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Invited Review

The cognitive neuropsychology of obsessive-compulsive disorder: A critical review

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ABSTRACT

For over a quarter century, a substantial body of literature investigating neuropsychological test performance in obsessive-compulsive disorder (OCD) has yielded inconsistent results. Thus, it has been continuously challenging to draw conclusions regarding an OCD-specific neuropsychological profile. In this comprehensive review of the neuropsychological literature in OCD, we critically review neuropsychological test performance by domain, as well as potential moderators of neuropsychological functions, proposed endophenotypes, neuropsychological predictors of treatment response, and contemporary controversies in the field. Previous qualitative/systematic reviews of this body of literature have repeatedly noted its inconsistency, concluding that more research is needed. Unfortunately, the accumulation of neuropsychological research is OCD has not yet promoted our ability to draw conclusions about a distinct neuropsychological profile of OCD. Thus, we conclude this review with novel suggestions for future investigations.

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1. Introduction

More than 250 peer-reviewed journal articles have been published in the past quarter century exploring neuropsychological test performance in obsessive-compulsive disorder (OCD). However, this body of literature is characterized by unusual inconsistencies that have persisted for nearly two decades (Abramovitch, Abramowitz, & Mittelman, 2013; Kuelz, Hohagen, & Voderholzer, 2004; Tallis, 1997). Furthermore, there have been numerous controversies regarding the specificity of cognitive deficits in OCD, the functional and clinical correlates of neuropsychological test performance, and their etiological role. In this paper, we provide an up-to-date comprehensive and critical review of the literature on the cognitive neuropsychology of OCD. Following a brief introduction to the neurobiology of OCD, we evaluate the neuropsychological literature by cognitive domain. Subsequently, we review neuropsychological findings associated with symptom dimensions, moderators of neuropsychological test performance, neuropsychological correlates of treatment response, and neurocognitive endophenotypes. We then discuss three major contemporary controversies in the field and conclude this review with novel recommendations for future research.

2. Neurobiology of OCD

Neuropsychological test performance is thought to reflect neurobiological abnormalities, predominantly insult to brain tissue (Lezak, Howieson, Bigler, & Tranel, 2012). However, with the emerging notion that psychiatric conditions may be associated with functional and structural brain abnormalities, as well as the development of sophisticated neuroimaging technology in the late 1980s, a growing interest in neurobiological mechanisms of psychiatric disorders has yielded a vast body of research. Consequently, a substantial body of imaging research investigating neural substrates of OCD has accumulated, and the findings are considered among the most robust in the psychiatric literature (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005).

Although numerous brain regions have been implicated in the pathophysiology of OCD, the prevailing model proposes that obsessive-compulsive (OC) symptoms are associated with dysfunction in the cortico-striato-thalamo-cortical circuitry (CSTC; Huey et al., 2008; Pauls, Abramovitch, Rauch, & Geller, 2014; Saxena & Rauch, 2000). This 'frontostriatal' model stipulates that a feedback loop imbalance leads to hyperactivity of the orbitofrontal–subcortical pathways in OCD (Melloni, Urbistondo, Sedeno, Gelormini, Kichic, & Ibanez, 2012; Pauls et al., 2014). As a result, individuals with OCD exhibit a bias toward, and excessive attention to, threatening stimuli and may consequently engage in compulsive behaviors (Pauls et al., 2014; Saxena & Rauch, 2000).

The majority of neuroimaging studies have identified significant resting state hyperactivation in the frontal and basal brain regions and their connecting pathways, including the orbitofrontal cortex (OFC; Huey et al., 2008), the caudate nucleus (Baxter, Phelps, Mazziotta, Guze, Schwartz, & Selin,1987), the anterior cingulate cortex (ACC; Breiter et al., 1996), and the thalamus (Lacerda, Dalgalarrondo, Caetano, Camargo, Etchebehere, & Soares, 2003; Perani et al., 1995). Resting state connectivity studies provide additional support for the CSTC model, indicating aberrant hyperactivation along the frontostriatal circuitry (Fitzgerald et al., 2010; Harrison et al., 2009). Furthermore, converging evidence from symptom provocation (Adler, McDonough-Ryan, Sax, Holland, Arndt, & Strakowski, 2000; Rauch et al., 1994) and treatment studies (Benkelfat, Nordahl, Semple, King, Murphy, & Cohen, 1990; Kang et al., 2003; Perani et al., 1995; Saxena et al., 1999) implicate the CSTC network in the pathophysiology of OCD. More recent studies have focused on additional regions and circuits that may play a role in the pathophysiology of OCD, including the dorsolateral prefrontal cortex (DLPFC), the parietal cortex, and their connection with frontal structures (Milad & Rauch, 2013). Combined with evidence from functional imaging during neuropsychological task performance, the prevailing neurobiological model of OCD predicts neuropsychological deficits, predominantly in the domain of executive function, which the frontostriatal system is thought to subserve (Pauls et al., 2014).

3. Neuropsychological performance across domains

3.1. Executive functions

Functional abnormalities depicted in the CSTC model of OCD may be associated with broad neurocognitive deficits. However, the frontostriatal network is thought to predominantly subserve higher-order executive functions, including response inhibition, planning, set shifting, and verbal/figural fluency (Chudasama & Robbins, 2006). Indeed, numerous studies investigated these functions in OCD. Below we review this body of literature by subdomain.

3.1.1. Response inhibition

Response inhibition (RI), the ability to inhibit a pre-potent motor response, has been extensively studied in individuals with OCD. The ever-growing interest in this construct across populations stems primarily from its known association with behavioral impulsivity depicted in classic neuropsychology (Keilp, Sackeim, & Mann, 2005). While OCD is not associated with behavioral impulsivity per se (see discussion below under 'Controversies'), some authors have hypothesized that the inability to stop the overflow of obsessive thoughts, and particularly the view that individuals with OCD exhibit an inability to stop ongoing repetitive rituals, may stem from impairment in response inhibition (Chamberlain et al., 2005).

RI is most commonly assessed using the Continuous Performance Test (CPT), the Go/No-Go Task (GNG), and the Stop Signal Task (SST). A related construct – interference control – is frequently assessed using the Stroop task. Individuals with OCD tend to exhibit deficient performance on the SST (Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; de Wit et al., 2012; Menzies et al., 2007; Penades, Catalan, Rubia, Andres, Salamero, & Gasto, 2007). However, among studies examining commission errors (CErr; the gold standard

indicator for RI) performed during CPT and GNG tasks, only a minority report more CErr among individuals with OCD compared to controls (Abramovitch, Dar, Schweiger, & Hermesh, 2011; da Rocha, Alvarenga, Malloy-Diniz, & Correa, 2011; Ghisi, Bottesi, Sica, Sanavio, & Freeston, 2013; Penades et al., 2007), while the majority of studies report no difference in the number of CErr on the CPT (Krishna et al., 2011; Lee, Yost, & Telch, 2009; Tolin, Villavicencio, Umbach, & Kurtz, 2011; Ursu, Stenger, Shear, Jones, & Carter, 2003) and GNG tests (Bohne, Savage, Deckersbach, Keuthen, & Wilhelm, 2008; Page et al., 2009; Thomas, Gonsalvez, & Johnstone, 2013; Watkins et al., 2005). Mixed results have been found among studies examining performance on the Stroop test. Whereas the majority of studies report deficient performance (primarily increased Stroop interference; Abramovitch et al., 2011; Martinot et al., 1990; Penades, Catalan, Andres, Salamero, & Gasto, 2005), other studies did not find such differences between OCD and control samples (Moritz et al., 2002; Rao, Reddy, Kumar, Kandavel, & Chandrashekar, 2008).

Methodological differences between studies examining RI, particularly the use of different tests to measure RI, may contribute to the contrasting results in this subdomain. The GNG task – where examinees are required to avoid responding to a no-go stimuli – is considered a measure of action suppression, whereas the SST – a task involving stopping an ongoing response – assesses action cancellation (Eagle, Bari, & Robbins, 2008). The SST is also a test of higher inhibitory load (Schachar et al., 2007), which may confound comparisons between studies of RI. Indeed, recent research suggests that there are different underlying neural substrates and neurochemical correlates associated with RI, as measured by the GNG/CPT versus the SST (Eagle et al., 2008; Swick, Ashley, & Turken, 2011; van Velzen, Vriend, de Wit, & van den Heuvel, 2014).

A recent meta-analysis of neuropsychological test performance in OCD found a medium effect size for RI, indicating reduced performance in OCD. This accompanied a statistically significant effect size heterogeneity across subdomains reflecting the known between-studies inconsistency (Abramovitch et al., 2013). As discussed later in this review, in light of the contrasting results, methodological variations, and the lack of association with behavioral impulsivity or OCD-specific symptomatology, the extent of an RI deficiency and its etiological role in OCD has been subject to recent controversy.

3.1.2. Planning

Planning, the ability to achieve a goal through a series of steps, is an important aspect of higher order cognitive functions. Planning is usually assessed using the Tower of London (TOL) and Tower of Hanoi (TOH) tasks, in which effective planning minimizes the number of steps required to complete these versions of a peg-and-disc puzzle. Most studies report reduced planning ability among individuals with OCD, as measured by the TOL (Lennertz et al., 2012; Rampacher et al., 2010; Tukel et al., 2012) and the TOH (Cavallaro et al., 2003; Cavedini, Zorzi, Piccinni, Cavallini, & Bellodi, 2010). Fewer studies report intact performance on these tests (Rajender, Bhatia, Kanwal, Malhotra, Singh, & Chaudhary, 2011; Schmidtke, Schorb, Winkelmann, & Hohagen, 1998). Although yielding slightly different results due to inclusion criteria and outcome measure differences, two recent meta-analyses reported differences in planning abilities between OCD individuals and control samples. One meta-analysis of 115 studies found reduced planning ability in OCD samples with an overall moderate effect size (Abramovitch et al., 2013). In a second meta-analysis of 88 studies, Shin et al. (2014) analyzed effect sizes exclusively for 'excessive moves' in the TOH and TOL, and found an overall large effect size for reduced performance in OCD samples compared to control samples. Overall, it appears that OCD is associated with deficits in planning that correspond to moderate to large effect sizes.

3.1.3. Set shifting

Set shifting, or attentional set shifting, is the ability to continuously disengage from irrelevant stimuli or information while engaging in relevant task features. Some studies found that individuals diagnosed with OCD exhibit deficiencies on tasks requiring set shifting, such as the Wisconsin Card Sorting Test (WCST), especially with regards to perseverative errors (Aigner et al., 2007; Bucci et al., 2007; Okasha et al., 2000; Tukel et al., 2012), as well as on the Trail Making Test Part B (TMB; Burdick, Robinson, Malhotra, & Szeszko, 2008; N. Hashimoto et al., 2011; T. Hashimoto et al., 2008). In addition, individuals with OCD have demonstrated deficient set shifting on the Cambridge Neuropsychological Test Automated Battery (CANTAB) Set Shifting Task (Fenger, Gade, Adams, Hansen, Bolwig, & Knudsen, 2005; Purcell, Maruff, Kyrios, & Pantelis, 1998b; Watkins et al., 2005).

However, most studies found no differences between OCD and control samples on set shifting tasks, including on the WCST (Burdick et al., 2008; Cavallaro et al., 2003; Hwang, Kwon, Shin, Lee, Kim, & Kim, 2007; Kim, Park, Shin, & Kwon, 2002; Kitis et al., 2007; Nakao et al., 2009; Roth, Baribeau, Milovan, & O'Connor, 2004; Simpson et al., 2006), the TMB (de Geus, Denys, Sitskoorn, & Westenberg, 2007; Hwang et al., 2007; Kivircik, Yener, Alptekin, & Aydin, 2003; Krishna et al., 2011; Roth et al., 2004), and the CANTAB Set Shifting Task (Basso, Bornstein, Carona, & Morton, 2001; Moritz, Birkner, et al., 2001; Nielen & Den Boer, 2003; Purcell, Maruff, Kyrios, & Pantelis, 1998a; Simpson et al., 2006). Similarly, although some studies reported reduced performance among individuals with OCD on the Object Alteration Task (OAT) and the Delayed Alteration Task (DAT; Abbruzzese, Ferri, & Scarone, 1997; Aycicegi, Dinn, Harris, & Erkmen, 2003; Moritz, Fricke, & Hand, 2001), more studies report comparable performance on these tasks between OCD and control samples (Bohne et al., 2005; Kashyap, Kumar, Kandavel, & Reddy, 2013; Kuelz, Riemann, Zahn, & Voderholzer, 2004; Moritz, Jelinek, Hottenrott, Klinge, & Randjbar, 2009; Voderholzer et al., 2013).

Overall, the majority of studies assessing set shifting indicate normative performance in OCD samples. This trend was previously identified by Kuelz, Riemann, et al. (2004), concluding that there was no sufficient evidence at the time to determine that OCD is associated with a deficit in this domain, and that more research is needed. A decade later, this trend persists, with the majority of studies indicating no deficient performance on set shifting tasks in OCD samples.

3.1.4. Verbal and figural fluency

Fluency tests are used to assess the voluntary generation of non-overlearned responses (Robinson, Shallice, Bozzali, & Cipolotti, 2012). Verbal Fluency (VF) tests, such as the Controlled Word Association Test, are generally comprised of a Semantic Verbal Fluency (SVF) trial where examinees are asked to provide as many items as possible related to specific categories (e.g., fruit) in a given timeframe, as well as a Phonemic Verbal Fluency (PVF) trial that requires production of words starting with a given letter. Non-verbal Figural Fluency tests require examinees to produce as many distinct (non-repeating) abstract designs in a given time. Although all fluency tests are considered tests of executive function, only PVF and Figural Fluency tests have been significantly associated with activation in frontal/prefrontal brain regions (Robinson et al., 2012).

A relatively small number of studies assessed SVF in OCD, yielding mixed results (de Geus et al., 2007; N. Hashimoto et al., 2011; Lacerda, Dalgalarrondo, Caetano, Haas, Camargo, & Keshavan, 2003; Watkins et al., 2005). Although more data are available on PVF in OCD, results are equally inconsistent: some studies report reduced performance in OCD compared to controls (Christensen, Kim, Dysken, & Hoover, 1992; Harris & Dinn, 2003), whereas others report intact performance

(Aycicegi et al., 2003; Krishna et al., 2011). Two meta-analytic investigations assessing fluency found small to moderate effect sizes (Henry, 2006; Shin et al., 2014), representing reduced performance on verbal fluency tests in OCD samples. Interestingly, in a meta-analysis specifically examining performance on VF tests and the WCST in OCD, Henry (2006) reported no significant discriminatory effect of verbal fluency tests between OCD and control participants. Additionally, the author concluded that reduced performance on VF tests did not reflect executive function deficits in OCD, but rather a general cognitive impairment. However, given the paucity of studies and the inconsistent nature of results in this domain, more studies are needed examining fluency tests in OCD samples.

3.2. Processing speed

The concept of 'obsessional slowness' in OCD was identified several decades ago (Rachman, 1974). However, later research has focused more on neurocognitive aspects of slowness, primarily processing speed. OCD samples have shown reduced processing speed on tests such as the Digit-Symbol Coding subtest of the Wechsler Adult Intelligence Scale (WAIS) and the Trail Making Test part A (TMA; Basso et al., 2001; N. Hashimoto et al., 2011; T. Hashimoto et al., 2008). In addition, mean reaction times on the GNG test and the Stroop congruent trials have revealed reduced processing speed in individuals with OCD (e.g., Abramovitch et al., 2011; T. Hashimoto et al., 2008; Penades et al., 2007). As discussed below, however, there is an ongoing debate about whether reduced processing speed is an epiphenomenon resulting from OC symptoms (Abramovitch et al., 2011), or instead a state-independent trait of OCD (Galderisi, Mucci, Catapano, D'Amato, & Maj, 1995). In fact, it has been argued that reduced processing speed may be the primary deficit in OCD, accounting for reduced performance on tests assessing other neuropsychological functions (Burdick et al., 2008). Although reduced processing speed may impact an array of neuropsychological functions (Ball, Edwards, & Ross, 2007), further research comparing timed versus untimed tasks is needed to substantiate this hypothesis in OCD.

3.3. Working memory

Working memory (WM) is conceptualized as the ability to store, manipulate, and integrate information for a very short amount of time. Working memory functions also facilitate integration of such information with long-term memories. Thus, not to be confused with memory function per se, WM is crucial for supporting executive functions and has been primarily associated with activation in the DLPFC and other prefrontal regions (Nee et al., 2013). Traditionally, WM is comprised of two subdomains: verbal working memory (VWM) and visuospatial-working memory (VSWM).

One of the most common tasks assessing VWM is the WAIS Digit Span test, on which very few available studies report reduced performance among individuals with OCD compared to controls (Sayin, Oral, Utku, Baysak, & Candansayar, 2010; Tukel et al., 2012). The majority of studies found no performance difference on this task (e.g., Boldrini et al. 2005; T. Hashimoto et al., 2008; Segalàs et al., 2010). Comparable performance among OCD and control samples was also found on the Wechsler Letter-Number Sequencing test (Krishna et al., 2011), a task with a higher verbal working memory load. Results from a study assessing VWM using a verbal N-Back task, reveal an interesting pattern of results. In this study (Kashyap et al., 2013), individuals with OCD performed similarly to controls on the 1-back condition, but worse on the 2-back condition of the Verbal N-Back task, suggesting that deficient performance on VWM tasks in OCD may be pronounced only on tasks/trials of higher VWM load. These results correspond to an overall small effect size found on tasks of VWM (Abramovitch et al., 2013).

Tasks assessing VSWM vary in task load, complexity, and difficulty. For example, the Wechsler Memory Scale (WMS) Spatial Span test is a spatial analogue to the WAIS Digit Span test and is considered of average load. Research indicates similar performance among individuals with OCD compared to controls on the WMS Spatial Span task (Krishna et al., 2011; Nakao et al., 2005). However, reduced performance was reported on the CANTAB Spatial Span task, an analogue computerized version of the WMS Spatial Span test (Nedeljkovic et al., 2009; Purcell et al., 1998b). In addition, a handful of studies found that OCD samples demonstrated reduced performance compared to controls on the CANTAB SWM task, a more complex and demanding VSWM test (Nedeljkovic et al., 2009; Purcell et al., 1998b), whereas other studies reported comparable performance (Morein-Zamir et al., 2010; Nielen & Den Boer, 2003). A similar pattern was found when assessing performance on the Spatial N-Back task. Some studies reported comparable performance between OCD and controls samples (Koch et al., 2012; Nakao et al., 2009), while a few studies reported reduced performance, but only on higher load trials (i.e., 3 back; de Vries et al., 2013; van der Wee et al., 2003). Overall, small to moderate effect sizes were found for VSWM (Abramovitch et al., 2013; Shin et al., 2014).

Although results vary, an overall trend emerges where compared to non-psychiatric controls, individuals with OCD perform progressively worse on more complex, higher load trials assessing VWM and VSWM, while performing comparably on simpler tasks associated with lowered task load. The evidence for normative performance on VWM and VSWM on low and medium load, but deficient performance on higher load tasks or trials, may correspond to similar results on tests of RI where OCD is associated with deficient performance on an RI task of higher load (SST) and intact performance on RI tasks of lower load (i.e., GNG and CPT).

3.4. Attention

Omission errors (failing to respond to a 'go' stimuli) on the CPT and GNG tests are frequently used to assess general attention. Most studies using these paradigms report a comparable number of omission errors between individuals with OCD and healthy controls (Krishna et al., 2011; Penades et al., 2007; Watkins et al., 2005), although there are a few reports of increased omission errors among individuals with OCD (da Rocha et al., 2011). Other studies have used different outcome measures to assess sustained attention on GNG and CPT tasks, such as the CPT's sensitivity index change over time, and found no difference between OCD and control groups (Milliery, Bouvard, Aupetit, & Cottraux, 2000). However, studies assessing the reaction time standard deviation index of the GNG test found reduced sustained attention in an OCD sample compared to non-psychiatric controls (Abramovitch et al., 2011), as well as in a subclinical OC sample (Abramovitch, Shaham, Levin, Bar-Hen, & Schweiger, 2015). Overall, it appears that individuals with OCD do not underperform controls on outcome measures assessing general and sustained attention, but more research is needed to examine fluctuations in reaction times using computerized tasks.

3.5. Memory

The domain of memory functions can be divided in various ways, from immediate and delayed memory to semantic and episodic memory. This section will focus on the common division between verbal and non-verbal (visual) memory. Verbal memory is most commonly assessed by word list tests such as the California Verbal Learning Tests (CVLT), or in the form of story recall assessed using

the Wechsler Memory Scale's Logical Memory test (WMS-LM). Using these tasks, some studies have found reduced immediate and delayed verbal memory abilities in OCD individuals (Exner, Kohl, Zaudig, Langs, Lincoln, & Reif, 2009; Exner, Martin, & Rief, 2009; Tukel et al., 2012). However, the majority of studies report comparable performance between OCD and control groups (e.g., Christensen et al., 1992; Nakao et al., 2005; Sayin et al., 2010). Thus, it is safe to conclude that OCD is not associated with meaningful deficits in verbal memory. This conclusion corresponds to an overall small effect size for verbal memory, as reported in two meta-analyses (Abramovitch et al., 2013; Shin et al., 2014).

Non-verbal memory is the most highly researched neuropsychological domain in OCD, with nearly all studies utilizing the Rey-Osterrieth Complex Figure Test (ROCF or RCFT). The first condition of this task requires examinees to copy a complex figure. This trial is frequently considered a measure of visuospatial abilities. According to standard administration instructions, two subsequent memory trials are recommended: an immediate trial following the copy phase and a delayed recall trial either 20 or 30 min after the copy phase. However, one trial (i.e., copy+delayed or copy+immediate) can be used as well (Shin, Park, Park, Seol, & Kwon, 2006). The vast majority of studies report significantly deficient performance on the ROCF immediate and delayed recall trials among individuals with OCD (e.g. Penades et al., 2005; Rajender et al., 2011; Shin et al., 2004), and only a few studies reported intact performance among OCD samples (Exner, Kohl, et al., 2009). This corresponds to large effect sizes found in two meta-analyses (Abramovitch et al., 2013; Shin et al., 2014).

The ROCF is a unique memory test because examinees are never informed of the test's true nature (i.e., that this is a memory test). This is in contrast to other verbal (e.g., CVLT, WMS-LM) and non-verbal memory tests (e.g., WMS Faces). In the ROCF, examinees are presented with a figure and are asked to copy it. Once they are done, the figure is removed, and examinees are subsequently requested to recreate the figure from memory on a blank piece of paper, without any preceding conscious attempt to code the information. In addition to the stress individuals with OCD may experience under any conditions associated with testing, they also require structure, prefer explicit instructions, and respond unfavorably to 'surprises'. Thus, it is possible that the surprise element inherent to the ROCF may negatively influence their performance on this test, and possibly to a significantly larger extent than a study's control participants. It would be useful for future research to explore this possible disorder-specific effect.

This may be a particularly important line of research in OCD given three main reasons. First, nearly every study to date that assessed non-verbal memory in OCD has utilized the ROCF. Reliance on more than a single measure for a given neuropsychological construct within a given study (as well as across studies), should be the rule and not the exception (Lezak et al., 2012), especially when there is a reason to question a tests' psychometric integrity in a given population. In fact, studies utilizing other non-verbal memory tests (where examinees were informed as to the nature of the test) found comparable performance in OCD samples compared to controls (Moritz, Kloss, von Eckstaedt, & Jelinek, 2009; Moritz, Ruhe, Jelinek, & Naber, 2009; Moritz, Wahl, Zurowski, Jelinek, Hand, & Fricke, 2007). Second, as demonstrated by two recent meta-analyses (Abramovitch et al., 2013; Shin et al., 2014), effect sizes for non-verbal memory/ROCF were found to be large (~0.75), while effect sizes for verbal memory and visuospatial functions were found to be small. If basic visuospatial functions in OCD are not significantly impaired (see section below), such a gap in memory functions within a given population is unusual.

Third, performance on the ROCF is possibly mediated by organizational strategies. In a seminal study, Savage, Baer, Keuthen, Brown, Rauch, & Jenike (1999) demonstrated that a deficit in organizational and planning abilities in the copy phase of the ROCF mediated deficient performance on the immediate memory phase of the ROCF. This result led to the development of several scoring systems to assess organizational abilities using the ROCF (for a review see, Shin et al., 2006). Using these scoring systems, this mediation effect has been reliably replicated (e.g., Penades et al., 2005), suggesting that deficient organizational abilities of non-verbal information partially account for deficient performance on memory trials of the ROCF. This notion received support from a recent meta-analysis indicating that effect sizes for executive functions in OCD were significantly and positively correlated with effect sizes for non-verbal memory ($r=0.60$) but not with those of verbal memory (Abramovitch et al., 2013). These results suggest that organizational deficits may specifically mediate visuospatial memory functions in OCD, and further underscore the importance of a critical examination of the ROCF's construct validity in the context of OCD. Indeed, some researchers argue that non-verbal memory deficits may be secondary to executive deficits in OCD (Olley, Malhi, & Sachdev, 2007). Thus, whereas individuals with OCD exhibit substantial underperformance on the ROCF, potential confounding factors hinder our ability to determine to what extent individuals with OCD exhibit a specific deficit in non-verbal memory.

3.6. Visuospatial abilities

The most common tests used for the assessment of visuospatial abilities in OCD are the copy phase of the ROCF and the WAIS Block Design. Most studies revealed deficient performance on the Block Design test in OCD samples (Moritz, Hottenrott, Jelinek, Brooks, & Scheurich, 2012; Moritz, Kloss, Jahn, Schick, & Hand, 2003; Tukel et al., 2012), while fewer studies found intact performance (Martin, Pigott, Lalonde, Dalton, Dubbert, & Murphy, 1993; Roth, Milovan, Baribeau, & O'Connor, 2005). However, the majority of studies assessing visuospatial abilities using the ROCF copy phase found comparable performance (Kim et al., 2002; Moritz et al., 2003; Roth et al., 2004), with only a minority of studies reporting deficient performance among participants with OCD (Choi et al., 2004; Lacerda, Dalgalarrondo, Caetano, Haas, et al., 2003). Given the average slower processing speed associated with OCD, a possible explanation for the relatively intact performance on the ROCF copy trial, as opposed to the deficient performance on the Block Design test, could be the unique factor of time included in the scoring process of the latter. This explanation supports the hypothesis that processing speed deficiencies in OCD may underlie deficient performance on tests tapping different domains (Burdick et al., 2008).

4. Neuropsychological functions and OCD symptom dimensions

OCD is clinically heterogeneous, and patients may present with one or more distinct symptoms (e.g., checking, washing; Stewart et al., 2008). To characterize these symptoms, factor-analytic studies have generally distinguished four primary OC symptom dimensions: contamination, unacceptable/taboo thoughts, symmetry/ordering, and hoarding (Bloch, Landeros-Weisenberger, Rosario, Pittenger, & Leckman, 2008). Notably, a large multisite study recently suggested 'responsibility for harm' as a fourth factor instead of hoarding, which is now considered a distinct condition in the DSM-5 (Abramowitz et al., 2010). Consequently, research has investigated whether distinct neural and neuropsychological profiles underlie the expression of specific OCD symptom dimensions. A number of imaging studies have investigated patterns of neural activation in OCD individuals thought to underlie different

symptom dimensions (Mataix-Cols et al., 2003), generally reporting differences between patients with washing symptoms and patients with checking symptoms.

Washing symptoms have been associated with increased activation in brain regions related to disgust, including the insula, inferior frontal sites, and parahippocampal regions (Phillips et al., 2000; Shapira et al., 2003), while checking has been associated with activation in the dorsal prefrontal regions (Mataix-Cols et al., 2003). A small number of studies have also looked at brain activation associated with hoarding symptoms, suggesting distinctive patterns of brain activity in the ventromedial prefrontal cortex (e.g., An et al., 2009). Taken together, these studies may provide preliminary evidence that various neural substrates mediate the symptom dimensions of OCD (Mataix-Cols, Wooderson, Lawrence, Brammer, Speckens, & Phillips, 2004; Pauls et al., 2014). However, these studies were constrained by somewhat arbitrary symptom dimension cut-offs, limited categories of symptoms assessed, and small sample sizes, therefore necessitating further research to replicate and substantiate these findings.

Similar correlational analyses have been conducted comparing symptom dimensions and neuropsychological profiles. The symmetry/ordering dimension has been associated with poorer performance on tests of executive functioning and verbal memory (N. Hashimoto et al., 2011), as well as nonverbal memory (Jang et al., 2010). Several studies have also reported significant deficits in executive functioning and non-verbal memory among "checkers," including worse performance on the subtracted score of the Trail Making Test (N. Hashimoto et al., 2011), the Stroop and GNG tests (Omori et al., 2007), and the ROCF (Cha et al., 2008). Furthermore, one study directly comparing OCD individuals with primary checking symptoms and those with primary symmetry/ordering symptoms found significant differences in neuropsychological test performance such that the former showed dysfunction in organizational strategies and the latter in nonverbal memory (Jang et al., 2010). Notably, examining the contamination/cleaning dimension, N. Hashimoto et al. (2011) found that these symptoms were associated with *better* performance on tests of verbal memory and inhibition.

On the other hand, numerous studies have not found associations between OC symptom dimensions and neuropsychological test performance. For example, Tallis, Pratt and Jamani (1999) did not find a relationship between checking symptoms and non-verbal memory. In fact, most studies that did find such associations identified neuropsychological correlates of symptoms dimensions in only one or two tests out of comprehensive neuropsychological test batteries. Additionally, the research on hoarding symptoms and neuropsychological performance is still very limited, and thus conclusions cannot yet be drawn (Mataix-Cols, Pertusa, & Snowdon, 2011). The paucity of research, characterized by inconsistent findings, questions the notion of distinct neuropsychological profiles associated with OCD symptom dimensions. Notably, the major limitation of these studies is the multicollinearity among OCD symptom dimensions. That is, individuals with OCD often score higher on multiple symptom dimensions scales. This phenomenon poses a challenge for both studies comparing OCD samples formed based on a single dimension (e.g., 'checkers' or 'washers'), as well as for correlational studies.

5. Moderators of neuropsychological test performance

The inconsistencies across neuropsychological investigations in OCD lead to a necessary examination of potential confounding factors (or moderators) of neuropsychological test performance. Some researchers reported that comorbid Major Depressive Disorder (Basso et al., 2001), or severity of depressive symptoms regardless of a depression diagnosis (Moritz, Fricke, et al., 2001), may in part account for neuropsychological deficits found in OCD. However, the majority of studies did not find a major moderating role of depression or depressive severity in OCD. Others suggested that age of onset may moderate neuropsychological functions in OCD, but very few studies reported such an effect (Roth et al., 2005). Studies examining the impact of Selective Serotonin Reuptake Inhibitors (SSRIs) on neuropsychological test performance in OCD (Mataix-Cols, Alonso, Pifarre, Menchon, & Vallejo, 2002), as well as gender as a confounding factor, revealed no meaningful effect (Mataix-Cols et al., 2006). Other researchers argue that memory confidence, and not memory impairments per se, characterizes OCD (e.g., Dar, Rish, Hermesh, Taub, & Fux, 2000). In addition, as noted above, slow processing speed has been proposed as a confounding factor underlying underperformance on tests of executive functions among individuals with OCD (Burdick et al., 2008). Finally numerous studies tested the hypothesis that OCD symptom severity may impact neuropsychological performance. However, from over 60 studies assessing this association, less than 10 studies reported significant correlations between symptom severity and specific neuropsychological outcome measures in OCD. Overall, meta-analytic investigations of neuropsychological test performance in OCD have revealed no meaningful moderator effects, apart from a few trends (Abramovitch et al., 2013; Shin et al., 2014).

6. Neuropsychological functions and treatment studies

The first line treatments for OCD are cognitive-behavioral therapy (CBT; particularly exposure and response prevention) and medication management with SSRIs (Abramovitch, Elliott, Wilhelm, Steketee, & Wilson, 2014; Koran, Hanna, Hollander, Nestadt, & Simpson, 2007). OCD treatment studies have generally found consistent neurobiological changes as a result of both psychological and pharmacological interventions. Individuals with OCD demonstrate significant reduction in cerebral metabolism in the OFC, ACC, caudate nucleus, cerebellum, and thalamus post-treatment (Benkelfat et al., 1990; Kang et al., 2003; Perani et al., 1995; Saxena et al., 1999, 2009; Schwartz, Stoessel, Baxter, Martin, & Phelps, 1996).

Several studies have examined treatment's impact on neuropsychological test performance in OCD. This type of research has generally taken two different approaches: investigating neuropsychological functions as predictors of OCD treatment response, or if and how cognitive functioning changes after treatment. Although research on the former is only preliminary, D'Alcante et al. (2012) found that higher verbal IQ, better verbal memory, and better performance on the Stroop test significantly predicted greater treatment response to either CBT or fluoxetine. More specifically, higher verbal IQ was more predictive of response to CBT with a smaller influence in response to fluoxetine, whereas verbal memory was more predictive of response to fluoxetine than to CBT. Interestingly, the researchers reported that individuals with OCD who made fewer perseverative responses on the CVLT responded better to CBT, but not to fluoxetine. Subsequently the authors suggested that certain cognitive functions may be more advantageous in certain settings. For example, greater mental flexibility (represented in this study by fewer CVLT perseverations) may allow for more effective learning in CBT (D'Alcante et al., 2012). However, the association between neuropsychological markers of cognitive flexibility and CBT treatment response requires more research.

Studies focusing on neuropsychological changes pre- and post-treatment have yielded highly contrasting results. Voderholzer et al. (2013) found that OCD individuals demonstrated improvements in information processing, verbal and nonverbal fluency,

nonverbal memory, visuo-constructive ability, and set shifting ability following CBT. These improvements were observed regardless of treatment response levels. A study by Kuelz et al. (2006) found a significant improvement in nonverbal memory, set shifting, and self-guided behavior abilities for OCD patients after CBT. Furthermore, Moritz, Kloss, Katenkkamp, Birkenr, and Hand (1999) reported improvements on the WCST and Stroop tasks for CBT responders, but not for non-responders. For medication treatment studies, Kang et al. (2003) found that OCD patients significantly improved on the ROCF following SSRI treatment, and that this improvement was associated with brain metabolic changes. However, Nielen & den Boer (2003) did not report any such improvements after treatment with fluoxetine. Finally, several studies investigating the effects of combination treatments (i.e., CBT plus medication) found that OCD patients did not exhibit improvement on neuropsychological tests post-treatment (Bannon, Gonsalvez, Croft, & Boyce, 2006; Kim et al., 2002; Roh et al., 2005). These mixed results further obscure any resolution in the debate on whether neuropsychological abilities in OCD are trait- or state-like in nature (see below for a detailed discussion). Notably, for such research designs, learning effect between the two administrations may pose an important challenge that may contribute to the contrasting nature of these findings.

Importantly, it is essential to take into account information regarding objective impairments on neuropsychological tasks at baseline (compared to test norms). This factor is not discussed in most of the studies mentioned above. Improvement may be within the normative range of performance, or alternatively, an improvement that represents transition from impaired performance to normative performance. Thus, more research is necessary to fully understand the association between neuropsychological test performance and OCD treatment response, including evaluation of objective levels of performance.

7. Endophenotypes

Endophenotypes are internal and quantifiable components that mediate genes and behavioral/clinical phenotypes in psychiatric disorders (Gottesman & Gould, 2003; Gould & Gottesman, 2006). Endophenotypes can be predominantly neuropsychological or neurobiological in nature, and help identify both the traits of clinical phenotypes and the consequences of gene expression in specific disorders (Gottesman & Gould, 2003). While biomarkers encompass biological features associated with specific conditions, psychiatric endophenotypes are distinguished by five specific criteria: association with a mental disorder, heritability, state-independence (occurring within an individual whether or not he or she displays the disorder), co-segregation between the endophenotype and illness within families, and presence in unaffected relatives at a higher rate than in the general population (Gottesman & Gould, 2003; Gould & Gottesman, 2006). Endophenotypes provide an avenue for broadening, and potentially challenging, current classification systems (Lilienfeld, 2014) to encompass genetic and biological correlates of various disorders and constructs. However, it has been argued that endophenotypes may not be more genetically informative than traditional exophenotypes (Flint & Munafò, 2007). Nevertheless, attending to some important challenges, endophenotype research has potential to aid in identifying genetic markers and their association with clinical presentation (Lilienfeld, 2014).

A number of neurobiological and neuropsychological constructs have been proposed as candidate endophenotype markers of OCD (Taylor, 2012). Chamberlain et al. (2008) used fMRI to measure brain activation in OCD patients and unaffected relatives during reversal learning tasks. The authors reported hypoactivation in the OFC in both groups, and suggested that OFC hypofunction related to reversal learning, may be a sound candidate endophenotype for OCD (Chamberlain et al., 2008). Similarly, cognitive inflexibility and deficient motor inhibition have been found in OCD samples and their unaffected relatives, providing evidence that these deficits may also be appropriate candidate endophenotypes (Chamberlain et al., 2007). Moreover, nonverbal memory deficits were present in both recovered OCD patients and matched healthy controls, suggesting that these deficits are state independent (Rao et al., 2008), and decreases in visuospatial working memory, resulting from feelings of uncertainty, have been suggested as a marker of top-down processing deficits in OCD individuals (Lambrecq et al., 2014). However, as detailed below, the endophenotype approach in OCD research has been subject to controversy pertaining to the state versus trait debate, particularly concerning neurocognitive markers.

8. Controversies in OCD neuropsychological research

8.1. Response inhibition

RI has been recently subject to controversy among OCD researchers. This controversy revolves around two main themes: the construct and ecological validity of RI in OCD (i.e., its traditional association with behavioral impulsivity), and RI's role in the etiology of the disorder. As other challenges pertaining to the examination of this construct in OCD were detailed in previous sections (e.g., factors associated with task selection, specificity), this section will focus on the two aforementioned themes.

Behavioral impulsivity is a complex non-unitary construct, particularly when considering neuropsychological and neurobiological correlates (Bari & Robbins, 2013); and inhibition is considered to be an aspect of impulsivity according to contemporary theoretical models (Bari & Robbins, 2013). Some researchers maintain that OCD is a disorder of deficient stopping, especially in the context of deficient performance on the SST found in OCD samples (Chamberlain et al., 2005; Penades et al., 2007; Purcell et al., 1998b). Individuals with OCD can perform a ritual that may include numerous repetitions (e.g., washing hands, repeatedly checking the stove) and experience an overflow of obsessive thoughts that are difficult to 'stop'. However, compulsive rituals are carefully planned and timed, and are frequently governed by complex rules (such as 'do X while not doing Y'; Boyer & Lienard, 2006). For example, it is not uncommon for patients with OCD to be able to postpone performance of rituals for prolonged periods of time. Moreover, there is evidence that OCD is characterized by increased restrained behavior/behavioral inhibition (Coles, Schofield, & Pietrefesa, 2006), and is associated with increased premeditation (Zermatten & Van der Linden, 2008). Research further indicates that when impulsivity is found among OCD patients, it stems from the presence of impulsivity-related comorbid disorders (Kashyap et al., 2012).

Overall, there is strong evidence that OCD is not associated with the behavioral manifestation of disinhibition, deficient control of behavior, or impulsivity (McKay, 2014). Indeed, impulsivity and compulsivity may lie on different ends of a continuum in terms of neural substrates, pharmacotherapy, behavioral manifestation, and phenomenology (Hollander, 2005). However, as mentioned above, research indicates deficient response inhibition in OCD when assessed by the SST. It is important to note, however, that the SST assesses *action cancellation*, while the vast majority of studies assessing *action suppression* (using GNG tests) do not report deficits in OCD samples. In fact, a recent principal component analysis conducted by Broos et al. (2012) found that self-reported impulsivity did not load onto the same factor as the SST.

Another study demonstrated that while individuals with OCD exhibited response inhibition deficits compared to controls, self-reported behavioral impulsivity was comparable between groups (Abramovitch, Dar, Hermesh, & Schweiger, 2012). Thus, it appears that OCD may be associated with deficient performance on tasks of action cancellation, but not action suppression, and that these deficits in OCD may be dissociated from behavioral impulsivity. Indeed, one recent study that conducted a multidimensional assessment of these constructs in OCD utilizing tasks assessing RI and risk taking found that individuals with OCD exhibited deficient performance on the SST but at the same time exhibited reduced risk taking compared to controls (Sohn, Kang, Namkoong, & Kim, 2014). None of the tasks were associated with OCD symptoms, however. Thus, while this is an encouraging first step, the specificity of these putative deficits in OCD remains a major limitation of current research. In addition, researchers are encouraged to attend to the dissociation between RI and behavioral impulsivity that may be uniquely pronounced in OCD (Abramovitch et al., 2012).

A second controversy associated with RI pertains to its role in the etiology and maintenance of OCD. Some researchers argue for the role of RI in the etiology of OCD, suggesting that RI deficits may an important part in the development and maintenance of OCD, particularly with regards to compulsions (Harsányi et al., 2014; Linkovski, Kalanthroff, Henik, & Anholt, 2013). However, as noted by Abramovitch and Abramowitz (2014), this preliminary hypothesis suffers from some fundamental flaws. First, given the nature of neuropsychological research, it is nearly impossible to determine whether RI deficits cause OCD, are caused by having OCD, or are caused by a latent third factor. Second, such etiological hypothesis suffers from a specificity problem. That is, given that other disorders (e.g., ADHD, schizophrenia, etc.) exhibit RI deficits, what would determine whether RI deficits lead to OCD or other conditions? Indeed, the neglected question about the specificity of neuropsychological deficits OCD deserves more research attention, particularly in cases where etiological hypotheses are offered.

8.2. State versus trait

The endophenotype approach to neurocognitive deficits assumes, a priori, that neurocognitive deficits are trait features of a given psychiatric disorder. In fact, as mentioned above, one of the criteria for endophenotypes is that they should be state independent (Gottesman & Gould, 2003). State dependent factors, such as symptom severity, thus should not have a significant impact on neuropsychological test performance in OCD patients, especially in tests assessing constructs suggested as OCD endophenotypes. For example, if a neurocognitive deficit is an endophenotype of OCD, successful OCD treatment should not impact an individual's performance on tasks assessing that function. Alternatively, the neuropsychological deficit would be demonstrated in both OCD and remitted patients.

Although the results are contrasting, the reported improvement on some neuropsychological tests following successful treatment (D'Alcante et al., 2012; Nielen & Den Boer, 2003; Voderholzer et al., 2013) challenges the notion of state-independence of neuropsychological deficits in OCD. Other studies that have demonstrated deficient neuropsychological functioning in remitted patients with OCD (Bannon et al., 2006), as well as unaltered neuropsychological test performance following treatment (Kim et al., 2002; Nielen & Den Boer, 2003; Roh et al., 2005) are in support of the trait hypothesis. However, it is important to remember that in nearly every cognitive domain, a significant body of literature demonstrates only small to moderate degrees of deficits, some of which would be considered within the rate of normative performance. Thus, a significant change may not be expected following successful treatment. Moreover, given that neuropsychological test performance is thought to reflect brain abnormalities, the changes in patterns of brain activity among treatment responders with OCD (Nakao et al., 2005; Saxena et al., 2009) should theoretically accompany changes in neuropsychological tests performance. This dissociation between neurobiological and cognitive processes in OCD deserves more research attention.

Research reporting associations between neuropsychological test performance and OCD symptom severity may also play a role in the controversy over the state dependence of neuropsychological deficits (Abramovitch et al., 2011; Moritz et al., 2012). However, only a minority of studies report an association between OC symptom severity and neuropsychological test performance (Abramovitch et al., 2011, 2012; Kitis et al., 2007; Lucey et al. 1997; Nedeljkovic et al., 2009; Penades et al., 2005; Segalas et al., 2008; Trivedi et al., 2008). The variety of measures for OCD severity and the high probability of a restricted range may hinder the identification of an association between neuropsychological test performance and OCD severity. Indeed, some studies did not focus on traditional measures of OCD severity and instead experimentally manipulated levels of meta-cognitive symptoms (e.g., ruminations) and cognitive biases (e.g., overestimation of threat) related to OCD. Results from these studies demonstrate how state dependent increases in symptom severity partially mediate cognitive dysfunctions in OCD, compared to depressive, social phobic, and non-psychiatric control samples (Exner, Kohl, et al., 2009; Exner, Martin, et al., 2009; Exner, Zetsche, Lincoln, & Rief, 2014; Zetsche, Rief, Westermann, & Exner, 2014).

To account for the impact of state-related increases in intrusive and obsessive thoughts, Abramovitch et al. (2012) offered the Executive Overload Model of OCD. This model highlights a process in which the (state dependent) overflow of obsessive thoughts overloads the executive system in a way which is similar to having numerous open programs on a personal computer that overloads the RAM memory and cause the primary program to operate more slowly. This overload may then result in neuropsychological deficits in OCD (Abramovitch et al., 2012). This model corresponds to other models demonstrating the impact of anxiety on cognitive performance (Eysenck, Derakshan, Santos, & Calvo, 2007). Together, these models challenge the endophenotype/pure trait-like nature of neuropsychological deficits in OCD.

In sum, research support exists for both sides of this controversy, and more research is required to determine whether neuropsychological underperformance is state dependent or a trait of OCD. Notably, a third option exists where a moderate general reduction in cognitive capacity may be an OCD trait. This in turn may make individuals with OCD more susceptible to state related factors (Kalanthroff, Anholt, & Henik, 2014), producing an acute increase in obsessive thought overflow, as suggested by the Executive Overload Model. However, evidence for such a trait+-state hypothesis in OCD is lacking, and its specificity to OCD is questionable. Indeed, a moderate degree of cognitive deficits may be associated with having a psychopathology in general (Caspi et al. 2013) and may be exacerbated by state related psychopathological factors (e.g., stress, anxiety).

8.3. Impairments versus underperformance

A qualitative review of neuropsychological literature usually focuses on null hypothesis testing of differences between groups (e.g., differences in task performance between OCD and non-psychiatric control samples). On the other hand, a quantitative review (i.e., meta-analysis) transcends the significance of various results and focuses on estimating effect sizes to exemplify the magnitude of differences across studies. However, both methodologies do not take into account the association between underperformance on neuropsychological tests and the degree of clinical/functional impairments.

For example, it is unknown whether a person with OCD exhibiting a moderate-severe deficit on a non-verbal memory test would present functional impairments that differ from an individual with OCD who does not exhibit this deficit. In fact, it is possible that the small to moderate degree of underperformance on neuropsychological tests among individuals with OCD, is not associated with clinically significant impairments. Unfortunately, no available research has yet examined functional correlates of neuropsychological test performance in OCD. Such research would include examination of the association between neuropsychological functions and activities of daily living, vocational and academic functioning.

Abramovitch et al. (2013) recently offered a novel perspective about whether neuropsychological deficits in OCD may be considered as clinically significant impairments versus underperformance in the normative range. The authors described how Cohen's *d* effect sizes are equivalent to standard deviation difference between groups. For example, the overall effect size for executive functioning in their study was 0.5, which indicates that OCD participants performed half a standard deviation below controls on tests of executive function. However, the cutoff for clinically significant impairment is considered to be performance of two or more standard deviations below controls/test norms (Lezak et al., 2012). In fact, some researchers suggest that an effect size greater than 3.0 is the most appropriate cutoff in establishing neuropsychological test markers (Zakzanis, 2001). It should be noted, however, that a more liberal heuristic for defining meaningful neuropsychological impairments is suggested to be a 1 standard deviation difference (Taylor & Heaton, 2001). Nevertheless, half a standard deviation – the average difference found in this meta-analysis for neuropsychological underperformance among OCD samples – would clearly be considered underperformance within the normative range, and not a significant impairment. In fact, all effect sizes calculated for neuropsychological test performance in OCD are well below 1.0 (Abramovitch et al., 2013; Shin et al., 2014).

For some studies that found reduced task performance among OCD samples compared to controls, authors often concluded that OCD is associated with "significant impairments", despite participants' performance being within a normative range when compared to population norms. Similarly, a recent meta-analysis of executive functions in OCD, concluded that there are "broad impairments" in executive function in this population, even though all effect sizes ranged between only small to medium (Snyder, Kaiser, Warren, & Heller, 2014). Additionally, as noted above, there is a lack of research examining ecologically valid everyday functional correlates of the hypothesized impairments. Such associations have been reported in the context of executive function tests across some neurological and psychiatric populations, particularly when utilizing informant ratings (Chaytor & Schmitter-Edgecombe, 2003).

Overall, the impairment versus underperformance controversy further emphasizes the need for utilization of ecologically valid functional measures to assess the validity and nature of any hypothesized cognitive impairment. Recent attempts to assess the magnitude of underperformance in OCD are a promising first step, potentially leading to a much needed integration of cognitive neuroscience with clinical and behavioral aspects of psychiatric disorders. This type of integration would be especially important in OCD, given the complexity of neuropsychological findings.

9. Summary and future directions

The neuropsychological literature in OCD may be the most divergent across psychiatric disorders. This inconsistency, identified more than a decade ago (Greisberg & McKay, 2003; Kuelz , Hohagen, et al., 2004), seems to persist regardless of the more sophisticated methodologies and increasing awareness of potential confounding factors. Meta-analytic investigations have demonstrated that OCD may be associated with small to moderate degrees of underperformance on a wide range of neuropsychological tests involving most domains (Abramovitch et al., 2013; Shin et al., 2014). However, some fundamental questions remain controversial, such as whether performance on neuropsychological tasks is considered underperformance in the normative range or a clinically significant impairment. Furthermore, it is still unclear how neuropsychological underperformance integrates into psychological models of OCD. Other issues remain unanswered, including the specificity of cognitive dysfunction, their association with functional impairment, and whether neuropsychological deficits are state dependent or a trait of OCD.

Neuropsychological test performance remains an informative and objective means of investigation, especially when applied to psychiatric disorders, where research and treatment rely heavily on self-report measures. However, because research has used the same classic neuropsychological tests, with the vast majority of studies employing the same classic study design, we propose that different analytical perspectives and modifications of existing tests are warranted. This, in turn, may yield more informative findings, and may assist in resolving some of the controversies in the field. For example, there are indications that, compared to controls, individuals with OCD may be more susceptible to increased cognitive load and subsequently underperform on tests that present more difficulty or complexity (e.g., Kashyap et al., 2013). It would be important to examine the type of tasks and associated domains where individuals with OCD may be more susceptible to task load.

It may also be useful to consider modifying existing tasks. For example on CPT and GNG tests, researchers may manipulate the nature of the go and no-go stimuli (e.g., disorder-relevant images), alter inter-stimulus intervals, and percent no-go stimuli that has been shown to impact exertion of control (Kalanthroff et al., 2014). Such manipulations, including the addition of blocks that include distractors, may allow researchers to control for the effects of error monitoring, processing speed, excess control, and distractibility. This would also potentially aid in discovering more subtle and disorder-specific deficits, facilitating their incorporation into current psychological models of OCD.

Another means to advance the field of neuropsychology of OCD is to examine neuropsychological function using ecologically valid measures, and to assess the association between the former and clinical and functional indices. For example, one could test the hypothesis that response inhibition deficits are associated with difficulty inhibiting an ongoing ritual by formulating a study design whereby participants are required to stop or delay an ongoing ritual while assessing clinical, neuropsychological, and psychophysiological correlates. In addition, given the recognized impairment on the ROCF in OCD, it would also be useful to assess non-verbal memory for real life items in the lab or in a virtual reality environment.

In general, it is recommended that neuropsychological research in OCD progress to more theory-driven hypotheses rather than employing large test batteries to explore group differences on executive functions or other neuropsychological domains. With the aim to solidify neurobiological models, the traditional neuropsychological study design inherently has difficulties resolving the current challenges in the field of OCD. This type of research is already emerging in other disorders allowing identification of more specific cognitive deficiencies (e.g., Elshaikh, Sponheim, Chafee, & MacDonald Iii, 2014). It is also recommended that researchers carefully select appropriate tasks, ascertaining the specific construct validity of each of the measures in order to increase their ability to assess more specific hypotheses. In

addition, researchers in the field are encouraged to take into account the vast body of neuropsychological literature in OCD in order to address limitations and potential confounding factors highlighted in previous research. These include for example age of onset, depressive severity, and medications, but also the issue of corrections for multiple comparisons. In addition, future studies are encouraged to address the magnitude of underperformance, the state versus trait controversy, and issues pertaining to specificity. The latter may be achieved by comparing OCD samples to psychiatric control groups, facilitating our understanding of disorder-specific deficiencies.

We strongly believe that these directions for research would contribute to our understanding of the neuropsychology of OCD. However, in order to move the field of neuropsychology of OCD forward, interdisciplinary collaborations are encouraged (Ahmari, Eich, Cebenoyan, Smith, & Blair Simpson, 2014), particularly amongst OCD researchers that operate in a field where there seems to be a noteworthy (but potentially bridgeable) gap between psychological, neurobiological and neuropsychological models.

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