast, OCD is associated with frontostriatal *hyperactivity* and the severity of OCD symptoms is positively correlated with increased brain activity (Harrison *et al.*, 2009; Lacerda *et al.*, 2003a, 2003b), as well as with impaired neuropsychological test performance (Lacerda *et al.*, 2003b; Penades *et al.*, 2005; Segalas *et al.*, 2008). Indeed, Rubia *et al.* (2011) recently found opposite brain activity in ADHD and OCD adolescents in which brain activity was positively correlated with obsessive symptoms within the OCD group, and negative correlation between ADHD symptoms and brain activity was found within the ADHD group.

In sum, as presented in Table 1, our review of the literature demonstrates that ADHD and OCD are associated with opposite frontostriatal brain activity, similar neuropsychological impairment (especially in executive functions), and opposite phenomenology.

Table 1. Comparative summary of neurobiological, neuropsychological, and phenomenological characteristics of ADHD and OCD

	ADHD	OCD
Functional neurobiology	Hypoactivitation OFC, BG, ACC, thalamus, DLPFC, and frontostriatal connectivity	Hyperactivation OFC, BG, ACC, thalamus, and frontostriatal connectivity
Neuropsychology	Wide-ranging impairments Executive functions: planning, response inhibition, working memory, set shifting; attention, information processing speed, visuo-spatial working memory	Wide-ranging impairments Executive functions: planning, response inhibition, working memory, set shifting; attention, information processing speed, visuo-spatial working memory
Phenomenology*	Impulsive Difficulty reflecting on consequence of their actions Risk takers Lowered sensitivity to social cues	Inhibited behaviour Excessively concerned with the consequences of their behaviour Harm and risk avoidance Heightened alertness in social situations

OFC = orbitofronal cortex; BG = basal ganglia; ACC = anterior cingulated cortex; DLPFC = dorsolateral prefrontal cortex. *Modified from Carlsson (2001).

The principal aim of the present study was to explore how clinically antithetical disorders, which are characterized with opposite brain activity, can display similar neuropsychological impairments. We hypothesized that this pattern of findings can be explained by assuming that neuropsychological impairments in OCD stem from a unique mechanism that is very different from the one hypothesized for ADHD. Specifically, we propose that the overflow of OC symptoms in OCD causes an overload on the executive system that result in neuropsychological impairments.

Impairments in executive functioning are manifested behaviourally in daily life (e.g., being late to appointments, difficulty concentrating, being forgetful, etc.). We further propose that because OCD is associated with heightened need for control (Moulding & Kyrios, 2006; Reuven-Magril, Dar, & Liberman, 2008), and 'perceived impulsivity' related to fear of causing harm (Cottraux & Gerard, 1998), awareness of 'real life' impairments leads individuals with OCD to perceive themselves as out of control. This biased perception of self-control, which can be termed 'fear of impulsivity', leads to effortful attempts to regain control over behaviour and thoughts and in turn increases the overload on the executive system, resulting in a vicious circle.

In this study, we examine this novel hypothesis by conducting a direct neuropsychological comparison between ADHD and OCD and examining the interactions between each disorder's hallmark symptoms (i.e., OC and impulsiveness) and neuropsychological performance. Based on previous findings, we expected that in comparison to HCs, the ADHD and OCD groups would be impaired on most neuropsychological domains, especially executive functions. We also expected that both ADHD and OCD participants would display response inhibition deficit (e.g., higher number of commission errors) in comparison to the HC participants. We also predicted that the ADHD group would report higher levels of behavioural impulsivity and risk taking than the OCD and HC groups.

Based on our hypothesis that impairments in executive functioning in OCD are associated with overflow of OC symptoms, we predicted that response inhibition impairments would be positively associated with OC symptoms in the OCD group but not in the ADHD or HC group. Finally, based on the hypothesized 'fear of impulsivity' in OCD, we predicted that OCD participants would not report more impulsive behaviours than control participants but would perceive themselves as more behaviourally impulsive.

Method

Participants

Participants were 30 individuals with OCD, 30 individuals with ADHD, and 30 individuals with no psychiatric diagnosis (HC). All participants were male, as the 1:3 to 1:10 female to male ratio of individuals with ADHD (Biederman *et al.*, 2002) makes it very difficult to recruit female participants with ADHD. The initial OCD sample consisted of 37 individuals diagnosed with OCD, who were recruited from an outpatient unit in a large mental health centre in Israel. Inclusion criteria were male gender age range 18–60 and a primary diagnosis of OCD. Participants with a history of any neurological or psychotic disorder, post-traumatic stress disorder, bipolar depression, *Tourette's* syndrome, tic disorder, substance abuse disorder, or DSM-IV axis II disorder were excluded from this study. Diagnoses were reassessed for the present study using the Mini International Neuropsychiatric Interview (MINI) (Sheehan *et al.*, 1997, 1998). Seven OCD participants were excluded from the study based on these criteria (two were in remission and did

not meet formal criteria for diagnosis of OCD, four had psychotic disorders, and one had substance abuse disorder and bipolar depression), resulting in 30 participants in the final OCD group. Thirteen of these were un-medicated, 13 were taking selective serotonin reuptake inhibitors (SSRIs), and four were taking a combination of SSRIs and a low dose of antipsychotic medication. Twenty-four of the OCD patients had additional DSM-IV axis I disorders (dysthymia, social phobia, panic disorder with agoraphobia, generalized anxiety disorder, and depression), whereas six were diagnosed exclusively with OCD.

The ADHD group comprised 30 male college students diagnosed exclusively with ADHD who enrolled in a support centre for students with learning disabilities and ADHD at a college in northern Israel and volunteered to participate in this study. This sample was matched to the OCD and HC groups on age and education. In order to rule out other comorbid psychiatric disorders, participants in the ADHD group were screened using the MINI, a DSM-IV-based ADHD questionnaire, and available records. Exclusion criteria for this group were any other DSM-IV psychiatric disorders, any history of prescribed psychiatric medications or hospitalization, any history of a neurological disorder or insult, and any known or reported history of other learning disabilities. The control group comprised 30 males matched for age and education with the ADHD and OCD participants. In order to follow the strict procedure of matching established between the groups, participants in the control group were recruited from the community by the research assistants primarily by soliciting friends and relatives of the their peers. All participants in the control group were free from past or present learning disability and any neurological, developmental, or psychiatric condition, as verified with the MINI. This study was approved by the mental health centre and the university's Institutional Review Boards (IRBs). All participants signed an informed consent in accordance with the Declaration of Helsinki.

Procedure

Participants signed an informed consent form following a general explanation regarding the procedure. All participants completed a short personal information sheet and were administered the MINI semi-structured interview. Screening procedure was closely supervised by a licensed neuropsychologist (the first author). In order to control for the potential confounding effect of depression, the OCD group was also administered the Beck Depression Inventory II (BDI-II). Following the interview, all participants took the Mindstreams computerized neuropsychological battery (Neurotrax Corporation, 2003), using the same laptop computer. Subsequently, all participants completed the ADHD DSM-IV-based questionnaire, the Eysenck Impulsivity scale, and the Obsessive-Compulsive Inventory-Revised (OCI-R).

Measures

The Hebrew version of the MINI version 5.0.0 (Sheehan *et al.*, 1997, 1998) was used for the diagnosis of OCD and for the screening of comorbidities for both groups. The MINI is a well-validated brief structured psychiatric diagnostic instrument that was found to have good psychometric properties (Sheehan *et al.*, 1997).

The OCI-R (Hajcak, Huppert, Simons, & Foa, 2004) is an empirically derived 18-item self-report measure that was specifically developed to assess severity of OCD symptoms. Each item is rated on a 5-point Likert scale for the degree of experienced distress. The OCI-R comprises six subscales, three items each assessing OCD symptom dimensions (washing, checking, ordering, obsessing, hoarding, and neutralizing). The OCI-R has

been shown to have strong psychometric properties in both clinical (Foa *et al.*, 2002) and non-clinical samples (Hajcak *et al.*, 2004). The OCI-R is sensitive but consistent and was found to be an excellent outcome measures in clinical studies (Abramowitz, Tolin, & Diefenbach, 2005).

A *DSM-IV-based self-report questionnaire* was developed in Hebrew to cover all the necessary criteria for ADHD diagnosis. Because the DSM-IV criteria require that some of the current ADHD symptoms should have been evident before the age of 7, participants are asked to indicate whether they exhibited each behaviour in the past 6 months and/or in childhood (before the age of 7). Notably, research suggests that DSM-IV-based self-report questionnaires in adult ADHD population have sound psychometric properties and do not differ from other ADHD rating scales (Mccann & Roy-Byrne, 2004).

BDI-II (Beck, Steer, & Brown, 1996). The BDI-II is a gold standard self-report measure for severity of depressive symptoms. The BDI-II comprised 21 groups of statements. Participants are asked to choose the statement that best describes the way they felt in the past 2 weeks. The BDI-II has strong psychometric properties and was found to have excellent internal consistency in clinical and non-clinical populations (Cronbach's α = *r's* .92–.93) (Beck *et al.*, 1996).

Eysenck Impulsiveness Venturesomeness empathy scale (IVE) (Eysenck & Eysenck, 1978) is a 63-item questionnaire designed to assess impulsivity. This questionnaire, which was translated into Hebrew, measures impulsivity, venturesomeness, and empathy and is considered one of the most commonly used self-report questionnaires for the assessment of impulsivity (Parker & Bagby, 1997). The items from the empathy scale were originally added in order to be used as buffer items (Eysenck & Eysenck, 1978). For the purpose of the present study, interest was focused only on the impulsivity and venturesomeness scales. The questionnaire is in a 'yes – no' format and has reported reliability coefficients of .79 and .85 for venturesomeness and impulsivity, respectively (Eysenck & Eysenck, 1978).

Mindstreams computerized neuropsychological battery. The Mindstreams is a comprehensive computerized neuropsychological battery. A detailed treatment of the Mindstreams battery is reported elsewhere (Dwolatzky *et al.*, 2003; Schweiger, Abramovitch, Doniger, & Simon, 2007). In brief, the Mindstreams is a relatively short (approximately 45 min), comprehensive, and well-validated computerized neuropsychological battery that samples a wide range of cognitive domains, including verbal memory, non-verbal memory, executive function, visual-spatial orientation, information processing speed, and motor skills. The battery has strong reliability and construct validity in accurately identifying cognitive deficits in psychiatric (Paleacu *et al.*, 2007; Ritsner, Blumenkrantz, Dubinsky, & Dwolatzky, 2006; Schweiger *et al.*, 2007; Strous *et al.*, 2007) and neurological disorders (Doniger *et al.*, 2006; Dwolatzky *et al.*, 2003; Hausdorff *et al.*, 2006). More than 100 reports using the Mindstreams battery in research have been published to date (Neurotrax Corporation, 2003). In the current study, we used the Hebrew version of the battery that utilizes Hebrew normative data to produce scaled score. More than 40 reports using the Mindstreams' Hebrew version in research have been published (Neurotrax Corporation, 2003).

The battery provides raw data, and scaled scores (i.e., similar to Wechsler IQ scales, with $M = 100$ and $SD = 15$) that are produced comparing each individual's performance to an age and education matched control group. Where relevant, the battery also provides two types of performance indexes. Every test that involves speed and accuracy provides an index score that combines the two ([accuracy/reaction time] $\times$ 100). Finally, the battery provides index scores for each cognitive construct (memory, attention, executive

functions, psychomotor skills, visuo-spatial, verbal functions, information processing speed, and a global performance index score), which is derived from similar elements across different tests. The following nine Mindstreams tests were used in this study:

Expanded Go-NoGo test. In this timed Go-NoGo continuous performance test that measures executive functions (response inhibition) and sustained attention, participants are asked to respond as quickly as possible to a series of any coloured stimuli (squares), except red. The stimuli are presented at a pseudo-random interval, but in fact there are four blocks (Base Line, Shorter intervals between stimuli, More 'No Go' stimuli, and Distracters present) presented consecutively. The Mindstreams' Go-NoGo test was found to have strong construct validity with the Conners' CPT II (Conners, 2000) (e.g., commission errors $r = .79$, mean response time $r = .72$) (Schweiger *et al.*, 2007). Good internal consistency for the Mindstreams Executive Function domain was found across HC and clinical populations (*r's* .65–.97) (Doniger, 2011).

Mindstreams' Stroop test. This computerized version of the well-known Stroop test (Stroop, 1935) assessing executive functions and attention comprises three phases. In the first phase, the participant is asked to choose the colour of the letters of general words. In the second phase (the choice reaction time [CRT] phase), the participants are asked select the colour named by a word in white letter-colour. In the third and final phase (the Stroop phase), the participant is presented with words that name colours, but the word is coloured with a different colour (e.g., the word Red in blue font). The Mindstream's Stroop test has satisfactory psychometric properties in clinical populations (e.g., correlations with TMB, *r*'s .56–.62 (Doniger, 2011]).

Staged Information Processing (SIP) test. This test, which assesses attention and information processing speed, comprises three levels of information processing load, in which single digits, two digit arithmetic problems (e.g., $9 - 3$), or three digit arithmetic problems (e.g., $7 - 2 + 1$) are presented. For each of the three levels, the stimuli are presented in three fixed rates that increase as the tests progresses. Participants are asked to press as fast as possible the left mouse key if the result is equal or less than 4, or the right mouse button is the result is greater than 4. The test has strong psychometric properties and was found to be significantly correlated with several well-known pencil–paper tests in clinical and non-clinical cohorts (e.g., Wechsler Digit Span and Digit Symbol, Trail Making A (TMA), and TMB, *r*'s .52–.77) (Doniger, 2011). The Staged information Processing test was found to have good alternate form reliability *(r*'s .73–.85) (Melton, 2005).

Finger Tapping. Participants are asked to press the mouse key repeatedly as fast as they can for 12 s (while an empty bar is coloured). This test examines hand–eye coordination and psychomotor functions and it is repeated twice. The test was found to have good construct validity against the TMA and TMB test (*r*'s .60–.62) and has good alternate form reliability ($r = .80$) (Doniger, 2011).

Catch Game. This test incorporates and assesses reaction time (RT), hand–eye coordination, and planning. Participants are required to 'catch' a 'falling' object (a rectangle) by moving a 'paddle' horizontally using the right and left mouse keys. The 'falling' rectangle increases its speed on every consecutive trial. The test has strong psychometric properties and was found to be significantly correlated with several well-known pencil–paper tests in clinical and non-clinical cohorts (e.g., Wechsler Block Design, TMB, and tower of Hanoi, *r*'s .51–.70) (Doniger, 2011).

Visual Spatial Imagery. Computer-generated three-dimensional scenes are shown in which a red pillar is located differently in every scene. Participants are asked to select one of four scenes that correspond to the correct vantage point of the pillar. This test,

assessing visuo-spatial orientation, was found to be correlated with the copy condition on the Rey Osterreith Complex Figure Test, the Wechsler IQ test picture completion, and the WMS-III Spatial Span test (*r*'s .50–.57) and has good internal consistency (Cronbach's $\alpha = .68–.80$) (Doniger, 2011).

Non-Verbal Memory. Eight images are presented (e.g., a key pointing upwards) followed by a recognition phase in which each of the initially presented images is presented together with the same image in three different orientations (the same key pointing up, left, diagonally or right). Four consecutive recognition trials are presented during the learning phase. After a 10-min filled delay, participants are presented with an additional recognition phase. The Mindstreams' Non-Verbal Memory has strong psychometric properties, including good reliability (Cronbach's $\alpha = .65–.71$) and was found to correlate with several subtests of the WMS-III, the Rey Auditory Verbal Learning Test, and the Rey Osterreith Complex Figure Test in cohorts of clinical and healthy participants (*r*'s .52–.77) (Doniger, 2011).

Verbal Functions. Participants are presented with pictures of items. Following each presentation, participants are presented with four words and are requested to select the word that rhymes with the name of the object in the picture. This test was found to have strong internal consistency (Cronbach's $\alpha = .91–.96$) and is highly correlated with the Boston Naming Test, the Controlled Oral Word Association FAS test, Boston Naming Test, WMS-III Logical Memory I, Verbal and Category Fluency, WAIS-III Verbal IQ, and the WAIS-III Verbal Comprehension Index (*r*'s .52–.93) (Doniger, 2011).

Statistical analysis

In order to examine differences between the groups on neuropsychological domain scores, we conducted eight univariate analyses of variance (ANOVA). To avoid inflation of type I error, we used a significance level of .00625 (.05 divided the by eight tests) for each test. We used the same approach in examining differences in neuropsychological performance on individual subtests comprising the domain index scores, dividing the .05 significance level by the number of subtests comprising each domain. We conducted Pearson's zero-order correlation analyses to examine associations between OC symptom severity and neuropsychological performance within the OCD group. We did not employ alpha correction for correlation analyses in line with recent arguments that alpha corrections might inflate type II errors, particularly in the context of correlation analyses in which significance level is highly dependent on the number of participants (O'Keefe, 2003; Weber, 2007). Cohen's *d* effect sizes were also computed. In order to examine difference in proportions of dichotomic responses to a specific questionnaire item, we used chi-squared crosstabs analyses. Finally, in order to compare correlation coefficients between the groups, we used Fisher's *z* transformation.

Results

The univariate ANOVA revealed no significant difference between the groups on age and education (Table 2). As shown in Table 2, the groups differed significantly on the IVE impulsiveness scale ($p < .001$). Least significant difference (LSD) pairwise contrasts revealed that the ADHD group scored significantly higher on the impulsivity scale than the control and the OCD groups ($p < .001$), with no significant difference between the OCD and the HC groups ($p = .41$). Groups also differed on the IVE risk-taking scale ($p <$

Table 2. Demographic and clinical characteristics of the OCD, ADHD, and control groups

	OCD		ADHD		HC			
	(*N* = 30)		(*N* = 30)		(*N* = 30)			
	Mean	*SD*	Mean	*SD*	Mean	*SD*	*F*(2, 87)	Sig.
Age	31.95	7.86	29.54	6.80	30.19	6.36	0.942	N.S.
Education (years)	13.17	1.84	13.50	1.31	13.97	1.65	1.860	N.S.
IVE impulsiveness	8.50[a]	4.13	15.00[a]	3.63	7.63[a]	4.64	29.212	<.0001
IVE risk taking	7.27[b]	4.06	13.57[b]	3.10	10.87[b]	2.69	25.798	<.0001
OCI-R total score	32.90[c]	11.96	21.15[c]	9.10	8.30[c]	5.62	52.790	<.0001
Age of onset	18.96	6.80						
BDI-II	13.26	8.66						

IVE = Eysenck's Impulsiveness Venturesomeness Empathy scale; OCI-R = Obsessive-Compulsive Rating Scale Revised; BDI-II = Beck Depression Inventory 2nd ed. [a]LSD pairwise contrasts, ADHD > HC = OCD, $p < .0001$. [b]LSD pairwise contrasts ADHD > HC, $p < .01$; ADHD > OCD, HC > OCD $p < .0001$. [c] LSD pairwise contrasts OCD > ADHD, OCD > HC, ADHD > HC, $p < .0001$.

.001). LSD analyses revealed that the ADHD group scored significantly higher than the control ($p = .003$) and the OCD groups ($p < .001$). The control group scored significantly higher than the OCD group on risk taking ($p < .001$). As presented in Table 2, significant differences between the groups were also found on the OCI-R total score ($p < .001$). LSD analyses revealed that the OCD group scored significantly higher than the HC ($p < .001$) and the ADHD groups ($p < .001$). The ADHD group scored significantly higher than the control group on the OCI-R total score ($p < .001$). Notably, the BDI-II scores (presented in Table 2) represent minimal severity, which is accounted for by the small number of participants in the OCD group who had a comorbid diagnosis of depression.

In order to assess the potential impact of comorbid disorders on neuropsychological test performance, we compared OCD participants with and without comorbid conditions using univariate ANOVA. There was no significant difference on any of the domain indexes between participants with and without depression, dysthymia, social phobia, panic disorder, and generalized anxiety disorder. Similarly, medicated and un-medicated participants within the OCD group did not differ significantly on any neuropsychological domain index. Finally, there were no significant correlations between age of onset and neuropsychological test performance within the OCD group (r's between −.195 and .205).

Neuropsychological domain indexes

Eight separate ANOVA's were used to examine group differences on the domain indexes (alpha = .00625). Analyses revealed a significant difference ($p < .001$) between the groups on all domain indexes (Table 3). As shown in Table 3, the largest differences between the ADHD and HC participants and between the OCD and HC participants were found on the Composite Performance Index (Cohen $d = 1.85$ and 1.92, respectively) and the Executive Functions Index (Cohen $d = 1.99$ and 1.39, respectively). LSD contrasts revealed that the OCD group performed more poorly than the control group on all domain indexes ($p < .001$). The OCD and ADHD groups differed significantly only on the Attention, Motor Skills, and Memory indexes (all p's < .01). The OCD group

Table 3. Neuropsychological domain indexes

	OCD		ADHD		HC			LSD pairwise contrasts			Cohen's *d* effect size		
	(*n* = 30)		(*n* = 30)		(*n* = 30)								
Domain index [a]	Mean	*SD*	Mean	*SD*	Mean	*SD*	*F* (2, 87)	OCD versus HC	OCD versus ADHD	ADHD versus HC	OCD versus HC	OCD versus ADHD	ADHD versus HC
Composite performance index	86.73	11.58	90.49	8.78	104.68	6.36	33.757***	***	N.S.	***	1.92	-	1.85
Executive functions	87.9	14.86	83.86	11.40	104.38	9.10	25.541***	***	N.S.	***	1.39	-	1.99
Attention	86.25	16.93	96.25	9.80	104.89	8.09	17.477***	***	**	**	1.40	0.72	0.96
Information processing speed	85.72	16.13	91.25	21.12	104.18	11.28	9.497***	***	N.S.	**	1.33	-	0.76
Motor skills	94.25	11.74	85.11	18.27	106.86	7.58	20.230***	***	**	***	1.28	0.59	1.56
Verbal function	82.29	27.44	92.02	21.08	106.29	9.60	10.174***	***	N.S.	**	1.17	-	0.87
Memory	84.38	19.56	95.21	15.65	100.53	9.58	8.471***	***	**	N.S.	1.05	-	0.61
Visuo-spatial	88.11	21.04	87.99	12.81	105.60	12.90	11.953***	***	N.S.	***	1.00	-	1.37

[a] Domain index scores are normalized on a Wechsler IQ scale (Mean = 100, *SD* = 15). HC = healthy controls, $^{*}p < .05$; $^{**}p < .01$; $^{***}p < .0001$.

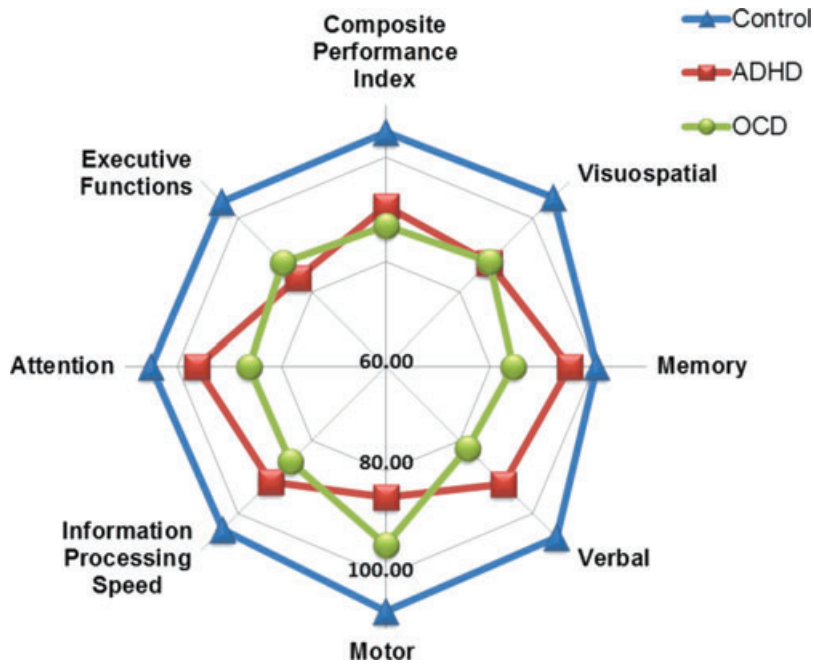

Figure 1. Neuropsychological performance across domains. A comparative graphic illustration of the seven Mindstreams neuropsychological battery domain index scaled scores (similar to the Wechsler IQ scale with $M = 100$, $SD = 15$), and the Composite Performance neuropsychological index scaled score, between the ADHD, OCD, and control groups.

performed more poorly than the ADHD group on the Attention and Memory domain indexes but better on the Motor Skills domain. Apart from the Memory domain, the ADHD group performed more poorly than the HC group on all domains (significance levels between $p < .01$ and $p < .001$). Graphic illustration of the neuropsychological profiles of the three groups is presented in Figure 1.

Neuropsychological domain index subtests

Executive functions

The composite score of the Mindstreams' Go-NoGo, Stroop, and Catch Game subtests was analysed using separate ANOVA's (Table 4, alpha = .0167). An overall significant difference was found on the Go-NoGo and Stroop composite scores ($p < .001$). LSD analyses revealed that the ADHD and OCD groups scored significantly lower than the HC group on these subscales ($p < .001$). Groups did not differ on the Catch Total score. No significant differences were found between the OCD and ADHD groups on any of the three measures comprising the Executive Functions domain.

Attention

The five subtest scores comprising the Attention domain index were analysed using separate ANOVA's (Table 4, alpha = .0083). Groups differed significantly on the Go-NoGo RT ($p = .001$). The Stroop CRT ($p = .001$) and on the SIP RT on the low-load

Table 4. Neuropsychological domain subtests raw scores

	OCD ($n = 30$)		ADHD ($n = 30$)		HC ($n = 30$)			LSD pairwise contrasts			Cohen's d effect size		
	Mean	*SD*	Mean	*SD*	Mean	*SD*	$F(2, 87)$	OCD versus HC	OCD versus ADHD	ADHD versus HC	OCD versus HC	OCD versus ADHD	ADHD versus HC
Executive functions													
Go-NoGo composite score[a]	19.73	5.32	21.00	3.80	25.14	2.50	13.677***	***	N.S.	***	1.30	–	1.29
Stroop composite score	17.93	8.75	17.85	8.36	25.85	6.71	9.825***	***	N.S.	***	1.02	–	1.05
Catch game total score	849.73	141.37	848.27	166.80	938.30	84.03	4.361* ±	*	N.S.	*	0.76	–	0.68
Attention													
Go-NoGo RT (ms)	488.59	114.18	491.89	121.45	389.44	39.50	7.473**	**	N.S.	**	1.16	–	1.13
Go-NoGo RT *SD* (ms)	130.77	119.62	109.62	60.73	68.52	18.88	4.598* ±	**	N.S.	N.S.	0.73	–	–
Stroop RT level 2 (ms)	519.73	168.98	565.79	259.91	388.97	87.49	7.299**	**	N.S.	***	0.97	–	0.91
Staged info RT 1.2 (ms)	618.65	146.75	629.29	144.59	474.40	71.72	14.066***	***	N.S.	***	1.25	–	1.36
Staged info accuracy 2.3 (%)	73.57	24.98	70.36	22.85	85.00	13.83	3.940* ±	*	N.S.	**	0.57	–	0.77
Information processing speed													
Staged info composite score 1.1	14.89	3.93	14.78	3.53	18.66	2.51	12.711***	***	N.S.	***	1.14	–	1.27
Staged info composite score 1.3	15.74	3.91	16.51	3.54	20.50	2.89	16.097***	***	N.S.	***	1.38	–	1.23
Staged info composite score 2.1	7.98	2.18	7.79	1.92	10.23	1.77	14.001***	***	N.S.	***	1.13	–	1.32
Staged info composite score 2.2	10.47	2.65	9.73	2.07	13.09	2.23	16.783***	***	N.S.	***	1.07	–	1.56

Table 4. Continued

	OCD ($n = 30$)		ADHD ($n = 30$)		HC ($n = 30$)			LSD pairwise contrasts			Cohen's *d* effect size		
	Mean	*SD*	Mean	*SD*	Mean	*SD*	$F(2, 87)$	OCD versus HC	OCD versus ADHD	ADHD versus HC	OCD versus HC	OCD versus ADHD	ADHD versus HC
Motor skills													
Finger tap inter-tap interval (ms)	195.79	45.56	171.73	29.29	171.43	25.28	4.853**	**	**	N.S.	0.66	0.63	–
Finger tap inter-tap interval *SD* (ms)	39.76	13.71	43.53	19.67	24.00	14.95	12.027***	***	N.S.	***	1.10	–	1.12
Catch game time to first move (ms)	462.50	136.00	493.53	85.95	386.77	65.11	9.012***	**	N.S.	***	0.71	–	1.72
Verbal functions													
Rhyming accuracy (%)	82.12	19.73	89.66	12.65	95.10	8.29	6.619**	*	*	N.S.	0.86	0.45	–
Memory													
Non-verbal memory accuracy(%)	72.50	22.25	83.28	13.14	91.90	6.74	11.882***	***	**	*	1.18	0.59	0.82
Delayed non-verbal memory accuracy(%)	76.97	27.48	89.43	13.43	95.53	8.83	7.852**	***	*	N.S.	0.91	0.58	–
Visuospatial													
Visuo-spatial accuracy (%)	62.90	24.24	75.43	15.18	85.77	13.43	11.730***	***	*	*	1.17	0.62	0.7

*$p < .05$; **$p < .01$; ***$p < .0001$. [a] All composite scores are calculated: ([accuracy/reaction time] × 100). HC = healthy controls. ms = milliseconds; LSD = least significant difference; RT = response time; ± not significant after alpha correction for multiple univariate analyses (0.05/the number of domain subtests). Stroop level 2 = choice reaction time, selecting colour named by a word in white letter-colour. Staged info. 1.1 = staged information processing task low load low speed, 1.2 = low load medium speed, 2.1 = medium load low speed, 2.2 = medium load medium speed.

phase ($p < .001$). LSD analysis revealed that in comparison to the HC group, the OCD group performed more poorly on the Go-NoGo RT ($p < .001$), the Stroop CRT ($p < .008$) and had longer RT in the low-load phase on the SIP test ($p < .001$). Similarly, the ADHD group performed more poorly than the HC group on the Go-NoGo RT ($p < .007$) and the Stroop phase ($p <. 001$) and had longer RT in the low-load phase of the SIP test ($p < .001$). No significant differences were found between the ADHD and the OCD groups on any of the Attention domain measures.

Information processing speed

The composite scores ([accuracy/reaction time] $\times$ 100) of the four phases comprising the SIP Speed domain index were analysed using separate ANOVA's (alpha = .0125). As shown in Table 4, significant difference was found between the groups on all four subscales ($p < .001$). Both the OCD and the ADHD groups performed significantly more poorly than HC on all subscales ($p < .001$), with no significant differences between the ADHD and OCD on all subtests.

Motor skills

The finger tap inter-tap interval (FTI), the inter-tap interval standard deviation (FTISD) from the Finger-Tapping test, and the 'time to first move' from the 'Catch Game' were analysed using separate ANOVA's (alpha = .0167). Significant difference was found between the groups on the FTI ($p = .01$) and the FTISD ($p < .001$) as well as in the 'Catch' subtest ($p < .001$). LSD analyses revealed that the OCD group performed significantly more poorly than the HC group on all the three measures ($p = .008$, $p < .001$, and $p = .004$, respectively). The ADHD group performed significantly more poorly than the HC group on the 'Catch' subtest and the FTISD ($p < .001$). The OCD group performed significantly more poorly than the ADHD group on FTI ($p = .009$) but did not differ on the other two measures in the Motor Skills domain.

Verbal functions

Results of the ANOVA performed on the rhyming accuracy measure revealed an overall significant difference ($p = .003$). LSD analysis revealed that the OCD group performed significantly more poorly than the HC ($p = .001$) and the ADHD ($p = .047$) groups. The ADHD group's performance did not significantly differ from the HC group on this measure.

Memory

The non-verbal memory accuracy and the delayed non-verbal memory accuracy were analysed using separate ANOVA's (alpha = .025). Overall significant differences were found on the total non-verbal memory accuracy ($p < .001$) and the delayed non-verbal memory accuracy ($p = .001$). The OCD group performed significantly more poorly on the total non-verbal memory measure than HC ($p < .001$) and ADHD ($p = .009$) groups. The ADHD group performed significantly more poorly than HC on this measure ($p = .035$). The OCD group performed significantly more poorly than HC ($p < .001$) and the ADHD groups ($p = .012$) on the delayed non-verbal memory accuracy. No significant difference was found between the ADHD and HC groups on this measure.

Table 5. Pearson's correlations between the OCI-R, and neuropsychological scaled domain indexes, impulsivity ratings, and number of commission errors

	OCD	ADHD	Fisher's *z*	Sig.
Composite performance index	−.17	.16	1.22	.055
Executive functions	−.29	.33	2.34	**.005**
Attention	−.03	.24	0.99	.079
Information processing speed	.09	−.27	1.34	**.045**
Motor skills	−.03	.32	1.35	**.044**
Verbal function	−.19	−.10	0.31	.189
Memory	−.06	−.24	0.70	.121
Visuo-spatial	−.19	.17	1.34	**.045**
Impulsivity	**.38**[*]	.01	1.43	**.038**
Commission Errors[!]	**.40**[*]	.22	0.76	.111

[*] $p < .05$; OCI-R = Obsessive-Compulsive Inventory-Revised. [!]Commission errors = raw number of commission errors on the Go-NoGo test.

Visual-spatial orientation

A univariate ANOVA was performed between the groups on the visuo-spatial accuracy measure. The groups significantly differed on this measure ($p < .001$). The OCD group performed significantly more poorly than the HC ($p < .001$) and the ADHD ($p = .011$) groups. The ADHD group performed significantly more poorly than the HC group on this measure ($p = .035$).

Clinical symptoms and neuropsychological performance

In order to compare the association between OC symptoms and performance on neuropsychological domains, we correlated the OCI-R total score with the scaled score on each on the eight neuropsychological domains within each of the three groups. As presented in Table 5, while none of the correlations between the OCI-R and the neuropsychological domains in either group were significant, there was a consistent pattern of negative correlations between neuropsychological domains and OCI-R total score within the OCD group and a non-significant trend for positive correlations within the ADHD group. We examined the differences between the correlation coefficients in the OCD and the ADHD group using Fisher *z* transformation. Significant differences were found between the OCD and ADHD groups on correlations between the OCI-R and the Executive Functions, Motor Skills, and Visuo-spatial domains (Table 5). No significant correlations were found between impulsivity ratings and neuropsychological domain index scores, either in the ADHD group (r's between −.204 and .208) or in the OCD group (r's between −.312 and .203). Most of the correlation coefficients were near zero with no discernible pattern.

Response inhibition, impulsivity, and OC symptoms

A univariate ANOVA was conducted in order to compare the overall number of commission errors between the groups. A significant overall difference was found between the ADHD ($M = 8.09$, $SD = 7.54$), the OCD ($M = 8.10$, $SD = 9.69$), and the HC group ($M = 3.75$, $SD = 2.15$) on the total number of commission errors on the Go-NoGo test ($F(2, 87) = 3.418$, $p = .038$). LSD analysis revealed that both the ADHD

($p = .026$) and the OCD groups ($p = .027$) made significantly more commission errors than HC. No significant difference was found between the OCD and ADHD groups ($p = .99$), which made nearly identical number of commission errors. Significant positive correlation between commission errors and the OCI-R total score was found only within the OCD group ($r = .40$, $p = .027$). Similarly, the IVE impulsiveness scale and the OCI-R total score were significantly positively correlated only within the OCD group ($r = .38$, $p = .04$). Subsequent z test revealed that the association between impulsivity and the OCI-R total scores significantly differed between the ADHD and OCD groups (Table 5, $p = .038$).

In order to examine the differences in self-perception of behavioural impulsivity between the groups, we analysed the percentage of 'YES' responses on the IVE item 'Are you an impulsive person?' using chi-square analyses. There were significant differences between the three groups, with 17% of the HC participants, 50% of the OCD, and 80% of the ADHD groups marking 'YES' on this item (χ^2 (2) = 24.101, $p < .001$).

Discussion

The aim of the present study was to directly compare the neuropsychological performance and the hallmark symptoms of ADHD and OCD, while controlling for potential confounding factors such as age, education, comorbid conditions, and medications. To our knowledge, no research to date conducted a direct neuropsychological and symptomatological comparison between ADHD and OCD.

In line with our hypothesis, domain analysis revealed that ADHD and OCD participants showed comparable and wide-ranging neuropsychological impairments on the Composite Performance, Executive Functions, Attention, Information Processing Speed, Motor Skills, Verbal Functions, Memory, and Visuo-spatial domain indexes. These results remained significant after employing alpha correction. Compared to the HC group, neuropsychological impairments in OCD were robust on all domains, with Cohen's *d* effect sizes between 1.0 and 1.92. The ADHD group showed more variability in effect sizes (Cohen's *d* 0.76–1.99*)* but apart from lack of difference from the control participants on the memory domain, the ADHD group showed robust effect sizes and wide-ranging impairments. Our pairwise analyses revealed that the OCD group performed more poorly than the ADHD group in the Attention and Memory domains, while performing better in the motor domain. Nevertheless, in accordance with our hypothesis, performance in all other domains did not differentiate between the OCD and ADHD participants.

In a secondary analysis (Table 4), groups were compared on individual test scores comprising the domain indexes. Similar to the first analysis, the OCD group performed more poorly than HC on all measures. The most robust differences were evident in measures of executive functions, information processing speed, attention, and visuo-spatial abilities. These findings are in accord with the majority of neuropsychological studies on OCD (Bucci *et al.*, 2007; Greisberg & McKay, 2003; Lacerda *et al.*, 2003b; Muller & Roberts, 2005; Penades *et al.*, 2005; Rao, Reddy, Kumar, Kandavel, & Chandrashekar, 2008; Savage *et al.*, 2000; Segalas *et al.*, 2008). Similarly, the ADHD group was impaired on most of the measures (14 out of 19) as compared to the HC group, especially in measures of executive functions, information processing speed, and attention. These findings are in line with the bulk of the literature on the neuropsychology of ADHD (Biederman *et al.*, 2009; Frazier *et al.*, 2004; Hervey *et al.*, 2004). Overall,

neuropsychological profiles in OCD and ADHD were very similar, especially on subtests within the Executive Functions, Attention, and Information Processing Speed domains. Furthermore, in accordance with our hypothesis and previous literature, both the ADHD and OCD groups showed significant and equal response inhibition deficit, expressed as significantly more commission errors than the HC group on the Go-NoGo test.

Whereas our results confirm that both ADHD and OCD are associated with similar wide-ranging cognitive impairments, we found significant symptomatological differences. In line with our hypothesis, we found that ADHD participants were significantly more behaviourally impulsive than OCD participants who did not differ from HCs on this measure. This finding is in agreement with several studies reporting a lesser or equivalent degree of impulsivity in OCD in comparison to HC (Alonso *et al.*, 2008; Chamberlain *et al.*, 2006; Shoval *et al.*, 2006; Fullana *et al.*, 2004; Wu *et al.*, 2006). In addition, we found that the OCD group reported significantly less risk-taking behaviours than the ADHD and the HC group.

In the realm of neuropsychological assessment, a high number of commission errors on a Go-NoGo test are considered an indicator of both response inhibition deficit and behavioural impulsivity (Chamberlain & Sahakian, 2007; Keilp *et al.*, 2005; Logan *et al.*, 1997; Spinella, 2004). Whereas the ADHD and OCD groups showed comparable response inhibition deficit, our results suggest that individuals with OCD are not behaviourally impulsive or risk takers. Thus, these results reveal a dissociation between behavioural impulsivity and response inhibition that seems to be specific to OCD. Moreover, OC symptom severity was significantly correlated with the number of commission errors only within the OCD group. This association between OC symptom severity and response inhibition, together with the lack of association between the latter and behavioural impulsivity in OCD, give rise to the possibility that the degree of response inhibition deficit in OCD is affected by OC symptoms and does not necessarily indicate an inherent response inhibition deficit (in terms of deficient ability to suppress already activated motor responses).

Whereas it is possible that impaired response inhibition may be a primary deficit in OCD, the lack of corresponding behavioural expression of this impairment (e.g., impaired control over behaviour) calls this assumption into question. However, in line with our model, it is conceivable that individuals with OCD are characterized with a primary impairment in response inhibition that manifests itself exclusively in the cognitive domain (e.g., Cognitive Impulsivity, viewed as impaired control over thoughts) (Summerfeld *et al.*, 2004). Awareness of this impairment may cause individuals with OCD to feel compelled to deter potential subsequent disinhibited behaviour, which may result in an increase in OC symptoms. This hypothetical process is consistent with the robust finding of 'thought-action fusion' in OCD (Shafran & Rachman, 2004), defined as the tendency to treat mental states as similar to behaviour in terms of physical or moral consequences.

Beyond the association with the number of commission errors, we did not find significant correlations between the OCI-R and other neuropsychological impairments. Nevertheless, we did find a consistent pattern of negative associations between the OCI-R and neuropsychological performance within the OCD group that was significantly different from the positive trend found between these measures in ADHD. In our view, these differences support our hypothesis that neuropsychological impairments in OCD and ADHD express different mechanisms as follows.

In order to examine our prediction that OCD participants would be biased towards perceiving themselves as impulsive, we compared the proportion of participants who

replied positively to the question: 'Are you an impulsive person?', in the three groups. In both the ADHD and HC groups, responses to this question were consistent with reported behavioural impulsivity. Specifically, 80% of the ADHD participants and 17% of the HC participants responded affirmatively to this item. In contrast, while the OCD group did not differ from the HC group on self-reported behavioural impulsivity, 50% of the participants in the OCD group responded affirmatively to this item, suggesting that individuals with OCD have a low threshold for perceiving themselves as behaviourally impulsive. In addition, we found a significant association between behavioural impulsivity and OCD symptom severity.

We believe that the association between OC symptom severity and self-reported behavioural impulsivity may be understood as a tendency to misinterpret obsessive symptoms (in the sense of lack of control over thoughts) as an indication for lack of control over behaviour (i.e., behavioural impulsivity). In our view, this association is circular: individuals with OCD react to their perception of being 'out of control' by increasing their attempts to regain control, as expressed by an increase in obsessive thoughts. In addition, the association between self-reported behavioural impulsivity and OC symptom severity within the OCD group implies that OCD are biased in their perception of behavioural impulsivity. Cottraux and Gérard (1998) proposed a theoretical model of 'Perceived Impulsivity' in OCD, defined as a tendency to believe that the individual is dangerous to others. The authors suggested that perceived impulsivity triggers compulsive behaviours (Cottraux & Gerard, 1998). To our knowledge, this study is the first to provide empirical evidence for the distortion in the perception of behavioural impulsivity in OCD and its association with OC symptoms. We believe that the biased self-perception of behavioural impulsivity is more generalized than a belief that one may cause harm to others. In our view, the lack of control over obsessive thoughts is associated with an indiscriminate fear of losing control over behaviour. Individuals with OCD are familiar with the power that obsessions and compulsions has on their behaviour, and the difficulty they encounter in suppressing these thoughts and ritualistic behaviours. Together with the perception of lack of control over obsessive thoughts, and the low threshold for perceiving their actions as impulsive, these individuals not only have a strong preference for controlled processing and dread automaticity (Rauch *et al.*, 1997; Soref, Dar, Argov, & Meiran, 2008), but experience fear of acting upon impulses – what we termed Global Fear of Impulsivity. This fear is termed 'global' because we believe that it is not restricted to conscious fear of harming other but rather encompasses fear of semi-conscious, lower – level automatic gestures as well. This may have important consequence in terms of heightened motivation to regain control as described below.

A proposed neuropsychiatric executive overload model in OCD

OCD and ADHD exhibit antithetical clinical pictures and opposite pattern of frontostriatal brain activity, but both disorders are characterized by comparable neuropsychological impairments. We believe that the interactions reviewed above and the dissociation between response inhibition and behavioural impulsivity in OCD suggest that neuropsychological impairments in ADHD and OCD stem from very different processes. Specifically, we suggest a hypothetical model of OCD (presented in Figure 2) in which neuropsychological impairments in OCD are seen as an epiphenomenon. The model postulates that the overflow of obsessive thoughts in OCD, which is associated with hyperactivity of the frontostriatal system, is a manifestation of a continuous attempt to control automatic processes. This overflow of obsessive thoughts, in turn, causes an

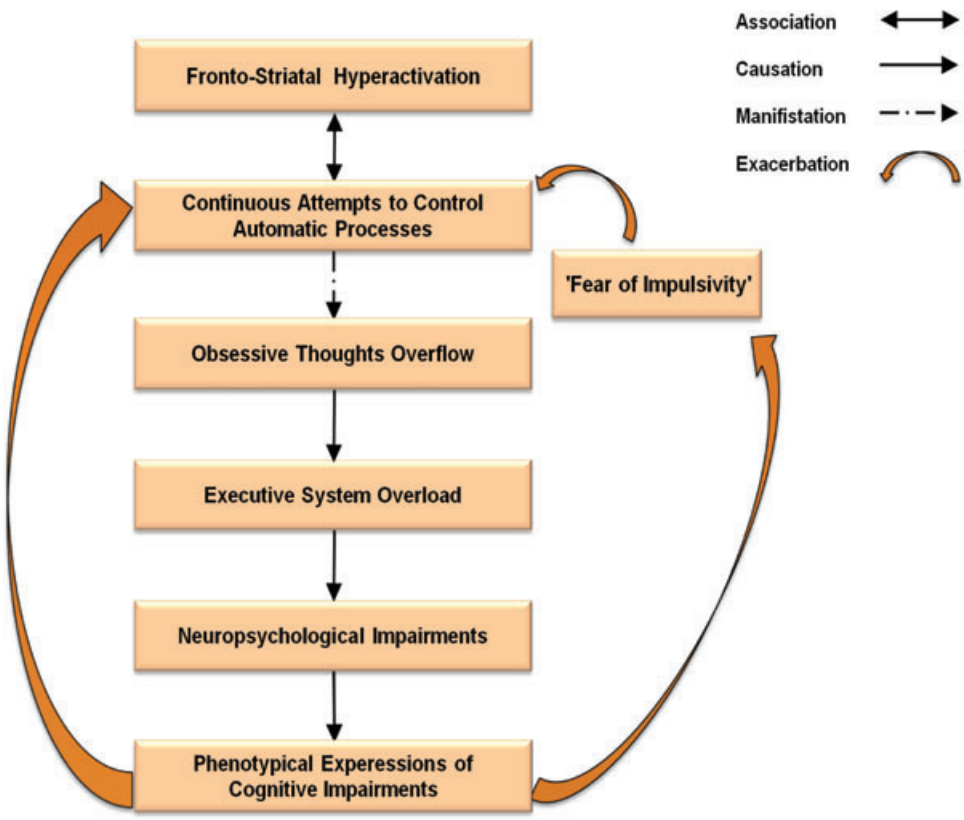

Figure 2. Neuropsychiatric executive overload model in OCD. The model postulates that the overflow of obsessive thoughts in OCD, which is associated with hyperactivity of the frontostriatal system, is a manifestation of a continuous attempt to control automatic processes. This overflow of obsessive thoughts, in turn, causes an overload on the executive system, which consumes cognitive resources and result in neuropsychological impairments. Awareness of these impairments increases the biased perception of control over behaviour. The activation of the 'general fear of impulsivity' increases attempts to control behaviour (e.g., by attending to lower level automatic processes). These attempts, in turn, lead to increase in obsessions and to further overload on the executive system, resulting in a vicious circle.

overload on the executive system, which consumes cognitive resources and result in neurocogntive impairments. Once individuals with OCD become aware of these deficits (e.g., making 'real life commission errors', being forgetful, arriving late to appointments, having difficulty concentrating), their fear of impulsivity increases, causing them to redouble their attempts to control their behaviour (e.g., by attending to lower level automatic processes). These attempts, in turn, lead to increase in obsessions and to further overload on the executive system, in a vicious circle. The paradoxical nature of this model corresponds to evidences regarding thought control in OCD. Continuous attempts to control thoughts in OCD are thought to result paradoxically in increase in obsessive thoughts and facilitate compulsive rituals (Clark & Purdon, 1993; Rachman, 1997; Salkovskis, 1985). This hypothetical chain of events contrasts with a simpler picture in ADHD, where neuropsychological impairments directly reflect the underactivity

of prefrontal regions that hinders normal cognitive functioning. Furthermore, as noted above, there is a direct association between prefrontal underactivity and behavioural characteristics such as impulsiveness and risk taking in ADHD.

As reviewed above, numerous functional imaging studies provide evidence of abnormal hyperactivation of the frontostriatal system in OCD (including the prefrontal regions, striatum, and mutual connections) that is positively correlated with symptom severity and was found to decline after successful treatment (Baxter *et al.*, 1992; Schwartz, Stoessel, Baxter, Martin, & Phelps, 1996). Rauch *et al.* (1997) described this pattern of brain activity in OCD as a reflection of a strong tendency towards explicit, controlled, and conscious processing of information that non-patients process implicitly and without conscious awareness (Rauch *et al.*, 1997). The inverse association we found between OC symptoms and response inhibition abilities is in accord with other studies that found negative correlations between OC symptoms and different neuropsychological tests (Lacerda *et al.*, 2003b; Segalas *et al.*, 2008; Tallis, 1997; Tallis, Pratt, & Jamani, 1999). It is also consistent with the view that individuals experiencing anxiety and depression pay a "cognitive price" for emotionally loaded thoughts. It has been previously suggested that high levels of anxiety 'consume' working memory capacities during task performance due to irrelevant processing of intrusive information (Eysenck, 1992; Gotlib, Roberts, & Gilboa, 1996). More recently, Boyer and Liénard (2006) suggested that 'flooding of the working memory' occurs in OCD as a result of constant preoccupation with, and attempt to control fragmented lower level gestures (Boyer & Lienard, 2006). In fact, recent studies demonstrated how the propensity to maintain high level of control in OCD results in lack of cognitive flexibility that impairs performance on cognitive tasks (Soref *et al.*, 2008).

It should be noted, however, that correlation does not necessary reflect causation. While it is logical to hypothesize that an overflow of obsessive thoughts consumes neurocognitive resources needed for other cognitive task, there is always the possibility that individuals with OCD, who are more cognitively impaired, may be more likely to have more severe scores on the OCI-R. Nevertheless, this possibility is undermined by studies that found that cognitive behavioural therapy, focused on OC symptoms in OCD, resulted in reduction of symptoms *and* neuropsychological impairments (Kuelz *et al.*, 2006).

Our findings suggest that individuals with OCD experience a distortion in estimating their level of control over their own behaviour. In our view, this finding demonstrates a 'global fear of impulsivity' in individuals with OCD, which is associated with a low threshold for perceiving themselves as behaviourally impulsive or 'out of control' (Moulding & Kyrios, 2006; Reuven-Magril *et al.*, 2008). Our model postulates that facing real life situations that imply a partial loss of control, individuals with OCD experience fear of losing control, which in-turn will activate increased attempts to control automatic processes that eventually will exact a 'price' in terms of neuropsychological performance.

In designing the present study, we attempted to address potential confounding factors and methodological limitations of neuropsychological research in general as well as those pertaining to the specific populations under study. For example, some studies suggest that impairments in executive functioning in OCD may be accounted for by comorbid depression (Basso, Bornstein, Carona, & Morton, 2001). Our samples were carefully matched for age and education, and statistical analyses were performed in order to examine the potential impact of medication, comorbid conditions, and depressive severity in our OCD sample.

In addition, neuropsychological research typically examines several domains and subdomains of performance using multiple statistical tests, which make it susceptible to inflation of type I error rate. This issue has been discussed particularly with regard to OCD (Bedard, Joyal, Godbout, & Chantal, 2009; Kuelz *et al.*, 2004; Purcell, Maruff, Kyrios, & Pantelis, 1998). Two commonly accepted solutions for this problem are the merging of subdomains into larger domains and adjusting alpha levels to avoid inflation of type I error. In this study, we employed both practices by analysing neuropsychological performance on general normalized domain indexes as well as individual test parameters comprising each domain index. In addition, we adjusted the level of alpha to the number of statistical tests.

A number of limitations of this study, mostly in terms of our participant population, should be noted. First, only male participants were included in this study and our results should be replicated using both genders. Second, our sample size did not permit an analysis of OCD subtypes and comparisons of subgroups of OCD with and without comorbid axis I disorders resulted in small sample sizes. Several studies, however, demonstrate that apart from a possible impact of comorbid depressive severity, there is a surprising lack of impact of comorbid conditions on neuropsychological functioning in participants with primary OCD (Aycicegi *et al.*, 2003; Kuelz *et al.*, 2004; Simpson *et al.*, 2005). Third, whereas approximately half of our OCD participants were medicated, our ADHD participants were medication-free. Similarly, while we did not find significant differences in neuropsychological performance between medicated and un-medicated OCD participants, sample sizes were relatively small. Nevertheless, several studies suggest that neuropsychological performance is not affected by continuous SSRI treatment in individuals with OCD (Mataix-Cols *et al.*, 2004; Roh *et al.*, 2005; Savage *et al.*, 2000). Fourth, the OCI-R scores in our OCD sample reflect a rather severe degree of symptoms (Abramowitz *et al.*, 2005). Nevertheless, similar neuropsychological impairments were found in numerous OCD samples with various degrees of severity (Bucci *et al.*, 2007; Chamberlain *et al.*, 2006; Penades *et al.*, 2005; Rao *et al.*, 2008). Finally, our ADHD sample was drawn from one special learning disability program in an academic institution, which may limit the generalizability of our findings regarding this group. This may not pose a serious problem, however, as very similar neuropsychological impairments were found in numerous samples of adults with ADHD, and recently in a selected sample of high-IQ adults with ADHD (Antshel *et al.*, 2010).

Conclusion

By performing a direct neuropsychological and symptomatological comparison between ADHD and OCD, we were able to clarify how two disorders characterized with opposite pattern of brain activity and an antithetical clinical picture share similar neuropsychological impairments. Underactivity of brain regions in ADHD corresponds to behavioural impulsivity (hypocontrol over behaviour) and to neuropsychological impairments, especially in tests of executive functions. In OCD in contrast, hyperactivity of the frontostriatal system may reflect the tendency to control behaviour (hypercontrol) to a degree that impairs normal cognitive functioning. Notably, a similar notion of dissociation between disorder-specific symptoms and neurocognitive functioning was recently proposed in a study of implicit learning and delayed aversion in OCD and ADHD children (Vloet *et al.*, 2010). Our model may contribute to clinicians as well as individuals diagnosed with OCD in proposing that neuropsychological impairments may be viewed as a second-order consequence of OC symptoms. In addition, our results may

alert clinicians to attend to the fact that while patients with OCD accurately self-report low levels of behavioural impulsivity, they tend to perceived themselves as behaviourally impulsive and as a consequence increase their level of control. Furthermore, our model predicts that successful treatment reducing OC symptoms in OCD will be complemented by reduction in neuropsychological impairments (Kuelz *et al.*, 2006). In the future, it will be useful to further examine changes in neuropsychological functioning upon successful treatment in OCD. Finally, our proposition regarding opposite mechanisms underlying similar neuropsychological impairments in ADHD and OCD, together with contrasting clinical picture and pattern of brain activity, challenges the possibility of comorbidity between the two disorders in the adult population.

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