

REVIEW

Does neuropsychological performance in OCD relate to different symptoms? A meta-analysis comparing the symmetry and obsessing dimensions

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Abstract

Background: Investigations of neuropsychological functioning in obsessive-compulsive disorder (OCD) have produced mixed results for deficits in executive functioning (EF), attention, and memory. One potential explanation for varied findings may relate to the heterogeneity of symptom presentations, and different clinical or neurobiological characteristics may underlie these different symptoms.

Methods: We investigated differences in neuropsychological functioning between two symptoms groups, obsessing/checking (O/C) and symmetry/ordering (S/O), based on data suggesting an association with different motivations: harm avoidance and incompleteness, respectively. Ten studies (with 628 patients) were included and each investigation assessed at least one of 14 neuropsychological domains.

Results: The S/O domain demonstrated small, negative correlations with overall neuropsychological functioning, performance in EF, memory, visuospatial ability, cognitive flexibility, and verbal working memory. O/C symptoms demonstrated small, negative correlations with memory and verbal memory performance. A comparison of functioning between symptom groups identified large effect sizes showing that the S/O dimension was more strongly related to poorer neuropsychological performance overall, and in the domains of attention, visuospatial ability, and the subdomain of verbal working memory.

Conclusions: Findings support existing evidence suggesting that different OCD symptoms, and their associated core motivations, relate to unique patterns of neuropsychological functioning, and, potentially dysfunction in different neural circuits.

KEYWORDS

anxiety, attention, executive function, memory, meta-analysis, neuropsychology, obsessive-compulsive disorder

1 | INTRODUCTION

Neuropsychological tests may provide valuable information about underlying neurobiological processes (Abramovitch & Cooperman, 2015). There exists a large body of neuropsychological literature on obsessive-compulsive disorder (OCD), and yet results from this literature are highly mixed (Abramovitch, Abramowitz, & Mittelman, 2013). There is some evidence to suggest the presence of specific deficits compared to healthy individuals, including response inhibition and memory (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian,

2005). However, findings are mixed and a recent meta-analysis concluded that the significance of any findings is limited without taking into account the heterogeneity of clinical presentation (Abramovitch et al., 2013). Indeed, the heterogeneity in findings across studies may be due, at least in part, to differences in OCD symptom presentation across studies. However, almost half of the existing neuropsychological investigations in OCD do not take different symptom dimensions into consideration (Abramovitch, Mittelman, Tankersley, Abramowitz, & Schweiger, 2015). A growing body of evidence suggests that different symptom dimensions may relate to varying clinical characteristics,

neural mechanisms, patterns of neuropsychological performance, and treatment response (Mataix-Cols, do Rosario-Campos, & Leckman, 2005). Authors of a recent meta-analysis took this heterogeneity into account by comparing neuropsychological functioning in discrete OCD groups of “washers” versus “checkers” (Leopold & Backenstrass, 2015). Their results indicated that checkers were more impaired than washers in eight out of 10 cognitive subdomains. Deficits in three of these domains showed moderate (set-shifting) to large (planning/problem solving; response inhibition) effect sizes, whereas sustained attention, encoding strategies, verbal memory, nonverbal memory, and processing speed demonstrated small effect sizes (Leopold & Backenstrass, 2015). These findings underscore the importance of continuing to study the association between OCD symptom dimensions and cognitive functioning.

Factor and cluster-analytic investigations using the Yale-Brown Obsessive Compulsive Scale Symptom Checklist (Y-BOCS SC) (Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989) and Obsessive-Compulsive Inventory-Revised (Foa et al., 2002) have reliably identified specific symptom groups, with the most consistent categories consisting of symmetry/ordering (S/O), harming/checking, and contamination/washing (C/W) (Baer, 1994; Calamari et al., 2004; Leckman, Grice, Boardman, & Zhang, 1997; Mataix-Cols, Junqué et al., 1999; Summerfeldt, Richter, Antony, & Swinson, 1999). Obsessions and compulsions regarding symmetry, arranging, and ordering are common in OCD and exist in approximately 57% of patients (Mataix-Cols, Rauch, Manzo, Jenike, & Baer, 1999; Pinto et al., 2008; Rasmussen & Eisen, 1992). Symptoms may include a preoccupation with arranging items on a desk until they are equal distances apart. Although obsessions in OCD are often focused on some type of feared outcome (e.g., checking the stove to prevent a fire), individuals with OCD who report obsessions related to symmetry often have trouble articulating a feared outcome (Starcevic & Brakoulias, 2008). Indeed, S/O have been found to differ from other OCD symptoms in that they are less closely tied to harm avoidance (HA) and more often associated with incompleteness or “not-just-right experiences” (NJREs; Coles, Pietrefesa, Schofield, & Cook, 2008; Summerfeldt, Kloosterman, Antony, & Swinson, 2014). As opposed to symptoms driven by a fear to avoid harm, incompleteness-related symptoms are driven by the motivation to reduce discomfort, and this phenomena is often compared to premonitory urges seen in tic disorders (Leckman, Walker, Goodman, Pauls, & Cohen, 1994; Rasmussen & Eisen, 1990; Summerfeldt et al., 2014). Research has consistently shown a connection between the S/O symptom dimension and incompleteness (Bragdon & Coles, 2017; Coles, Frost, Heimberg, & Rhéaume, 2003; Summerfeldt, Antony, & Swinson, 2000; Taylor et al., 2014). In both clinical and nonclinical investigations, levels of incompleteness have been shown to uniquely predict S/O symptoms (Pietrefesa & Coles, 2008; Radomsky & Rachman, 2004).

Obsessing/checking symptoms (O/C) include obsessions related to harm, aggression, taboo topics, and checking compulsions. Obsessions may pertain to concern over harming the self or others, such as by hitting someone with a car, or going to hell for having blasphemous thoughts (Williams et al., 2014). Checking behaviors are the most common compulsions seen in individuals with OCD (Mataix-Cols, Baer,

Rauch, & Jenike, 2000). Symptoms in this dimension, as opposed to other dimensions such as C/W, have shown to be unique predictors of the other core motivational domain, HA (Ecker & Gönner, 2008). HA pertains to a perception of potential threat and the corresponding desire to avoid this threat. The HA dimension shares features with other anxiety disorders (Starcevic, Uhlenhuth, Fallon, & Pathak, 1996), is more consistent with traditional cognitive behavioral models of OCD (Ecker & Gönner, 2008), and has traditionally been the focus of most OCD research (Summerfeldt et al., 2014). C/W symptoms, unlike the S/O and O/C dimensions, do not clearly map onto the specific motivations of HA and incompleteness. Indeed, studies show that C/W symptoms, compared to the other symptom dimensions, can be motivated by both HA, incompleteness, or a combination of both and relate more to disgust avoidance (Mancini, Gagnani, & D'Olimpio, 2001; Taylor et al., 2014). In the meta-analysis by Leopold & Backenstrass (2015), washers performed significantly better than checkers in the majority of domains examined, and therefore C/W symptoms may be less related to neuropsychological functions than other OCD symptoms.

A number of studies suggest that the symptom dimensions of S/O and O/C vary widely in clinical characteristics. In addition to showing unique associations with different core motivations (Ecker & Gönner, 2008), they have shown to be related to differential patterns of comorbidity, age of onset, and treatment response (Coles, Pinto, Mancebo, Rasmussen, & Eisen, 2008; Hasler et al., 2005; Jenike, Baer, Minichiello, Rauch, & Buttolph, 1997; Landeros-Weisenberger et al., 2010; Leckman et al., 2010; Matsunaga et al., 2008; Rück, Larsson, & Mataix-Cols, 2012; Stein, Andersen, & Overo, 2007; Stewart et al., 2004). The mixed clinical presentation and treatment outcomes across symptom domains may suggest different underlying mechanisms (Rasmussen et al., 2013). Indeed, genetic investigations have demonstrated unique associations with variants of particular neurotransmitter receptor and transporter genes (Lochner et al., 2016; Viswanath et al., 2013). Neuroimaging studies offer evidence suggesting S/O and O/C dimensions may relate also to different structural and functional patterns. In one structural neuroimaging investigation, bilateral temporal lobe gray matter and white matter volume were positively correlated with the scores on the S/O dimension and negatively correlated with scores on the O/C dimension (van den Heuvel et al., 2009). In a positron emission tomography (PET) study using a continuous performance task, O/C symptoms were positively correlated with activation in the bilateral striatum, whereas the S/O dimension was negatively associated with activation in the right striatum only (Rauch et al., 1998).

What remains unclear from previous research, however, is how S/O compared to O/C symptoms may be related to differences in cognitive functioning. The aim of this article is to provide a current and systematic review of the neuropsychological performance in the OCD symptom dimensions of S/O and O/C and reconcile some of the inconsistencies in the neuropsychological literature. These two dimensions were chosen not only due to their prevalence, but their unique associations with different O/C core motivations and clinical characteristics. Our hope was that identifying possible neuropsychological differences between individuals endorsing specific symptoms could contribute to our understanding of the pathological mechanisms and ultimately inform treatment.

2 | METHODS

2.1 | Study selection

Study selection was based upon the following criteria: (a) adult sample; (b) diagnosis of OCD according to the International Classification of Diseases, ninth revision (ICD-9) OR 10th revision (ICD-10) OR the Diagnostic and Statistical Manual of Mental Disorders, third edition (DSM-III), fourth edition (DSM-IV) OR fifth edition (DSM-V); (c) one or more measures of neuropsychological performance; and (d) data reported could be converted to effect sizes. Studies were excluded based on the following criteria: (a) sample size less than 10, (b) S/O and O/C symptoms were not specified, (c) studies with subclinical samples, (d) studies that included patients with OCD due to a general medical factor), and (e) studies only providing graphical data. No specific criteria were set regarding number of symptom domains endorsed or specification of primary versus secondary symptoms.

2.2 | Search strategy

Literature review was conducted between May 2016 and March 2017, according to the criteria outlined in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Statement (Moher et al., 2009). Database search included PsychInfo, MEDLINE, PubMed, Google Scholar, ProQuest Dissertations & Theses, using the search terms ("symmetry*" OR "order*" OR "arranging*" OR "obsess*" OR "check*" OR "obsessive compulsive" OR "OCD") AND ("neuropsych*" OR "neurocogn*" OR "information processing" OR "memory" OR "attention" OR "executive" OR "set shifting" OR "cognitive *function" OR "cognitive deficit" OR "frontal *function" OR "frontal deficit"). Lastly, reference lists of articles were reviewed.

2.3 | Data extraction

The following variables were recorded: (a) publication year, (b) country of origin, (c) sample size, (d) percent females, (e) mean intelligence scores, (f) mean years of formal education, (g) mean OCD duration, (h) mean age of OCD onset, (i) percent of the sample taking psychotropic medication, (j) percent of the sample with comorbid condition, (k) mean severity scores for OCD, depression, and anxiety, (l) percent of the sample with a comorbid tic disorder diagnosis, and (m) neuropsychological variable statistics.

2.4 | Data analysis

Neuropsychological measures were organized into domains based on previous studies (Abramovitch et al., 2013; Leopold & Backenstrass, 2015; see Table 1). The effect size r was calculated from means, standard deviations, and Pearson's product moment correlations (Rosenthal, Rosnow, & Rubin, 2000). In two cases, only standardized (β) regression coefficients were reported. These data were converted to Pearson's r correlation coefficients using the formula ($r = \beta + .5\lambda$) outlined by Peterson and Brown (2005). In one study, only unstandardized beta coefficients and standard error were reported. These were subsequently converted to t -scores ($t = B/SE$)

TABLE 1 Overview of included cognitive domains and neuropsychological tests

Domain	Subdomain	Tests
Attention	Sustained attention	Go/No-Go (omission errors); CTT (part 1)
Executive functions	Decision-Making	IGT (total net score, disadvantageous card selections); CGT (rational decisions)
	Planning/problem-solving	WASI matrix reasoning; RCFT (copy time, organization); TOH
	Response inhibition	Color word Stroop (interference); Go/No-Go commission errors
	Cognitive flexibility	WCST (perseverative errors, categories completed); WASI (similarities); CTT (part 2); letter fluency; category fluency; TMT (subtracted score); COWA (words generated, switches); OAT (trials taken to criterion, perseverative errors); five-point test
Memory	Verbal memory	WMS-R LM (I, II); AVLT (total words recalled, immediate recall, delayed recall)
	Nonverbal memory	RCFT (delayed recall, immediate recall, recall time); CANTAB DMS
Visuospatial ability	Visuospatial ability	WASI Block design; RCFT copy; BGT (number of errors)
Working memory	Visuospatial working memory	CANTAB (SWM strategy and errors, SRM); WMS spatial span
	Verbal working memory	WAIS-III digit span; LNS; WMS digit span

Note. AVLT: Auditory Verbal Learning Test; BGT: Bender-Gestalt Test; CANTAB: Cambridge Automated Neuropsychological Test Battery; CGT: Cambridge gambling task; COWA: Controlled Oral Word Association Test; CTT: Color Trails Test; DMS: delayed matching to sample; IGT: Iowa gambling task; LNS: letter number sequencing; OAT: object alternation task; RCFT: Rey-Osterrieth Complex Figure Test; SRM: spatial recognition memory; SWM: spatial working memory; TMT: Trail-Making Test; TOH: Tower of Hanoi; WAIS-III: Wechsler Adult Intelligence Scale—third edition; WASI: Wechsler Abbreviated Scale of Intelligence; WCST: Wisconsin Card Sorting Task; WMS-R LM: Wechsler Memory Scale-Revised Logical Memory.

and then Pearson's r , Spearman's rho and Kendall's tau correlations were provided in two studies and were converted to Pearson's r correlation coefficients using the formulas outlined in Rupinski and Dunlap (1996).

Fisher's z -transformations were used to combine correlational data (Silver & Dunlap, 1987). A composite effect size was calculated for studies reporting more than one outcome per neuropsychological domain and variance was corrected using the methods outlined in Borenstein, Hedges, Higgins, and Rothstein (2009). Correlations between neuropsychological outcomes and symptom groups in each study were compared using Steiger's Z -test of dependent correlations (Steiger, 1980) and these Z -scores were then included in the between-groups meta-analysis. The studies included in this current meta-analysis did not provide data on the intercorrelations between symptom subscales, and therefore values were taken from existing literature to use as the dependent correlations: obsessive compulsive

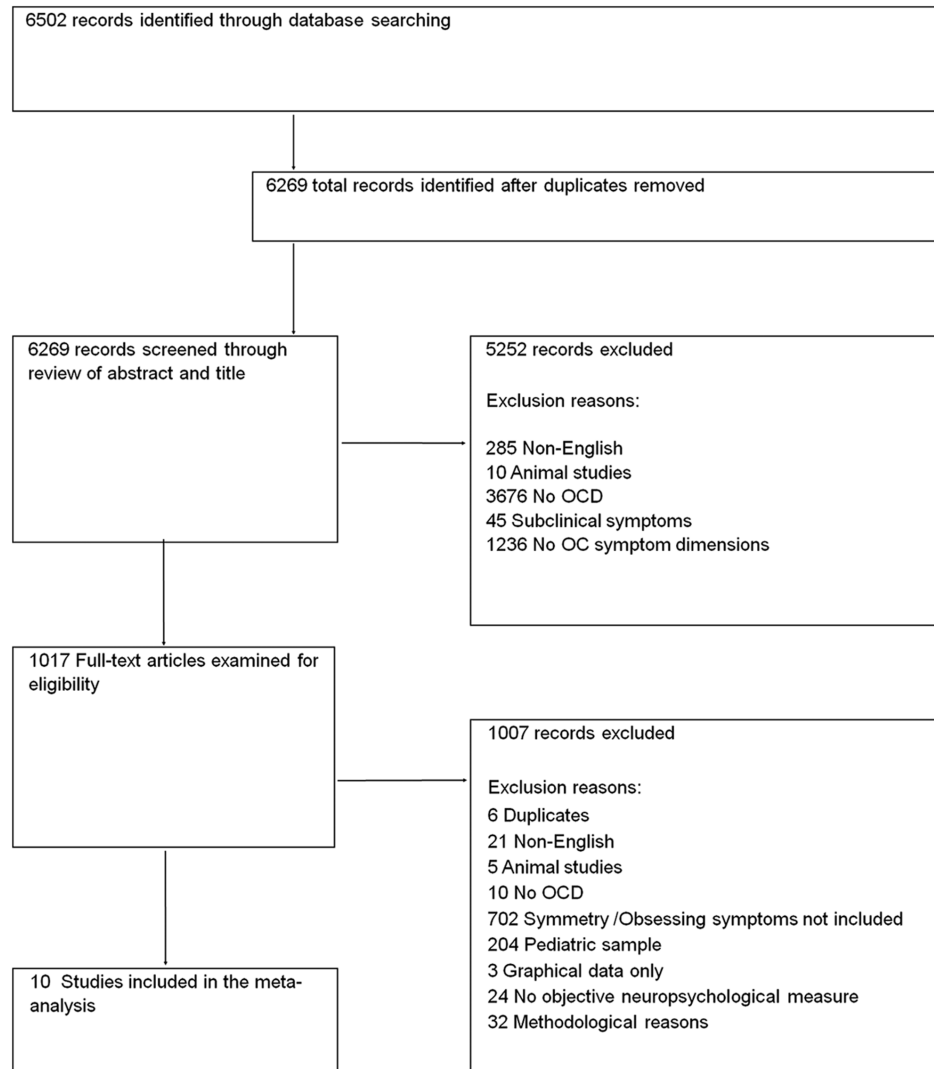


FIGURE 1 Search strategy used for study selection

inventory-revised (OCI-R) $r = .14$ (Ecker & Gönner, 2008); Dimensional Yale-Brown Obsessive Compulsive Scale (DY-BOCS) $r = .05$ (Pertusa, Fernández de la Cruz, Alonso, Menchón, & Mataix-Cols, 2012); Y-BOCS $r = .12$ (Sulkowski et al., 2008). Effect sizes were interpreted as small ($.10 \leq r < .30$), medium ($.30 \leq r < .50$), and large ($r \geq .50$; Cohen, 1988). Random-effects models were used to calculate a combined effect size across the studies and separately within cognitive subdomain. Q and I^2 statistics were calculated to assess heterogeneity. Publication bias was assessed by calculation of Egger's regression intercept examination of the funnel plot and calculation of Orwin's fail-safe N (Orwin, 1983).

If deemed necessary, the trim-and-fill method was used to adjust for publication bias (Duval & Tweedie, 2000). Meta-regression was used to investigate the influence of moderator variables. Variables included symptom severity (Y-BOCS score), percent of patients on medication, sex, country, percent of patients with a comorbid psychiatric diagnosis, percent of patients with a comorbid tic disorders diagnosis, and type of measure used to determine symptom dimensions. Due to missing data from studies, we were not able to include depression severity, or age. Analyses were conducted using Comprehensive Meta-Analysis 3.0.

3 | RESULTS

3.1 | Study selection

The search yielded 6,269 potentially eligible studies after the exclusion of duplicates. These were screened through review of the abstract and title, and 5,252 were excluded. The full text of these remaining 1,017 articles was reviewed and 10 studies were retained for comparison based on the previously established inclusion criteria. See Figure 1 and Table 2.

3.2 | Study characteristics

A total of 628 participants with OCD were included across studies. Eight studies reported correlation coefficients and two studies reported means and standard deviations.

3.2.1 | Country

Of the 10 studies, three were conducted in the United Kingdom (Dittrich, Johansen, Fineberg, & Inge Landrø, 2011; Dittrich, Johansen,

TABLE 2 Characteristics of the included studies

Study	Sample size	Diagnostic assessment	Cognitive domain
Dittrich, Johansen, Fineberg et al.(2011)*	S/O = 14 O/C = 31	DSM-IV, Y-BOCS	Decision-Making
Dittrich, Johansen, Inge Landrø et al. (2011)*	S/O = 14 O/C = 28	DSM-IV, Y-BOCS	Working memory
Hashimoto et al. (2011)	OCD = 63	DSM-IV, Y-BOCS	Response inhibition, cognitive flexibility, memory
Jang et al. (2010)	OCD = 144	DSM-IV, Y-BOCS	Problem-solving, spatial ability, memory
Kashyap et al. (2017)	OCD = 150	DSM-IV, Y-BOCS	Attention, decision-making, problem-solving, response inhibition, set-shifting, memory, working memory, visuospatial ability
Lawrence et al. (2006)	OCD = 39	DSM-IV, Y-BOCS	Decision-Making, cognitive flexibility
Martoni et al. (2015)	OCD = 42	DSM-IV, Y-BOCS	Working memory
Pedron et al. (2015)	OCD = 28	DSM-IV, Y-BOCS	Problem-Solving attention, response inhibition, cognitive flexibility, memory, working memory, visuospatial ability
Pinto et al. (2011)	OCD = 32	DSM-IV, Y-BOCS	Problem-Solving
Shin et al. (2012)	OCD = 85	DSM-IV, Y-BOCS	Cognitive-Flexibility

Note. O/C: primary obsessing/checking group; S/O: primary symmetry/ordering group. Studies marked with an asterisk * are categorical studies.

Inge Landrø, & Fineberg, 2011; Lawrence et al., 2006), two in South Korea (Jang et al., 2010; Shin et al., 2012), two in Brazil (Pedron et al., 2015; Pinto et al., 2011), one in Italy (Martoni, Salgari, Galimberti, Cavallini, & O'Neill, 2015), one in Japan (Hashimoto et al., 2011), and one in India (Kashyap, Kumar, Kandavel, & Reddy, 2017).

3.2.2 | Comorbidity

Two of the studies contained at least one participant with a co-occurring tic disorder diagnosis ($n = 1$, Shin et al., 2012; $n = 2$, Martoni et al., 2015) and two studies did not provide data on specific comorbidities within their sample (Pedron et al., 2015; Pinto et al., 2011). Five studies excluded participants with a co-occurring depressive disorder, three studies included individuals with a depressive diagnosis but did not provide data on the number of individuals, one study provided percent of the sample with comorbid depression (56.3%, Pinto et al., 2011), and one study did not provide any data on comorbid depression within the sample (Pedron et al., 2015).

3.2.3 | Symptoms

All studies used Y-BOCS or DY-BOCS to assess OCD severity. Symptom dimensions were assessed using the Y-BOCS Symptom Checklist ($n = 4$), DY-BOCS ($n = 5$), and Obsessive Compulsive Inventory—Revised (OCI-R; $n = 1$). Two of the studies categorized participants based on their current primary obsession and/or compulsion as assessed by the DY-BOCS (Dittrich, Johansen, Fineberg et al., 2011; Dittrich, Johansen, Inge Landrø et al., 2011).

3.3 | Neuropsychological functioning

3.3.1 | Correlations between symptom dimensions and cognitive functioning

Eight of the 10 studies reported correlational data and were included in within-group analyses. Combining results across these studies, S/O

symptoms demonstrated a small and significant negative correlation with overall neuropsychological functioning ($r = -.20, p < .01$). S/O symptoms were found to be associated with deficits in the domains of executive functions ($r = -.17, p < .05$), memory ($r = -.28, p < .05$), visuospatial ability ($r = -.14, p < .01$), and subdomains of cognitive flexibility ($r = -.28, p < .01$) and verbal working memory ($r = -.16, p < .05$). O/C symptoms demonstrated small, significant negative correlations with memory ($r = -.14, p < .05$) and verbal memory performance ($r = -.19, p < .05$), but not with the other domains. Effect sizes are shown in Figure 2.

3.3.2 | Comparison of cognitive performance between symptom dimensions

Effect sizes of S/O and O/C neuropsychological performance are shown in Figure 3. Positive effect sizes indicate that S/O performed better than O/C and negative effect sizes indicate that S/O performed worse than O/C. A significant, negative and large effect size was found for overall neuropsychological functioning ($Z = -.89, p < .05$), suggesting S/O symptoms were more strongly related to poor performance than O/C symptoms. Results also indicated that S/O symptoms were significantly more strongly associated with poorer performance compared to O/C symptoms in the domains of attention ($Z = -1.29, p < .01$), visuospatial ability ($Z = -1.11, p < .01$), and the subdomain of verbal working memory ($Z = -.80, p < .01$). There was a trend toward significance for differences between S/O versus O/C relations with cognitive flexibility ($Z = -1.10, p = .07$).

The S/O dimension was also associated with significantly poorer performance on four of the 40 outcomes in neuropsychological tasks after applying a Bonferroni correction. Large effect sizes were found for digit span ($n = 2, Z_p = -0.82, p < .00125$), Controlled Oral Word Association Test number of switches ($n = 1, Z = -2.67, p < .00125$), spatial working memory strategy ($n = 1, Z = -3.94, p < .00125$), and spatial recognition memory percent correct ($n = 1, Z = -3.92, p < .00125$). Following a Bonferroni correction, the

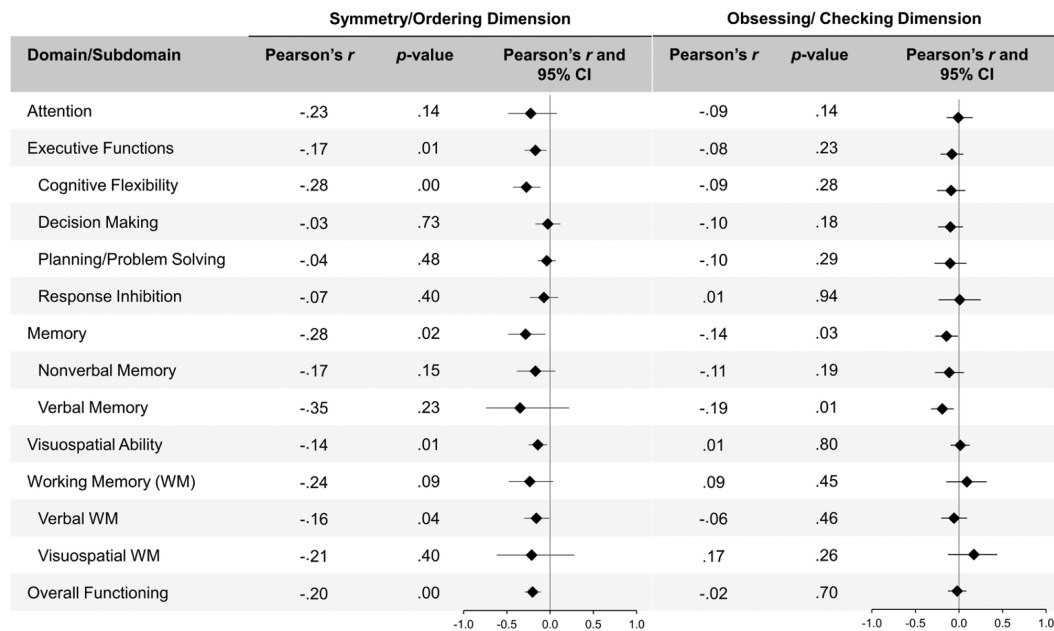


FIGURE 2 Forest plots of Pearson's *r* effect sizes for relation between symptom dimensions and neuropsychological functioning

O/C dimension was associated with significantly poorer performance on Auditory Verbal Learning Test delayed recall ($n = 1$, $Z = 3.31$, $p < .00125$).

Significant heterogeneity was found in all domains and subdomains except for attention and verbal working memory (See Table 3). The I^2 statistic, which measures variance explained by between-study heterogeneity, was between 39.70% and 99.47% for the domains and between 0 and 99.83% for the subdomains.

Meta-regression analyses were conducted to assess the influence of moderator variables on neuropsychological effect sizes. Due to missing data on duration and onset of OCD, educational level, age, and use of different measures of depressive and anxiety symptoms, analyses were not able to include these variables. Differences in OCD severity, country, symptom subtyping measure (Y-BOCS, DY-BOCS, OCI-R), percent of patients taking psychotropic medication, percent of patients with a comorbid psychiatric diagnosis, percent of patients with a comorbid tic disorder diagnosis, and sex distribution did not significantly contribute neuropsychological effect sizes.

3.4 | Publication bias

Assessing for publication bias is important in meta-analyses, especially when only a small number of studies are included. Risk of publication bias for the total effect across all 10 studies ($Z = -.89$, $p < .01$) was assessed by visual inspection of the funnel plot showing a slight asymmetry. After adjusting by means of the trim-and-fill method, the overall effect increased by a noticeable amount to $Z = -1.10$. Egger's regression intercept was not significant, $t(8) = 0.28$, $p = .79$. Orwin's fail-safe N was 38. Based on the 10 studies, almost three times as many unpublished studies with effect sizes greater than 0.10 would have been necessary to decrease the observed effect to a "trivial" size.

4 | DISCUSSION

The literature on neuropsychological deficits associated with OCD is substantial and seems to suggest certain patterns of deficits (e.g., impaired response inhibition; Chamberlain et al., 2005). However, findings are mixed, and these inconsistencies have been attributed to differences in symptom content (Abramovitch et al., 2013). Understanding the relations between cognitive performance and specific symptom phenotypes can provide valuable information about the underlying processes driving psychopathology and therefore inform treatment (Gottesman & Gould, 2003).

In the present study, we used meta-analytic techniques to investigate the relation between two common OCD symptom dimensions and neuropsychological performance with the goal of reconciling some of the inconsistencies identified in previous neuropsychological reviews (Abramovitch et al., 2013; Shin, Lee, Kim, & Kwon, 2014; Snyder, Kaiser, Warren, & Heller, 2015; Tallis, 2011; Watkins et al., 2005). Due to variability in associated clinical characteristics, including evidence demonstrating differential associations with core motivations (Bragdon & Coles, 2017; Pietrefesa & Coles, 2008; Summerfeldt et al., 2014), age of onset (Rosario-Campos et al., 2001), and patterns of comorbidity (Coles, Pinto et al., 2008), to name a few, we hypothesized that the S/O and O/C dimensions would show different patterns of performance. Our findings suggest that these dimensions may be associated with differential performance in the subdomains within executive functioning (EF). First, we found that higher levels of S/O symptoms were associated with worse overall neuropsychological performance. We also found that higher levels of S/O symptoms were associated with poorer performance in the domains of EF, memory, and visuospatial ability. Further, looking at even more specific abilities, poorer performance in the subdomains of cognitive flexibility and verbal working memory were also associated with increased S/O symptoms. In contrast, higher levels of O/C symptoms were associated with worse performance in

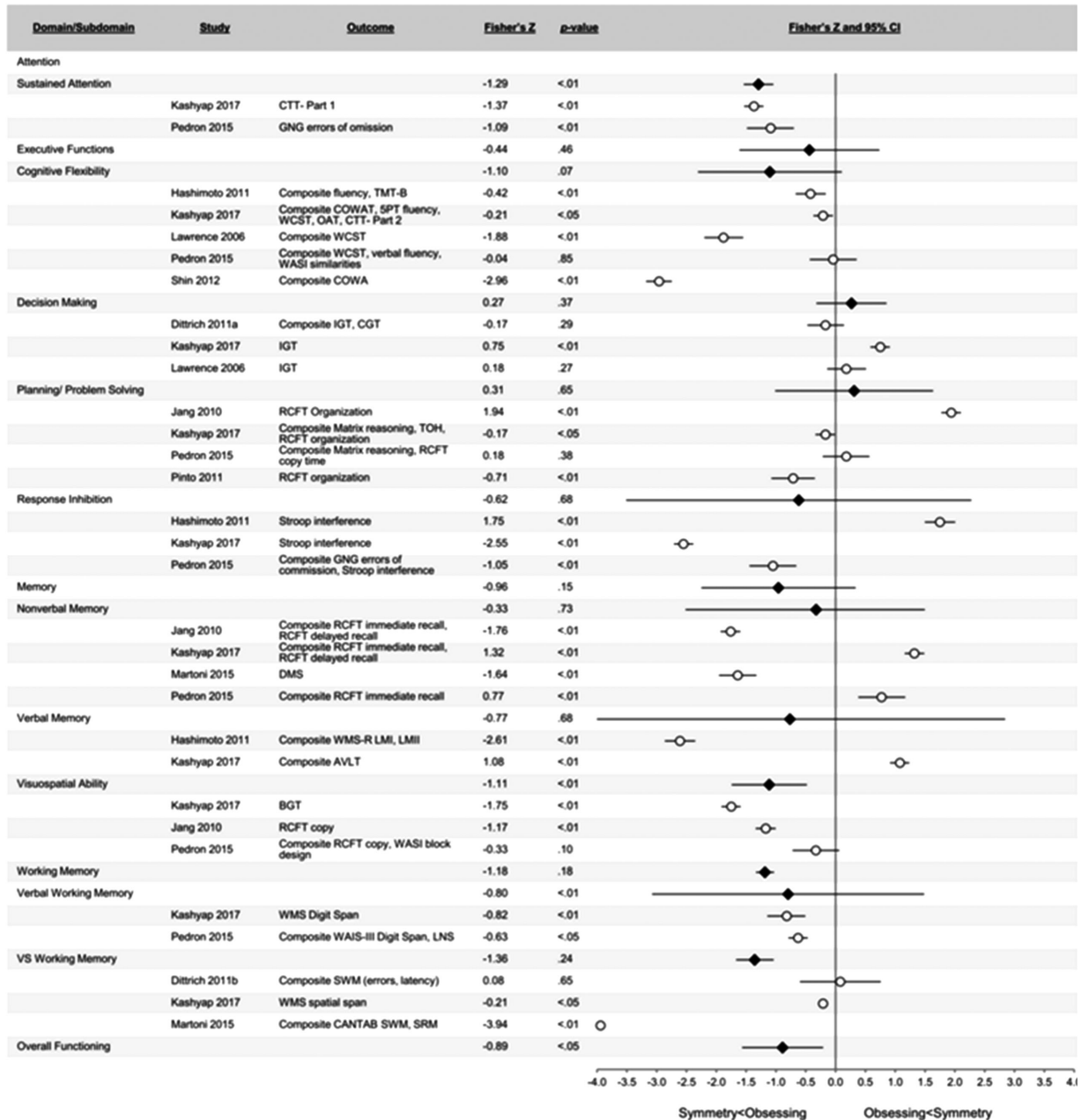


FIGURE 3 Forest plot of Fisher's z effect sizes comparing the relation between the symmetry/ordering dimension and neuropsychological functioning and the obsessing/checking dimension and neuropsychological functioning

the memory domain and the verbal memory subdomain. In comparisons of how strongly each of the two symptom dimensions were associated with performance in the various cognitive domains, results showed significant and large effects for relations between S/O symptoms and deficits in overall neuropsychological functioning, visuospatial ability, attention, and verbal working memory compared to O/C symptoms. Finally, given there has been a great deal of interest in the potential role of set-shifting deficits in individuals with OCD and their unaffected relatives (Chamberlain et al., 2007), we note that there was a large effect (but nonsignificant trend) suggesting that increased S/O symptoms may be associated with poorer cognitive flexibility compared to O/C symptoms. Given the focus on the role of executive functions in OCD, we were particularly interested in these results. On the

one hand, previous research and models have suggested widespread EF impairments in OCD (Abramovitch et al., 2013; Kuelz, Hohagen, & Voderholzer, 2004; Snyder et al., 2015; Tükel et al., 2012). However, we found differences between symptom types and specific EF abilities. Using broad categories of cognitive skills (e.g., "EF"), may obscure informative effects, and results of this study suggest that it may be important to distinguish subdomains of EF. We found that as symptoms of S/O increased, performance in the domain of EF and subdomain of cognitive flexibility decreased, however no relations were found between this symptom dimension and other subdomains of EF, and further, no differences were found in EF or its subdomains when comparing S/O to O/C symptoms (although cognitive-flexibility demonstrated a large effect and trend toward significance). In their comparison of checkers

TABLE 3 Mean effect sizes and heterogeneity statistics

Domain/Subdomain	Studies	Fisher's <i>z</i>	<i>p</i> -Value	95% CI	95% CI	<i>Q</i>	<i>df</i> (<i>Q</i>)	<i>p</i> (<i>Q</i>)	<i>I</i> ²
Attention	2	−1.29	<.01	−1.54	−1.04	1.66	1	.20	39.70
Executive functions	8	−0.44	.46	−1.61	0.74	1,318.43	7	<.01	99.47
Cognitive flexibility	5	−1.10	.07	−2.31	0.10	477.84	4	<.01	99.16
Decision-Making	3	0.27	.37	−0.32	0.85	30.62	2	<.01	93.47
Planning/Problem solving	4	0.31	.65	−1.01	1.64	392.89	3	<.01	99.24
Response inhibition	3	−0.62	.68	−3.51	2.28	787.98	2	<.01	36.99
Memory	5	−0.96	.15	−2.25	0.34	645.35	4	<.01	99.38
Nonverbal memory	4	−0.33	.73	−2.15	1.50	787.87	3	<.01	99.62
Verbal memory	2	−0.77	.68	−4.38	2.84	578.31	1	<.01	99.83
Visuospatial ability	3	−1.11	<.01	−1.74	−0.48	54.03	2	<.01	96.30
Working memory	4	−1.18	.18	−2.92	0.56	465.45	3	<.01	99.36
Verbal working memory	2	−0.80	<.01	−0.95	−0.65	0.81	1	.37	0.00
Visuospatial working memory	3	−1.36	.24	−3.64	0.93	464.18	2	<.01	99.57
Overall functioning	10	−0.89	<.05	−1.57	−0.21	650.89	9	<.01	98.62

and washers, Leopold and Backenstrass (2015) identified more consistent differences in EF suggesting that checkers demonstrate poorer functioning in all EF subdomains compared to washers (Leopold & Backenstrass, 2015). Together, these results undermine the assumption of the presence of EF impairment across symptom presentation in OCD, and instead suggest there may be specific patterns of performance across specific subdomains that relate to different symptoms.

Both psychological and biological models of OCD have been refined over the past decades. Traditional cognitive-behavioral models of OCD have emphasized the role of anxiety and HA in the etiology and maintenance of symptoms (Rachman, 1997; Salkovskis, 1985). Specifically, it has been proposed that the normal occurrence of an intrusive thought is interpreted in such a way that indicates increased likelihood of danger, and thus motivates behaviors aimed at reducing this harm (Rachman, 1997). Likewise, neurobiological models of OCD have traditionally implicated dysfunction in the orbitofronto-striato-thalamic circuit, which includes regions thought to be involved in affective regulation, thus also emphasizing the role of anxiety and emotion processing in OCD pathology (Saxena & Rauch, 2000). Evidence suggests an overlap between this neural circuit and regions involved in decision-making (Brand, Labudda, & Markowitsch, 2006; Hsu, Bhatt, Adolphs, Tranel, & Camerer, 2005; O'Doherty, Kringelbach, Rolls, Hornak, & Andrews, 2001; Ross, Sherrill, & Stern, 2011). Indeed, it has been suggested that dysfunction in this specific circuit is tied to decision-making deficits in other individuals with Bipolar I disorder (Murphy et al., 2001) and Parkinson's disorder (Thiel et al., 2003). Interestingly, decision-making was one of the subdomains we found impairment to be more closely (albeit not significantly) related to O/C rather than S/O symptoms. Findings from previous investigations have also found a connection between checking symptoms and activity in orbitofronto-striato-thalamic circuit regions, including the anterior cingulate cortex, anterior temporal lobe, and orbitofrontal cortex (Cottraux et al., 1996; Murayama et al., 2013; van den Heuvel et al., 2009). Decision-making impairment may relate to difficulty stopping checking compulsions (e.g., knowing for sure when the threat is

reduced) or problems selecting more effective strategies to regulate perseverative thoughts. As O/C symptoms are more likely to relate to a feared outcome and be motivated by a goal of reducing anxiety, they are more closely tied to traditional cognitive models of OCD and thus more closely resemble anxiety disorders (Coles, Pietrefesa et al., 2008; Starcevic & Brakoulias, 2008; Summerfeldt et al., 2014). There is evidence suggesting that anxiety influences decision-making (Engelmann, Meyer, Fehr, & Ruff, 2015; Hartley & Phelps, 2012; Luhmann, Ishida, & Hajcak, 2011; Miu, Heilman, & Houser, 2008; Raghunathan & Pham, 1999), and is impaired in anxiety disorders (Teng et al., 2016; Wölk, Sütterlin, Koch, Vögele, & Schulz, 2014). Therefore, when taken into context of existing literature, the current results concerning decision-making may suggest that the neural processes underlying O/C symptoms are similar to those seen in anxiety disorders.

More recent psychological models have sought to incorporate the role of non-fear based phenomena in the pathology of OCD. The motivation model posits that two core motivations underlie OCD symptoms: the "traditional" HA motivation, and the incompleteness motivation, where symptoms are driven by a desire to relieve discomfort, or "NJREs" (Rasmussen & Eisen, 1992; Summerfeldt, 2004; Summerfeldt et al., 2014). Newer neurobiological models have also expanded on the traditional orbitofrontal-striatal model. Menzies et al. (2008) proposed a revised model incorporating two circuits: the traditional orbitofronto-striatal loop, labeled the "Affective loop," and a dorsolateral prefronto-striatal loop, the "Cognitive loop" (Alexander, DeLong, & Strick, 1986; Menzies et al., 2008). This Cognitive loop is thought to be responsible for inhibition and switching between behavioral, sensory, and cognitive processes (Göttlich, Krämer, Kordon, Hohagen, & Zurowski, 2014). Regions involved in the cognitive loop, such as the parietal cortices, dorsolateral (DLPFC) and ventrolateral prefrontal cortices, and the caudate nucleus, have been shown to be involved in the neuropsychological processes we found to be impaired in the S/O dimension, including sustained attention (Coull, Frith, Frackowiak, & Grasby, 1996; Egner & Hirsch, 2005; Sarter, Givens, & Bruno, 2001), visuospatial ability (Kravitz, Saleem, Baker, & Mishkin, 2011), verbal

working memory (Petrides, Alivisatos, Meyer, & Evans, 1993), and cognitive flexibility (Buchsbaum, Greer, Chang, & Berman, 2005; Dajani & Uddin, 2015). For example, parietal areas and the DLPFC are proposed to be key neural regions involved in performance on the Wisconsin Card Sorting Task (WCST; Lie, Specht, Marshall, & Fink, 2006). As S/O symptoms are strongly related to "NJREs," and less often performed with the goal of preventing a feared outcome, it would be plausible that this domain is less strongly linked to activity in limbic regions (Starcevic & Brakoulias, 2008).

Symmetry behavior has been associated with activity in the DLPFC, medial thalamus, parietal and motor regions in healthy individuals (de Vries et al., 2013), OCD samples (Rauch et al., 1998; van den Heuvel et al., 2009), and related psychopathology (Suda et al., 2014). Additionally, all of these regions are thought to be involved in tic disorders, a psychopathology more related to OCD with S/O symptoms (Caligiore, Mannella, Arbib, & Baldassarre, 2017; Peterson et al., 1998; Singer, 2005; Stern et al., 2000). The sensory experiences reported in NJREs have often been compared to premonitory urges seen in tic disorders (Leckman et al., 1994; Prado et al., 2008). Neuroimaging studies have implicated the role of altered activation in somatosensory areas, including areas of the parietal cortex, in premonitory urges (Wang et al., 2011), and altered connectivity between the prefrontal cortex, caudate nucleus, and basal ganglia in tic suppression (Gerard & Peterson, 2003). Findings from neuropsychological literature also demonstrate similarities between deficits seen in individuals with tic disorders and the pattern of cognitive impairment related to S/O symptoms in the current study. Specifically, studies suggest that individuals with Tourette's syndrome show deficits related to cognitive flexibility (Bornstein, Baker, Bazylewich, & Douglass, 1991; Watkins et al., 2014) and visuospatial ability (Schultz, Evans, & Wolff, 1999). Similarly, greater deficits in visuospatial ability (Gruner, 2009) and cognitive flexibility (Gruner & McKay, 2013), have been found in individuals with OCD with tics compared to those with OCD without tics. Lucke et al. (2015) found greater sustained attention deficits in OCD with tics compared to both individuals with OCD alone and tics alone. Therefore, findings from the present study not only provide further support for an association between the dorsolateral prefronto-striatal loop (Cognitive loop) and S/O and related features (i.e., incompleteness) in OCD, but may also suggest that dysfunction in this circuit underlies incompleteness-related symptoms across multiple types of psychopathology.

5 | LIMITATIONS

The number of eligible studies was limited, and therefore results should be considered preliminary. However, the present results provide a clear picture of areas that have been well studied and areas that deserve more focus in the future. As more studies address neuropsychological functioning in OCD, including results by symptom types, our confidence in these findings will increase. Results presented herein suggest that caution should be taken when collapsing both symptom and neuropsychological categories. For example, neither collapsing subdomains to represent a domain such as "EF" nor grouping together

all individuals with an OCD diagnosis may be useful for furthering our understanding of the underlying psychopathological processes. At the same time, we must strike a balance between examining these constructs in an overly reductionist manner, or else we may fail to gain an understanding of the interaction of processes that contribute to an overall clinical picture.

Included papers varied in how symptoms were assessed, the amount of data provided regarding number of individuals endorsing specific symptoms, number of symptoms endorsed, and type of primary symptom. The majority of studies included in this meta-analysis examined symptoms dimensionally across the entire sample, regardless of primary symptom type. People with OCD generally do not endorse only one type of symptom, and therefore examining the data from a categorical perspective may limit the generalizability of findings. However, with increased generalizability comes elevated heterogeneity. The included assessments (Y-BOCS SC, DY-BOCS, OCI-R) vary slightly on how they categorize each symptom dimension, however the S/O and O/C subscales demonstrate good convergent and discriminant validity (Pertusa et al., 2012; Sulkowski et al., 2008) and unique relations with either incompleteness or HA (Pietrefesa & Coles, 2008; Taylor et al., 2014). Type of symptom measure was also included as a categorical variable in the meta-regression analyses and was not found to contribute to effect sizes.

Depression has been associated with neuropsychological deficits (Basso, Bornstein, Carona, & Morton, 2001), however the meta-analysis by Abramovitch et al. (2013) did not find any associations between depression and neuropsychological functioning. A limited number of studies included in this paper provided information on depression and therefore we were unable to include this variable in the moderator analyses. However, percent of patients diagnosed with any comorbid condition and percent of patients with a co-occurring tic disorder diagnosis were each included in the moderator analyses and were not associated with effect sizes.

Further, most tests used in standard neuropsychological assessments (such as the WCST) were developed to assess broad cognitive deficits and most likely tap into more than one domain (Eling, Derckx, & Maes, 2008; Reitan & Wolfson, 1994). Neurocognitive domains are not independent, and functioning in one domain (e.g., memory), may depend on ability in another (e.g., planning/problem solving), and therefore disentangling them is challenging (Miyake et al., 2000). Thus, we may be using inappropriate tools for looking at specific and nuanced characteristics of pathology, such as symptom dimensions. Additionally, many neuropsychological subdomains can be further divided into specific constructs. Cognitive flexibility, for one, is a broad category that includes set-shifting, reversal learning, task switching, and inhibition (Gruner & Pittenger, 2017). Though we found worse performance in cognitive flexibility subdomain was related to the S/O symptoms, data from the single study that included the Object Alternation Test (OAT) demonstrated that perseverative errors were significantly correlated with the O/C dimension (Kashyap et al., 2017). Even though both the WCST and OAT are measures of contingency learning, the WCST also measures set-shifting (Gruner & Pittenger, 2017). Further evaluating these different components might serve to provide more specific information about varying clinical characteristics.

Last, there was considerable heterogeneity within a number of subdomains. For example, within the planning/problem solving subdomain, two of the three correlations for the Rey–Osterrieth Complex Figure Task organization score showed large negative-effect sizes, the third showed a large positive-effect size. Therefore, this subdomain should be interpreted with caution and additional studies will be needed to further clarify whether relative impairment is clinically meaningful.

6 | CONCLUSION

In summary, results from this meta-analysis underscore the importance of taking into consideration OCD symptom heterogeneity. Previous neuropsychological research in OCD has provided varied results, and findings from this study further support the notion this could be due to differential symptom expression across studies (Abramovitch et al., 2013). Importantly, differences found within cognitive subdomains suggest that previous investigations reporting the presence of broad executive function impairments in OCD is overstated. In contrast, the cognitive profiles presently identified would suggest that the S/O symptom dimension may more strongly relate to altered functioning in the dorsolateral prefronto-striatal loop and O/C symptoms to functioning in the orbitofronto-striatal loop.

Results also emphasize the importance of studying non-fear-based phenomena in OCD and pending replication, could have important implications for treatment. It is possible that the longstanding research and clinical emphasis on HA-related symptoms in OCD may be one reason why a significant proportion of patients remain symptomatic following treatment (Abramowitz, Franklin, & Foa, 2002). Research should focus on continuing to understand the neural and psychological correlations of both HA and incompleteness in OCD and related disorders. Expanding our focus from just OCD to disorders with shared phenomena—for example, tic disorders—may enhance both our understanding of, and the way we treat incompleteness-related symptoms.

Clarifying the various mechanisms underlying clinical presentation is important for personalization of treatment. For example, gaining a better understanding of whether the proposed mechanisms of action in existing psychological treatments (e.g., habituation in exposure and response prevention) are effective for incompleteness-related symptoms. If certain symptom clusters do in fact reflect altered functioning in different neural circuits, a simple assessment would allow psychopharmacological and somatic therapies (e.g., transcranial magnetic stimulation) to better target affected neurotransmitters and neural regions.

Future investigations should, when possible, take into consideration the clinical characteristics of OCD rather than treating it as a single homogenous diagnosis. Additionally, many neuropsychological subdomains can be further divided into specific constructs. Evaluating these different components might serve to provide more specific information about varying clinical characteristics, ultimately serving to bridge the gap between treatment responders and non-responders.

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