Review

The impact of symptom severity on cognitive function in obsessive-compulsive disorder: A meta-analysis

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HIGHLIGHTS

- Neuropsychological meta-analyses in OCD demonstrate unexplained heterogeneity.
- No variable was found to moderate this heterogeneity including symptom severity.
- We conducted the first meta-analysis on the impact of OCD severity on cognitive functions.
- Predominantly small and homogeneous correlation effect sizes were found.
- However, important methodological and conceptual limitations were identified.
- These limitations hinder firm conclusions based on these results.

ABSTRACT

Research on cognitive functions in obsessive-compulsive disorder (OCD) is notoriously heterogeneous with no moderators identified that account for this variability. OCD severity is the primary potential moderator of interest given the longstanding trait versus state debate. Nevertheless, severity has been previously assessed exclusively as a moderator and was not directly and systematically investigated. To address this gap in the literature, the aim of this study was to conduct a systematic meta-analytic review of correlations between cognitive function and symptom severity in OCD samples. Thirty-eight studies were included, allowing for analysis of 132 effects and meta-regression analyses for potential moderators. Small effects were found for the association between cognitive function and symptom severity on major neuropsychological domains, and some subdomains exhibited medium effects for this association. However, several significant methodological and conceptual problems were identified, including the use of the Yale-Brown Obsessive-Compulsive Scale that assesses severity in the past week and not at time of testing, a tendency to not report non-significant correlations, and problematic ecological validity of neuropsychological tests in OCD. In conclusion, we found a small-to-moderate degree of association between OCD symptom severity and cognitive function, but results should be interpreted cautiously given the limitations identified. We offer recommendations that will facilitate future research into this association and move the field beyond the largely stagnant debate about the state versus trait nature of cognitive functioning in OCD, and across disorders.

1. Introduction

Obsessive-compulsive disorder (OCD) is a prevalent disorder, found in approximately 1 in 40 people (Ruscio, Stein, Chiu, & Kessler, 2010). OCD is characterized by intrusive, unwanted, and disturbing thoughts, images, and urges (obsessions), that elicit significant distress and repetitive behavioral or mental rituals (compulsions) that lead to a temporary reduction in anxiety and distress. In light of the clear phenomenological and psychopathological relevance of the need for exertion of

explicit hypercontrol in OCD (Bucci et al., 2007; Soref, Liberman, Abramovitch, & Dar, 2018), and the contrasting notion of diminished control in the context of compulsion (Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005), an extensive body of literature investigating cognitive function in OCD has accumulated. A large body of research has documented deficient performance across a wide range of cognitive domains including executive functions, non-verbal memory, processing speed, and visuospatial functions (for comprehensive systematic reviews see Abramovitch & Cooperman, 2015;

Kuelz, Hohagen, & Voderholzer, 2004). A number of recent meta-analytic investigations into neuropsychological test performance in OCD estimated the magnitude of underperformance on cognitive tests and found that most domain effect sizes range between small to medium in adult samples (Abramovitch, Abramowitz, & Mittelman, 2013; Shin, Lee, Kim, & Kwon, 2014; Snyder, Kaiser, Warren, & Heller, 2014) and null to small in pediatric samples (Abramovitch et al., 2015). The small-to-moderate magnitude of these effects and the notorious inconsistencies across this body of literature has led some researchers to question its clinical relevance as well as the all-too-frequent use of terminology such as 'impairment' and 'deficit' (Abramovitch, Abramowitz, & Mittelman, 2013; Mancini & Barcaccia, 2014; Moritz, Hauschildt, Saathoff, & Jelinek, 2017).

Corresponding to the inconsistent nature of this body of literature, most neuropsychological domain effect sizes were found to be associated with significant heterogeneity. In fact, the inconsistent nature of neuropsychological research in OCD has been repeatedly highlighted in systematic reviews over the past three decades (Abramovitch & Cooperman, 2015; Greisberg & McKay, 2003; Kuelz et al., 2004; Olley, Malhi, & Sachdev, 2007), and significant heterogeneity has been documented in several meta-analytic reviews (Abramovitch et al., 2013; Shin et al., 2014; Snyder et al., 2014). Attempts to identify moderators that may account for the persistent heterogeneity found in these meta-analyses revealed no significant moderating effect for cognitive function in OCD across demographic, neuropsychological, and clinical indices—including symptom severity. Although a large array of variables were tested for moderating effects, until recently, a notable gap in the literature existed, whereby a possible moderating effect may be related to difference in OCD symptom dimensions (e.g., 'checking', 'washing'). To address this question, two meta-analyses assessed the association between OCD dimensions and cognitive function. One study compared checking and washing dimensions on cognitive functions in OCD, and a more recent study assessed 'symmetry' versus 'obsessing' dimensions. Both studies generally reported small discriminatory effects that call into question the clinical relevance of these effects (Bragdon, Gibb, & Coles, 2018; Leopold & Backenstrass, 2015). Notably, these two meta-analyses included a small number of studies and reported significant heterogeneity across effect sizes but did not identify any moderators that account for this variability. However, to our knowledge, apart from being assessed for moderating effects in meta-regressions, a direct systematic quantitative review of the association between OCD severity and cognitive functions has never been conducted.

The importance of systematically assessing the association between symptom severity and cognitive function in OCD primarily stems from the controversy regarding the etiology of cognitive dysfunction in OCD— namely the state-trait debate. This long-lasting debate has been largely stagnant, with studies supporting both sides of the issue being published every year. On one side of the debate, some researchers argue that underperformance on neuropsychological tests in OCD is a trait-like, stable characteristic of individuals diagnosed with OCD and even a causal disorder-specific antecedent of OCD (e.g., Chamberlain et al., 2005). This view is supported by evidence suggesting that some domains of cognitive underperformance in OCD are found in unaffected relatives (Chamberlain et al., 2007), that cognitive dysfunction may be found in remitted OCD samples (Rao, Reddy, Kumar, Kandavel, & Chandrashekar, 2008), and that response to psychotherapy/pharmacotherapy may not be accompanied by improvement in neuropsychological test performance (e.g., Bannon, Gonsalvez, Croft, & Boyce, 2006). However, contrasting evidence has been used to support the state-dependent nature of cognitive dysfunction in OCD. Some studies did not find cognitive dysfunction in unaffected relatives of individuals diagnosed with OCD (e.g., de Wit et al., 2012) and some investigations demonstrated improvement in some cognitive domains in OCD following treatment (e.g., D'Alcante et al., 2012; Voderholzer et al., 2013). In fact, a recent study documented such improvement and reported that

the improvement in cognitive function was stable at a two-year follow-up (van der Straten, Huyser, Wolters, Denys, & van Wingen, 2018). Moreover, although found in a minority of studies, some investigations report a significant association between cognitive function and OCD severity (e.g., Abramovitch, Dar, Hermesh, & Schweiger, 2012; Nedeljkovic et al., 2009; Segalas et al., 2008), while others demonstrated cognitive underperformance in the context of OCD-specific content (Zetsche, Rief, Westermann, & Exner, 2014). Lending support to the state-related nature of underperformance on cognitive tasks in OCD, Kalanthroff, Aslan, and Dar (2017) demonstrated how underperformance on a cognitive test in OCD may result from threatened morality. In this recent study, a sample of individuals with OCD were asked to report an immoral deed they committed. Participants were then randomized into an experimental and control condition. Those in the experimental condition-that allowed for participants to subsequently wipe their hands- exhibited significantly better performance on a response inhibition task compared to those in the control condition, that were not allowed to wipe their hands (Kalanthroff et al., 2017).

It is evident that the state-trait debate is inconclusive, ongoing, and limited progress has been achieved. Nevertheless, some researchers have offered novel insights that may advance research into this question, but these may be considered anecdotal suggestions as this time. First, in the context of inhibitory control, it has been suggested that the nature of underperformance on cognitive tasks in OCD is comprised of both state and trait elements. It has been proposed that subtle trait-like cognitive dysfunction may be enhanced by state-related factors (Kalanthroff, Anholt, & Henik, 2014); but this speculation is yet to be supported by evidence. Second, a recent novel perspective was offered suggesting that some basic cognitive processes in OCD such as selective attention and response inhibition may not correspond to either the traditional state or trait definition, implying that the state-trait debate may be more domain-specific (De Putter, Cromheeke, Anholt, Mueller, & Koster, 2018; De Putter & Koster, 2017). Finally, in a recent study investigating the association between executive function and treatment response in pediatric OCD, Hybel and colleagues (2017) demonstrated no significant change on executive function as assessed via neuropsychological tasks post successful treatment. However, a significant improvement in executive function-related behaviors was found (via self and parental reports) suggesting a discrepancy in the association between neuropsychological test performance in the lab versus a more ecologically valid assessment of real-life behaviors associated with cognitive function (Hybel et al., 2017). In light of this debate, it would be important to transcend the null hypothesis tests pertaining to the existence or absence of an association between symptom severity and cognitive functions and to systematically assess the magnitude of such an association.

In the context of a meta-analytic investigation, examination of symptom severity as a moderator of cognitive function is based on the premise that if significant, symptom severity scores across studies would be able to explain a significant portion of the variability among effect sizes for cognitive functioning. Although meta-analytic moderator analysis is a methodologically legitimate mean to assess such associations, (transcending the need to identify and record correlation coefficients from individual studies), it also has some inherent shortcomings. Firstly, analysis of direct correlation coefficients may be more informative than a moderator analysis, particularly given the ability to assess direct explained variance. Secondly, comparative meta-analysis, assessing neuropsychological function exclusively, includes controlled studies, whereas correlation meta-analysis is more inclusive in that it can include uncontrolled studies that are sufficient to extract within-groups correlations. Thirdly, meta-analyses usually compute multivariate meta-regressions assessing the relative partial contribution of moderators. Although this is a more realistic and ecologically valid method, potential multicollinearity may obscure the discovery of specific phenomena. Fourthly, a meta-analysis systematically assessing direct correlations is not only more informative, it further allows for

assessment of heterogeneity among correlation effect sizes. Subsequently this type of study facilitates the discovery of potential moderators of the association between symptom severity and cognitive function.

To address this gap in the literature and the need for a direct and systematic examination of this association, the purpose of the present study was to conduct the first systematic quantitative review of the association between symptom severity and neuropsychological test performance in OCD.

2. Methods

2.1. Retrieval and study selection

A literature search was conducted throughout March 2018, using MEDLINE, ISI Web of Knowledge, Google Scholar, and publications' reference lists. Since the goal of the literature search was to identify correlation coefficients between severity scores from validated OCD severity scales and validated neuropsychological measures across neurocognitive domains, the focus of the literature search was a combination of four elements: an OCD sample, a validated neuropsychological test, a validated OCD severity scale, and a reported correlation between severity and neuropsychological test performance. Such studies may include imaging studies, clinical trials, neuropsychological studies, and experimental studies employing cognitive tasks. To accomplish the goals of this complex literature search, we employed a broad search strategy designed to identify studies assessing cognitive function and symptom severity in OCD. The search employed a combination of keywords pertaining to OCD and OCD symptom severity (e.g., 'OCD', 'obsessive-compulsive disorder', 'obsessions', 'compulsions', 'severity', 'symptom severity', 'Yale-Brown Obsessive-Compulsive Scale', 'correlation', 'association', etc.) in conjunction with keywords associated with cognition terminology (e.g., 'cognitive', 'neurocognitive', 'neuropsychological', etc.), major cognitive functions (e.g., 'executive function', 'memory', 'processing speed', 'attention', 'visuospatial function', etc.), specific cognitive sub-domains (e.g., 'response inhibition', 'planning' 'working memory', 'verbal memory', etc.), and searches using neuropsychological test names (e.g., 'Wisconsin Card Sorting Test', 'Stroop', 'Digit Span', 'RCFT', 'Stop Signal Test', etc.). This preliminary search yielded 435 studies.

We included studies that were published in peer-reviewed journals in English and that included an OCD sample diagnosed using a psychometraically valid procedure, at least one validated neuropsychological task, and at least one correlation coefficient reported between the Y-BOCS total score (or other validated measures of OCD severity) with at least one of the tests' outcome measures. Given the substantial difference between pediatric and adult OCD in terms of cognitive functions and various clinical aspects as well as severity measurement, pediatric studies were a priori excluded. In further careful examination of the 22 pediatric studies found in this literature search, only one study would have been included in the meta-analysis, contributing one effect size. Studies that exclusively employed cognitive tasks that were not psychometrically validated and normed, were developed for a particular study, or included neuropsychological tasks modified significantly from the original version, were excluded. Using these inclusion and exclusion criteria, 396 studies were excluded (see Fig. 1). The first author reviewed and approved all included studies and examined any paper for which the co-authors did not reach a consensus upon. In addition, all excluded studies were labeled with a reason for exclusion (e.g., no correlation coefficients provided, lack of valid assessment procedure) by the co-authors. All manuscripts suggested for exclusion for which a decision was complex, unclear, or where a discrepancy between co-authors has been identified, were carefully reviewed by the first authors to make a final decision. This left a final count of 38 studies that were included in the present meta-analysis with publication years ranging from 1997 to 2018.
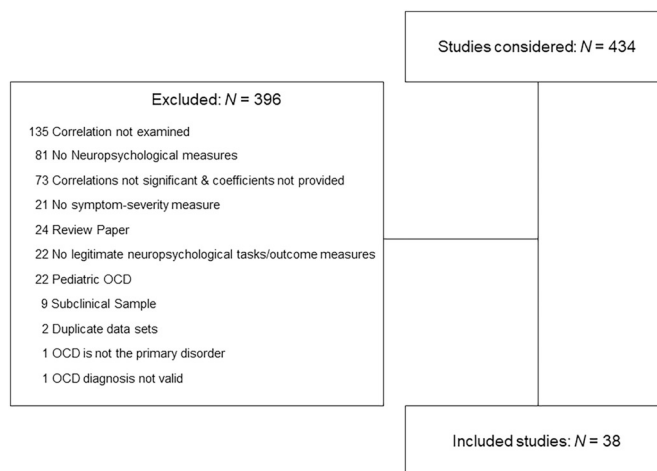

Fig. 1. Study selection process.

2.2. Variables recorded and coded from included studies

The following information was recorded from included studies: first author, year of publication, OCD sample size, age, years of education, type of severity scale, severity scores, age of onset, percent females, percent medicated participants, and percent of participants with at least one comorbid condition. In addition to the correlation coefficients between symptom severity measures and neuropsychological outcome measure, the type of neuropsychological domain (e.g., executive function), subdomain (e.g., set shifting), test name, and the type of neuropsychological outcome measure (e.g., time in seconds) were recorded. When available, correlations between neuropsychological measures and Y-BOCS subscale scores (i.e., obsessions, and compulsions) were recorded as well. However, only a small number of such effect sizes was available, and most studies that provided information regarding Y-BOCS subscales also provided the total score. Given the known association between the two subscales and between the subscales and total score, and considering the small number of subscale effect sizes available, we did not pursue a meta-analysis of subscales correlations. However, 4 out of the 38 included studies presented only subscale scores and thus 34 out of the 38 included studies were used for analyses of correlation effect size using the total symptom severity scores. A summary of the characteristics of the 38 included studies is presented in Table 1. Included studies originated from fifteen different countries: India ($k = 6$), US ($k = 4$), Spain ($k = 4$), Brazil ($k = 3$), Turkey ($k = 3$), South Korea ($k = 3$), Germany ($k = 2$), Australia ($k = 2$), Israel ($k = 2$), China ($k = 2$), Netherlands ($k = 2$), UK ($k = 2$), France ($k = 1$), Italy ($k = 1$), and Japan ($k = 1$).

2.3. Data analysis

A correlation meta-analysis utilizing Fisher's z transformation scale (Hedges & Olkin, 1982) was conducted using Comprehensive Meta-

Table 1
Characteristics of 38 studies included in the meta-analysis.

Study Characteristic	Mean	SD	Range	Studies reporting
Sample Size	34.44	21.14	11–107	38
Age (years)	31.08	4.64	23.00–42.95	38
Percent Females (%)	51.67%	22.43	0.00–100.00	37
Total Y-BOCS Score	24.11	2.70	19.50–29.34	34
Percent With Comorbidity (%)	22.30%	31.19	0.00–91.00	21
Percent Medicated (%)	56.06%	40.88	0.00–100.00	27

Y-BOCS, Yale-Brown Obsessive Compulsive Scale.

Table 2
Random model weighted mean effect sizes and tests of homogeneity by neuropsychological domains and subdomains.

Domain/Subdomain	K	ES r	Std. Error	Lower CI	Upper CI	Z	Sig	Q	p(Q)	Tau	I^2
Executive Functions	82	−0.270	0.013	−0.33	−0.21	−8.72	0.000	197.39	0.000	0.21	58.96
Planning	7	−0.429	0.053	−0.59	−0.23	−4.01	0.000	13.38	0.037	0.22	55.14
Response Inhibition	19	−0.280	0.039	−0.41	−0.14	−3.75	0.000	63.04	0.000	0.27	71.45
Fluency	6	−0.225	0.034	−0.39	−0.04	−2.41	0.016	9.94	0.077	0.16	49.71
Working Memory	12	−0.161	0.034	−0.31	0.00	−2.00	0.046	25.93	0.007	0.21	57.58
Set Shifting	38	−0.277	0.016	−0.35	−0.20	−6.61	0.000	72.95	0.000	0.18	49.28
Memory	26	−0.164	0.011	−0.24	−0.09	−4.23	0.000	31.08	0.186	0.09	19.56
Verbal Memory	12	−0.106	0.020	−0.23	0.02	−1.67	0.095	18.88	0.063	0.14	41.74
Non-verbal Memory	14	−0.226	0.014	−0.32	−0.13	−4.55	0.000	9.27	0.753	0.00	0.00
Processing Speed											
Processing Speed	13	−0.204	0.023	−0.33	−0.08	−3.11	0.002	15.69	0.206	0.12	23.51
Visuospatial Abilities											
Visuospatial Abilities	7	−0.194	0.051	−0.40	0.03	−1.73	0.083	11.36	0.078	0.20	47.19
Attention											
Sustained Attention	4	−0.311	0.048	−0.51	−0.08	−2.65	0.008	6.68	0.083	0.18	55.06

Analysis software (Borenstein, Hedges, Higgins, & Rothstein, 2014). A random effects model was employed and effect size coefficient r was used as the primary outcome index, where small, medium, and large effect sizes correspond to 0.1, 0.3, and 0.5, respectively (Cohen, 1992). In the present study, negative correlations indicate that increased OCD severity is associated with poorer performance. Thus, in cases where a positive correlation was originally found, such as between OCD severity and the number of errors or response time (indicating that worse performance is associated with increased severity), the direction of such a correlation was changed to negative. Effect size heterogeneity was assessed using Q statistics and significance of Q as well as the I^2 statistic. To assess publication bias, we produced a funnel plot that was inspected visually as well as by using the Egger's regression test (Egger, Davey Smith, Schneider, & Minder, 1997) and employing Orwin's Fail-Safe N method (Orwin, 1983). A moderator analysis was conducted using a meta-regression model where the following variables were entered as predictors: age (mean age in years), gender (percent females), medication status (percent of medicated participants), age of onset (mean age of onset in years), and comorbidity status (percent of participants diagnosed with a at least one comorbid condition).

3. Results

3.1. Publication bias

Visual inspection of the funnel plot (see supplementary materials) revealed a reasonable symmetry, where the clear majority of results were distributed around the main effect size. In addition, most results were in the upper part of the 'funnel', suggestive of overall symmetry and absence of a publication bias. A statistical examination of the funnel plot using Egger's regression test resulted in a non-significant model, suggestive of lack of significant publication bias $t(130) = 1.766$, $p = .08$. Additionally, we computed the Orwin's Fail-Safe N which suggested that 300 studies with the effect size of *positive* 0.1 were needed to bring the present study's aggregated total correlation effect size (−0.227) to a trivial effect of $r = 0.00$. The present study included 38 studies, and even when combined with the known studies that did not present coefficients, it would still require 3 times as many studies with a positive correlation (which is rare in this context) to bring the study's effect size to a trivial level. Thus, we concluded that although we present evidence for known missing information, the likelihood of a substantial publication bias with a potential to alter the present study's results is negligible.

Review of the literature for the present study resulted in an important finding relevant to publication bias. Out of a total of the 396 excluded manuscripts, 18% ($n = 73$) reported that they computed correlations between symptom severity and neuropsychological test performance and found no significant correlations but did not present these coefficients. Our review of the literature indicates that the average sample size in those studies is smaller than $n = 40$. Thus, it is theoretically plausible that cumulatively this body of literature did not find correlation coefficients larger than −0.257 (the correlation coefficient threshold for 0.05 alpha with $n = 40$). As presented below, this magnitude of correlation is very similar to the aggregated correlation found in the present study. Notably, whereas the theoretical possibility that these studies found an aggregated correlation effect size that was positive 0.257 exists, the rarity of positive correlations (implying better performance on cognitive tests associated with elevated symptom severity) in this body of literature evidences that this is highly improbable. It should further be noted that a number of studies included in this meta-analysis explicitly stated that they present only correlations that were found to be significant, which may theoretically lead to a slight inflation of some of the effect sizes reported in this study. In sum, the gold standard quantitative statistical assessment of publication bias employed here resulted in no evidence of a significant publication bias that could have substantially jeopardized the validity of the results of the present investigation.

3.2. Neuropsychological domains and subdomains

The aggregated overall mean correlation effect for the association between symptom severity and cognitive function in OCD calculated from 34 studies and 132 correlation coefficients, was found to be small in magnitude ($r = -0.227$, $p < .0001$; CI = −0.26, −0.20). This effect size corresponds to R^2 of 0.051, indicating that approximately 5.1% of symptom severity variance is explained by variance in cognitive test performance, and vice versa. As presented in Table 2 and Fig 2, all effect sizes for both major domains and subdomains were negative, exemplifying that worse neuropsychological test performance is associated with an increase in symptom severity. Apart from visuospatial function, all effect sizes for neuropsychological major domains were significant, with a medium effect size found for attention ($r = -0.311$) and an effect size for executive function that approached a medium effect ($r = -0.270$). The smallest effect size was found for memory ($r = -0.164$). In terms of subdomains of executive functions, all effect sizes were significant and negative, with planning approaching a large effect size ($r = -0.429$) and response inhibition and set shifting approaching a medium effect. Response inhibition, fluency, and set shifting were found to be similar to the overall study effect size and overall executive function effect size, ranging from −0.280 to −0.225. The smallest effect size among executive function subdomains was found for working memory ($r = -0.161$). Effect size for the subdomain of verbal memory was the smallest across domains and subdomains in the present study and was not found to be significant ($r = -0.106$).
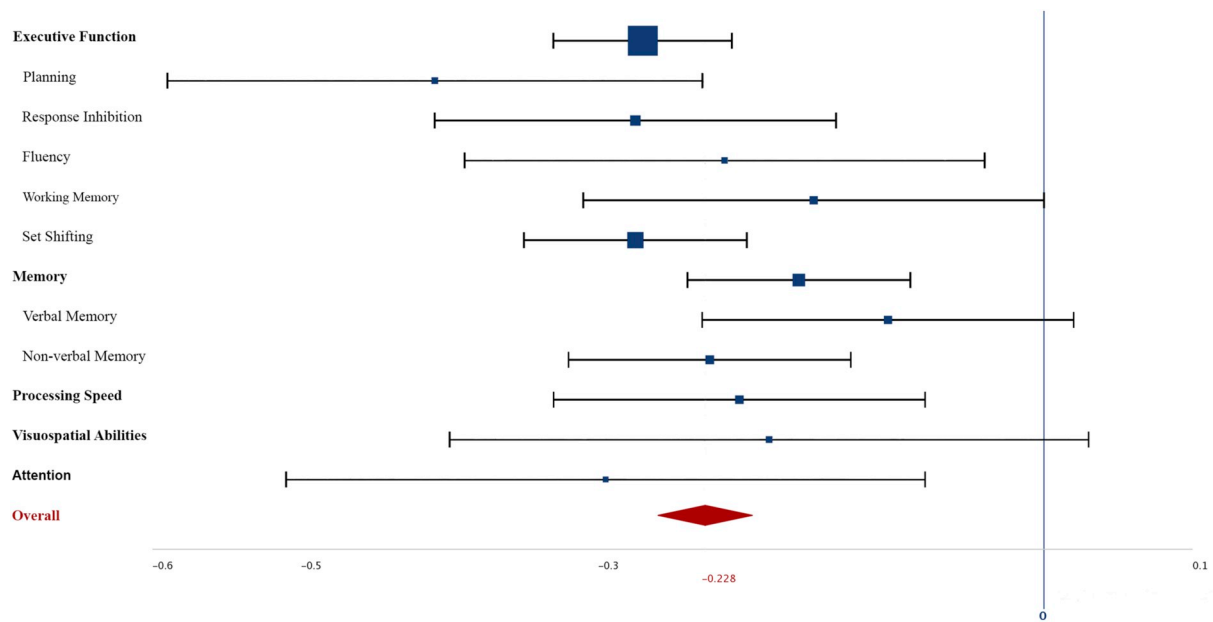
Fig. 2. Forest plot of correlation effect sizes and confidence intervals for the association between cognitive performance and OCD symptom severity across neuropsychological domains.

Non-verbal memory was found to be significantly associated with symptom severity, with an effect size of −0.226. Sustained attention was found to have a significant medium effect size ($r = -0.311$).

3.3. Test-specific effect sizes

Effect size for individuals' test outcomes was calculated (see Table 3 and Fig. 3). Test outcomes were included if at least two effect sizes were available for calculations. Significant medium negative effect sizes (indicating that poorer performance is associated with increased severity) were found for executive functions on category (semantic), and letter (phonemic) fluency, response interference (Stroop interference score), set shifting (i.e., Trail Making B, WCST failure to maintain set, WCST perseverative errors, and WCST total errors), and for other major domains including processing speed (Trail Making A), sustained attention (CPT/GNG omission errors), and visuospatial function (RCFT copy). The largest effect size was found for WCST perseverative response (large effect size, $r = -0.588$), accounting for 35% of symptom severity variance. Although the average effect size for verbal memory

approached zero, the learning index commonly extracted from verbal learning tests (e.g., CVLT, RAVLT, AVLT), in which correctly remembered words from the five repetitions of list A are summed, was found to have a significant negative medium effect size.

3.4. Heterogeneity and meta-regression

Although meta-analyses on neuropsychological functions in OCD repeatedly indicate substantial heterogeneity, a different picture emerges from the present correlation meta-analysis. In terms of the Q coefficient, across major neuropsychological domains, only the domain of executive function was found to demonstrate significant heterogeneity ($Q = 197.39$, $p < .0001$). Subdomains of executive functions were all found to have a significant Q, apart from fluency, even though this subdomain encompassed only 6 effect sizes. Examining I^2 coefficients, all domain and subdomains were found to have a small to moderate magnitude of heterogeneity. To examine the nature of the heterogeneity, a meta-regression was conducted to assess the role of age, age of onset, gender proportions, medication status, and

Table 3
Effect sizes of individuals tests/outcome measures representing the association between OCD symptom severity and test performance.

Task	K	ES r	Lower CI	Upper CI	z	p	Q	p(Q)	I^2
CPT go no go commission errors	7	−0.165	−0.36	0.05	−1.53	0.127	14.56	0.024	58.80
Digit Span	3	−0.131	−0.38	0.13	−0.97	0.333	4.77	0.092	58.03
Letter Fluency	2	−0.245	−0.46	0.00	−1.99	0.047	1.46	0.227	31.59
OAT perseverative errors	2	−0.057	−0.33	0.23	−0.39	0.698	0.16	0.689	0.00
OAT total errors	2	−0.037	−0.32	0.25	−0.25	0.799	0.05	0.816	0.00
SSRT	4	−0.154	−0.41	0.13	−1.08	0.280	6.83	0.078	56.06
Stroop interference	6	−0.367	−0.62	−0.05	−2.24	0.025	32.49	0.000	84.61
Trail Making Test B	4	−0.341	−0.54	−0.11	−2.81	0.005	5.97	0.113	49.72
Trail Making Test B-A	2	−0.222	−0.36	−0.07	−2.82	0.005	0.99	0.321	0.00
Tower of London # problems in min moves	3	−0.167	−0.38	0.06	−1.44	0.149	1.33	0.514	0.00
WCST categories completed	10	−0.249	−0.37	−0.12	−3.62	0.000	11.65	0.234	22.72
WCST failure to maintain	2	−0.406	−0.73	0.06	−1.70	0.088	2.76	0.097	63.79
WCST perseverative errors #	4	−0.345	−0.70	0.14	−1.40	0.163	24.69	0.000	87.85
WCST perseverative errors %	4	−0.144	−0.32	0.05	−1.49	0.137	0.49	0.921	0.00
WCST perseverative response	2	−0.588	−0.79	−0.27	−3.28	0.001	1.68	0.195	40.40
WCST total errors	3	−0.356	−0.61	−0.04	−2.21	0.027	4.19	0.123	52.21

OAT, Object Alternation Task; SSRT, Stop-Signal Reaction-Time; Trail Making Test B-A, total time to complete test B – total time to complete test A; WCST, Wisconsin Card Sorting Test.
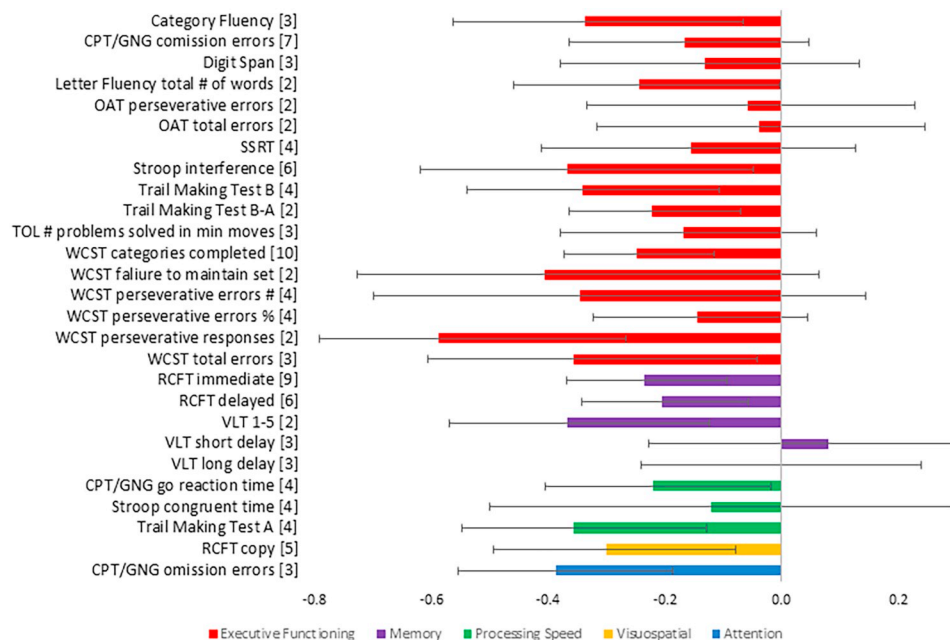
Fig. 3. Effect sizes of individuals tests/outcome measures representing the association between OCD symptom severity and test performance. [K], number of studies included for each outcome; CPT, Continuous Performance Test; OAT, Object Alternation Task; SSRT, Stop-Signal Reaction-Time; Trail Making Test B-A, total time to complete test B – total time to complete test A; WCST, Wisconsin Card Sorting Test; RCFT, Rey-Osterrieth Complex Figure Test; VLT, Verbal Learning Test includes the CVLT, AVLT, and RAVLT; VLT 1–5, total number of correct words recalled in lists 1–5; CPT GNG go reaction time, mean reaction time on go stimuli.

comorbidity status as potential moderators of the relationship between cognitive function and symptom severity. The random model meta-regression indicated that the model was significant ($Q(5) = 24.96$, $p = .0001$), with percent females ($z = -3.53$, $p = .0004$) and age of onset found to be significant. Nevertheless, this model included 58 effect sizes. When percent females was used as a univariate predictor, including 132 effect sizes, this variable was not found to be significant ($z = -1.10$, $p = .269$). Similarly, a univariate analysis encompassing all effect sizes for which information regarding age of onset was available ($n = 86$) was not found to have a significant mderating effect ($z = -1.29$, $p = .19$). Similar results were found when a meta-regression was performed for executive function effect sizes exclusively, which were found to be more heterogeneous. Thus, these results indicate that no moderator was found to account for the heterogeneity between effect sizes representing the association between symptom severity and cognitive function.

4. Discussion

Although a large body of research has accumulated on cognitive functions in OCD, several issues remain unaddressed. One of these areas is the question regarding the impact of symptom severity on cognitive function, which is related to the state versus trait nature of cognitive deficiencies in OCD. To our knowledge, this is the first systematic quantitative review of the association between OCD symptom severity and cognitive function. Although this type of literature has some inherent methodological and interpretational limitations (discussed below), the results of the present study are informative at the domain, subdomain, and test levels. Prior to discussing the results, it is important to reiterate that the magnitude of effect sizes for Cohen's *d* or Hedges' *g* are different than magnitudes of *correlation* effect size. That is, Cohen benchmarks for small, medium, and large correlation effect size are 0.1, 0.3, and 0.5 respectively (Cohen, 1992).

The overall effect size was small ($r = -0.227$) and significantly different than no effect. This suggests that increased symptom severity is associated with poorer performance on neuropsychological tests, but this association is small in magnitude, carrying only 5.2% explained variance. Major domain analyses resulted in small effect sizes for memory, processing speed, and visuospatial function ($r < 0.23$), and medium effect sizes for attention and executive functions. The latter two domains were found to account for 7–9% of the variance.

Subdomain analyses revealed that planning is associated with a medium to large effect size with 18% of variance explained. However, only 7 effect sizes were included in the planning subdomain calculations. Response inhibition and the set shifting subdomains (for which the most data was available for calculations) demonstrated medium effect sizes with approximately 7% explained variance. The effect size for the subdomain of verbal memory was not significant and had a negligible effect size. In terms of individual tests and outcome measures, WCST perseverative responses was found to have the largest effect size in the study ($r = 0.59$), accounting for 35% of variance. Other executive function tests had significant medium effect sizes, including semantic fluency and Stroop interference. Overall, these results indicate that a small to medium negative association exists between OCD symptom severity and cognitive function. Most outcome measure correlation effect sizes were smaller than 0.3, whereas planning and attention may be more strongly related to symptom severity, accounting for 9–16% of the variance. Thus, it would be reasonable to conclude that the impact of symptom severity on cognitive function in OCD is minor, and that more meaningful factors may be related to cognitive function in OCD. However, there are several issues that may hinder a clear and conclusive inference from these results.

Firstly, it is important to critically examine the psychometric properties and validity of the Y-BOCS, which was the primary measure for assessing symptom severity in 96% of all effect sizes included in this study. The Y-BOCS severity scale (Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989) is the gold standard measure for symptom severity in OCD. It has been translated to numerous languages and is considered the primary measure for determining if patients present an active clinical status or a subclinical (total Y-BOCS score ≥ 16), for the definition of treatment response (≥ 25%, or 35% reduction in symptoms), and for the assessment of symptomatic change in clinical care settings. The Y-BOCS includes 5 items assessing obsessions and 5 items assessing compulsions. These 5 items overlap and focus on time spent experiencing the symptom(obsessions or compulsions), interference resulting from the symptom, distress from the symptom, resistance to the symptom, and control of the symptom. For each of those 10 items, the examinee is rated on a 0 (no symptom) to 4 (severe symptom) scale. However, for each of the items, the examinee is asked to think about an *average day* in the *past week*. Thus, when considering the context of state-dependent cognitive dysfunction, assessment of an entire week is

potentially a problematic measure for a state related influence on performance. It is reasonable to assume that individuals with OCD may experience a relatively good week in terms of symptom severity, but a host of factors can exacerbate their symptoms at the time of testing. Individuals with OCD may experience more obsessive symptoms associated with perfectionism and fear of being wrong when assessed for several hours on cognitive tasks. In fact, some initial evidence exists for the impact of momentary contextual influences, test-related attitudes, and motivational aspects on task performance in some neuropsychological subdomains in OCD (Moritz et al., 2017; Moritz, Hottenrott, Jelinek, Brooks, & Scheurich, 2012). Nevertheless, it is plausible that the opposite may occur where individuals diagnosed with OCD may feel more relaxed and more focused in a relatively controlled safe, and distracter-free environment in the lab, even though they may have been struggling with increased symptoms in the past week. Unfortunately, a truly state-dependent measure such as the STAI-state, which assesses symptoms at present time (Marteau & Bekker, 1992), was not developed for assessment of OCD symptom severity. Furthermore, research repeatedly finds that the resistance and control items do not load on the same factor as other and more direct symptom severity items and have been found not to be as sensitive to symptomatic change as the other items (Deacon & Abramowitz, 2005; Kim, Dysken, Pheley, & Hoover, 1994). Thus, it is important to remember that the vast majority of studies assessing the association between cognitive function and symptom severity in OCD are de facto assessing performance on cognitive tests and their relations to symptoms severity experienced in an 'average day' in the past seven days, out of which 40% of the score weight is determined by four psychometrically problematic items. Therefore, inferences regarding the present influence of symptom severity on cognitive function would benefit from the development of a measure aimed at assessing *present* OCD symptom severity, or an attempt to modify existing measures (e.g., OCI-R, Y-BOCS) to allow for such an assessment.

A second issue that is oft neglected in the context of cognitive function and psychopathology research is the limited ecological validity of neuropsychological measures. Low to moderate ecological validly is consistently found in the context of assessment of cognitive functioning across populations, including samples with no known neurological or psychopathological problems (Chaytor & Schmitter-Edgecombe, 2003; Van der Elst, Van Boxtel, Van Breukelen, & Jolles, 2008). The underlying reasons for this problem are numerous and complex and are beyond the scope of this study. However, in the context of the present study, it is important to highlight three main issues. First, dysfunction on a cognitive task may not only stem from a dysfunction in the domain the task is set to examine (e.g., response inhibition), but also from deficiencies in other cognitive domains (e.g., attention, working memory). Second, performance on a test in a controlled environment may be very different than in real life, and the tasks may be too simplified compared to the behavioral construct assessed. Moreover, neuropsychological tests require focusing on the task at hand, whereas multi-tasking may be the core competency in everyday life (Burgess, 2000). For example, inhibitory control tests such as the Go/No-Go assess response inhibition, but inhibition of actions in real life is much more complex than inhibiting a mouse click or a keyboard stroke, particularly considering the host of internal and external distracters, emotional cues, and so forth. In addition, it is not unusual to find no association between two tasks that are thought to reflect the same latent construct, particularly in clinical samples. In fact, there is direct evidence for this effect in OCD research. In one study, Sohn, Kang, Namkoong, and Kim (2014) assessed inhibitory functions in OCD using cognitive tasks, including the Stop Signal Task. They found no correlations between this task and any of the Barratt Impulsiveness Scale sub-factor scores as well as the total score (all *r*'s approaching zero). Given that constructs that are considered very closely related show negligible correlations, it stands to reason that associations between cognitive functions and severity of OCD-specific symptoms may be inherently very low as well. For

example, OCD is characterized by inflexibility and rigidity, and thus it is only intuitive to expect that cognitive tasks assessing flexibility would point to a deficit in OCD samples compared to controls. However, a sophisticated recent meta-analysis found that there is no deficit in OCD pertaining to cognitive flexibility (Fradkin, Strauss, Pereg, & Huppert, 2018).

A third issue pertains to the common misconception that neuropsychological factors may be inherently disorder-specific (for a brief discussion see Abramovitch & Schweiger, 2015), promoting assessment of the associations that are the focus the present study. The premise is that underperformance on some cognitive domains may be conceptualized as disorder-specific markers or even endophenotypes of OCD. This may in turn lead to the assumption that the more severe the specific symptoms are, the larger the performance deficit on particular neuropsychological domains. However, an ever-growing body of literature indicates that nearly every form of psychopathology is associated with reduced cognitive function (e.g., Millan et al., 2012). For example, most conditions have been found to be associated with reduced performance on every subdomain of executive functions, including inhibition, set shifting, planning, working memory, and fluency (Lipszyc & Schachar, 2010; Snyder, 2013; Wright, Lipszyc, Dupuis, Thayapararajah, & Schachar, 2014). More importantly, recent research suggests that neuropsychological deficiencies are better conceptualized as part of the *p* (psychopathology) factor, in that the presence of psychopathology is predictive of underperformance on cognitive task more than the severity levels of specific symptoms (Caspi et al., 2013; Stordal et al., 2005). Indeed, it has been suggested that the burden of psychopathology, emotional reactivity, and availability of cognitive resources are the key factors influencing cognitive functions (Carver, Johnson, & Timpano, 2017), and not the diagnostic entity, specific symptoms, or symptom severity (Stordal et al., 2005).

Prima facie, the results of this study suggest an overall small to medium association between OCD symptom severity and cognitive functions. However, given the methodological and conceptual problems—some of which are associated specifically with OCD and some broader in scope—challenge our ability to extract a clear inference from these results. In order to promote future cogent inferences from this type of data, we offer a number of suggestions for future research.

1. First and foremost, researchers are encouraged to present all coefficients regardless of the significance of the correlations. This in turn will facilitate future meta-analyses.
2. In order to assess an association between OCD symptom severity at the time of testing and cognitive function, there is a need to develop a psychometrically valid measure of current OCD symptom severity. In lieu of such a measure, researchers may use a simple visual-analogue scale (VAS) to assess OCD severity at time of testing. In studies that use traditional measures, it is highly recommended to explicitly attend to the timeline which the measure pertains to.
3. In the context of investigations into cognitive functions, researchers are encouraged to assess general psychopathological burden in parallel to OCD severity.
4. Researchers are encouraged to use cognitive tasks with greater ecological validity and to augment this type of assessment with questionnaires assessing corresponding behaviors (e.g., Dimensions of Dysexecutive Questionnaire – DEX). Furthermore, researchers are encouraged to address particular issues associated with cognitive testing in OCD, such as perfectionism, reduced confidence in cognitive functions, etc.

The present study has several strengths, such as being the first to systematically investigate the associations between OCD symptom severity and cognitive functions as well as employing gold standard meta-analysis methodologies. However, the present study is not free of limitations. Nearly a fifth of the studies excluded from this study reported that they computed correlations but did not present those coefficients

because they were not significant. In addition, some included studies selectively presented only significant correlation coefficients. However, three statistical methods employed in the present study supported the absence of publication bias. In addition, the information regarding sample sizes of the relevant excluded studies allowed for an estimation of the maximum expected aggregated correlations, which may not pose a risk of altering the results of the present study significantly. Finally, a small number of studies assessed OCD symptom severity using a different measure than the Y-BOCS, but the small number of correlation coefficients (4% of the total number of effect sizes included) did not allow for examination of a potential moderating effect of type of measure. In addition, OCD severity measures are known to intercorrelate highly. In the same vein, our data did not permit a differential assessment of correlations between cognitive functions and the Y-BOCS obsessions and compulsions subscores. Nevertheless, only 4 studies reported correlations between cognitive function and Y-BOCS obsessions and compulsion scores separately. All other studies that provided information regarding correlations with subscores, reported both the total score and the subscores. Given that subscores are inherently highly correlated with total scores, the additional information from the 4 studies was not expected to alter the results, particularly given that Y-BOCS subscores recorded for this meta analysis for the two factors were very similar within studies.

5. Conclusion

The results of the present meta-analysis suggest an overall negative and largely small degree of association between OCD symptom severity and cognitive function. Only some subdomains and some test outcome measures reached a medium degree of association—all of which represented single-digit explained variance. These largely homogeneous results suggest that OCD symptom severity in and of itself plays a minor role in affecting cognitive functions. Nevertheless, the present investigation raises some important conceptual and methodological issues that hinder our ability to suggest a clear conclusion based on these results. These include partial presentation of results, poor ecological validity of neuropsychological tests in OCD and across populations, and assessment of symptom severity that includes problematic items and that focuses on the past seven days, rather than current symptoms at the time of testing. In sum, employing gold standard meta-analytic methodologies in the present study lends validity and credibility to our results, but caution should be exercised when interpreting these results. The outlined steps offered above should be implemented to improve future inferences from such data.

Authors contribution

AA conceived the study, oversaw the literature search and data recording, conducted the statistical analyses and drafted the first version of the manuscript. Authors MJ, DB, NW, and BM conducted the literature search, and data recording, as well as assisted in manuscript preparation.

Declarations of interest

None.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cpr.2018.09.003.

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