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Obsessive-Compulsive Disorder Is Associated With Broad Impairments in Executive Function: A Meta-Analysis

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Abstract

Obsessive-compulsive disorder (OCD) is a serious and often chronically disabling condition. The current dominant model of OCD focuses on abnormalities in prefrontal-striatal circuits that support executive function (EF). Although there is growing evidence for EF impairments associated with OCD, results have been inconsistent, which makes the nature and magnitude of these impairments controversial. The current meta-analysis uses random-effects models to synthesize 110 studies in which participants with OCD were compared with healthy control participants on at least one neuropsychological measure of EF. The results indicate that individuals with OCD are impaired on tasks measuring most aspects of EF, consistent with broad impairment in EF. EF deficits were not explained by general motor slowness or depression. Effect sizes were largely stable across variation in demographic and clinical characteristics of samples, although medication use, age, and gender moderated some effects.

Keywords

obsessive-compulsive disorder, executive function, meta-analysis

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Obsessive-compulsive disorder (OCD) is a serious and often chronically debilitating condition that affects 2% to 3% of the population (e.g., Kessler et al., 2005). OCD is characterized by obsessions (intrusive, distressing, and persistent thoughts and images) that are often accompanied by compulsions (ritualized, repetitive behaviors or mental acts) performed in an attempt to avoid or neutralize the distress resulting from obsessions or according to rules that must be applied rigidly (American Psychiatric Association, 2000). Neuroimaging research has emphasized neurobiological abnormalities that may underlie the clinical and neuropsychological symptoms of OCD. Indeed, the current dominant model of OCD focuses on abnormalities in prefrontal-striatal circuits (see Menzies, Chamberlain, et al., 2008, for review) that support executive function (EF). EFs are a set of general-purpose cognitive-control abilities, mainly supported by the prefrontal cortex (PFC), that allow individuals to regulate their thoughts and behaviors (e.g., Miyake & Friedman, 2012). EF regulates lower-level cognitive processes (e.g., perception, motor responses) and thereby enables self-directed behavior toward a goal (e.g., Banich, 2009), which allows individuals to break out of habits, make decisions and evaluate risks, plan for the future, prioritize and sequence actions, and cope with novel situations. EF deficits thus have important consequences for daily-life functioning and may be major contributors to the lack of cognitive flexibility and the perseverative, repetitive behaviors that are cardinal symptoms of OCD.

EF is best characterized as a set of separable but related cognitive processes that have both unique and shared individual differences, genetic influences, and neural substrates (e.g., Collette et al., 2005; Friedman

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et al., 2008; Miyake et al., 2000). One influential model of EF is the *unity/diversity model* (Friedman et al., 2008; Miyake et al., 2000; Miyake & Friedman, 2012), in which three fundamental aspects of EF are identified: (a) updating working memory (WM), (b) shifting (e.g., between tasks), and (c) inhibition, as well as a common EF ability (which is related to both updating and shifting and may subsume inhibition; Friedman et al., 2008; Miyake & Friedman, 2012). *Updating* is defined as monitoring and coding incoming information for task relevance and replacing no-longer-relevant information with newer, more relevant information. *Shifting* is defined as switching between task sets or response rules. *Inhibition* is defined as suppressing or resisting a prepotent (automatic) response to make a less automatic but task-relevant response. Common EF is posited to be the ability to monitor for and maintain goal and context information (Miyake et al., 2000; Miyake & Friedman, 2012). This hypothesis regarding the nature of common EF is compatible with the view that the central role of the frontal lobes is active maintenance of goals, plans, and other task-relevant information, which may be essential for all aspects of EF (e.g., Miller & Cohen, 2001). Critically, the unity/diversity model of EF may be a useful vantage for the investigation of cognitive deficits and biases in psychopathology, given that disorders such as OCD may be characterized by general (e.g., difficulty maintaining goals) or specific (e.g., difficulty shifting to a new set of behaviors) deficits in EF.

Although updating, shifting, and inhibition are important aspects of EF, this model in no way posits that these are the only components of EF. For example, WM is often considered a component of EF. *WM* is defined as maintaining or manipulating information across a short delay when that information is not available in the environment. WM-maintenance tasks (e.g., simple forward-span tasks) require keeping information in mind only temporarily (i.e., "holding on-line") and involve subsystems for active rehearsal and storage, whereas WM-manipulation tasks (e.g., complex and backward-span tasks) additionally require the reorganization of the information being maintained (e.g., Fletcher & Henson, 2001).[1] WM manipulation is strongly linked to other aspects of EF, whereas WM maintenance (sometimes called short-term memory) is less closely linked to other aspects of EF (e.g., Engle, Tuholski, Laughlin, & Conway, 1999). WM can also be divided into verbal and visuospatial components (e.g., Baddeley, 1992, 1996; Repovs & Baddeley, 2006). Given evidence for impaired visuospatial ability (e.g., block-design and design-copying tasks; Abramovitch, Abramowitz, & Mittelman, 2013) in individuals with OCD, which might affect visuospatial WM, it is thus important to evaluate visuospatial and verbal WM separately. In sum, when the unity/diversity model is applied to a clinical population, it is important to keep in mind additional domains of EF that may be affected in a particular disorder.

One challenge for the investigation of EF in clinical (or, indeed, any) populations stems from the fact that many complex tasks may tap multiple aspects of EF. For example, verbal-fluency tasks (the generation of words starting with a certain letter or from a category) likely tap several cognitive processes (Rende, Ramsberger, & Miyake, 2002). However, they have been shown to form a distinct component separable from other EF components (Fisk & Sharp, 2004) and to depend on prefrontal function (e.g., Alvarez & Emory, 2006). Planning tasks are also complex, involving multiple cognitive demands (e.g., Goel & Grafman, 1995), and so may not represent a single EF ability. It is notable that verbal-fluency and planning tasks are frequently used in clinical studies, including studies of individuals with OCD. Such tasks may be commonly implemented in clinical research because they are viewed as more ecologically sensitive: The complexity of verbal-fluency and planning tasks may make them more relatable to real-world tasks that require similar skills. Thus, there are both disadvantages (in terms of interpretability) and advantages (in terms of ecological validity) in the use of such complex EF tasks.

Although there is growing evidence for EF impairments associated with OCD, results have been inconsistent, thereby causing controversy about the nature and magnitude of these impairments (for review, see Chamberlain, Blackwell, Fineberg, Robbins, & Sahakian, 2005; Kuelz, Hohagen, & Voderholzer, 2004; Menzies, Chamberlain, et al., 2008; Olley, Malhi, & Sachdev, 2007). In two recent meta-analyses that target cognitive function more broadly in OCD, researchers found some evidence of impaired EF but inconsistent effect sizes, likely due to the differences in the way they operationalized EF. Specifically, Abramovitch et al. (2013) grouped tasks into composite measures of planning (d = 0.44), response inhibition (d = 0.49), set shifting/cognitive flexibility (which included verbal and design fluency and Wechsler Adult Intelligence Scale similarities in addition to traditional measures of shifting; d = 0.52), and verbal (d = 0.34) and spatial (d = 0.37) WM (which included measures of updating, in addition to WM maintenance and manipulation). Individual tasks and measures were not analyzed separately. N. Y. Shin, Lee, Kim, and Kwon (2014) included some individual EF tasks, and in comparison with Abramovitch et al., found a much larger effect for planning (d = 0.73), smaller effects on shifting tasks (d = 0.31–0.51) and verbal WM (d = 0.11), and somewhat comparable effects on inhibition (d = 0.55) and spatial WM (d = 0.45). In addition, both meta-analyses included a small number of studies in most EF analyses (e.g., 12 and 6 studies, respectively, for planning,

compared with 28 in the current meta-analysis), which potentially accounts for the variability in effect sizes. Such inconsistencies suggest the need for a larger-scale meta-analysis than has previously been performed to improve the reliability of EF estimates. The present analysis also uses the well-articulated unity/diversity model of Miyake and Friedman, in conjunction with other perspectives on EF, to provide a more specific rationale for decomposing EF tasks.

Specifically, in addition to variable effect sizes, previous meta-analyses have reported considerable variability in the specific pattern of impairment across different components of EF. Such differences may derive from discrepancies in how EF was operationalized (e.g., how domains of EF were defined). In one case, measures were combined into composites that do not conform to established models of EF, such as the unity/diversity model (e.g., fluency tasks, which tap multiple aspects of EF, were grouped with shifting tasks, and updating tasks were grouped with WM tasks); in the other case, only a handful of individual EF tasks were included, and composite measures were not analyzed. In sum, although these previous meta-analyses are valuable in providing a survey of cognitive function in OCD more broadly, they do not permit testing specific hypotheses about EF impairment in OCD.

The current meta-analysis thus addresses these limitations in the extant literature by taking a theoretically driven approach, applying well-established models of EF to comprehensive analyses, to test competing hypotheses about the nature of EF impairments associated with OCD. At least four hypotheses regarding executive dysfunction in OCD have been proposed. These hypotheses posit that individuals with OCD have (a) a broad impairment in EF, (b) specific impairments in the shifting or inhibition components of EF, (c) general slowing of motor responses that accounts for apparent EF deficits, or (d) co-occurring depression that accounts for EF deficits.

Hypothesis 1: Broad Impairment in EF

Evidence exists that individuals with OCD have abnormalities in a prefrontal-striatal network that is critical for EF, thereby suggesting that EF may be broadly impaired in individuals with OCD. A meta-analysis of functional MRI studies that reported case-control comparisons during a variety of cognitive tasks has shown evidence for activation abnormalities in a wide PFC network, including the anterior cingulate cortex, the lateral PFC, and the orbitofrontal cortex, as well as in the striatum (caudate and putamen; Menzies, Chamberlain, et al., 2008). For many different EF tasks, there is joint recruitment of these regions (e.g., Duncan & Owen, 2000). Meta-analyses of neuroimaging studies have shown reliable activation of dorsal and ventral lateral PFC and anterior cingulate cortex for inhibition (Nee, Wager, & Jonides, 2007), shifting (Wager, Jonides, & Reading, 2004), WM (Wager & Smith, 2003), and verbal fluency (Costafreda, David, & Brammer, 2009), and a narrative review concluded that these regions were also active for planning (Collette, Hogge, Salmon, & Van der Linden, 2006). Orbitofrontal cortex has been implicated in evaluating the reward probabilities associated with different response options (e.g., see Krain et al., 2006, for a meta-analysis) and, thus, could affect performance across EF tasks, especially if response feedback or reward is involved.

Thus, the frontal-striatal model of OCD would predict broad impairment across multiple aspects of EF that are all supported by the prefrontal areas that are altered in individuals with OCD. In support of this hypothesis, meta-analyses have reported deficits on a wide variety of EF tasks in individuals with OCD (Abramovitch et al., 2013; N. Y. Shin et al., 2014). However, as noted earlier, the magnitude of these effects is inconsistent across previous meta-analyses, and composite measure analyses conforming to established models of EF were not conducted, which makes it impossible to effectively compare the magnitude of deficits across different aspects of EF. Thus, the breadth and magnitude of EF impairments associated with OCD has not been clearly established, and other researchers have argued that such impairments are an artifact of co-occurring depression or general motor slowing (see discussions of Hypotheses 2–4).

Hypothesis 2: Specific Impairment in Shifting or Inhibition

Given that highly repetitive behaviors and thoughts are the hallmarks of OCD, researchers have proposed that individuals with OCD have particular difficulty shifting attention between different cognitive representations and behaviors or inhibiting inappropriate responses (e.g., Bannon, Gonsalvez, Croft, & Boyce, 2002; Chamberlain et al., 2005; Olley et al., 2007). Hypothesis 2 is distinct from Hypothesis 1 in that it does not predict equivalent impairment on other aspects of EF, which would show deficits only to the extent that tasks designed to assess other aspects of EF also tap inhibition or shifting (e.g., planning tasks may require inhibiting incorrect moves and verbal-fluency tasks may require shifting between subcategories). Thus, Hypothesis 2 is inconsistent with the general EF impairment posited by Hypothesis 1. Although impairments on shifting and inhibition tasks have been reported (Abramovitch et al., 2013; N. Y. Shin et al., 2014), it is unclear whether the magnitude of these deficits is larger than the deficits detected in other aspects of EF; a pattern would more clearly suggest the presence of impairments specific to shifting and inhibition.

Hypothesis 3: Apparent EF Deficits Are Due to General Motor-Response Slowing

Individuals with OCD are often significantly slower than are healthy individuals in completing everyday tasks, such as eating and dressing (e.g., Hymas, Lees, Bolton, & Head, 1991), and may perform more poorly on timed than on untimed tasks (e.g., Alarcón, Libb, & Boll, 1994). Researchers thus have proposed that individuals with OCD show a general slowing of motor responses, potentially because of abnormalities in the neuromotor system (e.g., Hymas et al., 1991). However, other researchers have argued that response slowing in individuals with OCD is limited to EF tasks and is not secondary to OCD symptoms, such as checking (e.g., Bucci et al., 2007). In addition, there has been no systematic evaluation of whether individuals with OCD are significantly impaired on untimed, accuracy-based measures of EF, which would suggest that EF deficits cannot be attributed to general motor slowing. Critically, previous meta-analyses did not conduct separate analyses of accuracy measures (Abramovitch et al., 2013) or included only a few accuracy-based measures, which were not compared with measures based on reaction time (RT; N. Y. Shin et al., 2014).

A broader impairment in *general processing speed* has also been proposed to account for impaired task performance associated with psychopathology (i.e., that the rate of processing limits performance on higher-level operations because if processing steps are carried out too slowly, the products of earlier operations may be lost or no longer relevant by the time later operations occur; Nebes et al., 2000). However, in its current form, this hypothesis is not empirically falsifiable. Given that cognitive slowing is posited to affect even untimed and unspeeded tasks, impairments on self-paced accuracy measures of EF would not be considered evidence against this hypothesis, nor would greater impairment on EF tasks than on processing-speed tasks, given that it is always possible to argue that more complex tasks may require more processing steps and are therefore more affected by cognitive slowing. Thus, although the motor-speed hypothesis can be empirically evaluated (including by meta-analyses), evaluation of the general-processing-speed hypothesis must await more complete specification of the theory in a way that makes it empirically falsifiable.

Hypothesis 4: Apparent EF Deficits Are Due to Co-Occurring Depression

There is a high rate of comorbidity between OCD and depression; more than 70% of individuals with a primary diagnosis of OCD also experience a mood disorder during their lifetime (61% experience a major depressive disorder, MDD; Brown, Campbell, Lehman, Grisham, & Mancill, 2001). Depression is also associated with broad impairments in EF (see Snyder, 2013, for a meta-analysis). Thus, some researchers have argued that EF deficits in individuals with OCD are due to co-occurring depression rather than to OCD per se (e.g., Basso, Bornstein, Carona, & Morton, 2001). Indeed, several studies have shown that the effects of OCD on EF were no longer significant after controlling for co-occurring depressive symptoms (Aycicegi, Dinn, Harris, & Erkmen, 2003; Basso et al., 2001; Moritz, Kloss, Jahn, Schick, & Hand, 2003). However, other studies have shown that co-occurring depressive symptoms do not account for EF deficits in individuals with OCD (Abramovitch, Dar, Schweiger, & Hermesh, 2011; Nedeljkovic et al., 2009), and most studies have not investigated the effects of co-occurring depression.

Current Meta-Analysis

In the current meta-analysis, we synthesized previous research findings and applied well-established models of EF to test the four hypotheses outlined in the previous sections. In addition, we examined the potential moderating effects of demographic (age and gender) and clinical (OCD symptom severity, psychotropic medication use, and co-occurring depression) variables on EF effect sizes. Findings are discussed in light of the barriers that may limit interpretation of the prior literature, and suggestions for potential solutions and future directions are presented.

Method

Inclusion and exclusion criteria

Studies eligible for inclusion were required to include a group of individuals with a diagnosis of OCD and a healthy control group with no diagnosed psychopathology. Studies were included if researchers tested participants on at least one EF task and reported sufficient information to calculate effect sizes. EF tasks were defined as detailed in the Coding Procedures section. Studies were excluded from analysis if they investigated OCD in samples of participants with organic brain damage (e.g., after a head injury).

Search strategies

Searches were conducted in PubMed and ISI Web of Science for articles published through October 2013 using the keywords *obsessive compulsive* paired with *executive function, working memory, response inhibition, inhibitory*

control, shifting, task switching, planning, verbal fluency, cognitive, or *neuropsychological*. In addition, a search for unpublished studies was conducted by e-mailing the corresponding authors of articles included in the meta-analysis and searching ProQuest for unpublished dissertations and master's theses. The first author conducted the search and screening procedures. An initial screen for study eligibility was conducted by examining titles to eliminate studies that clearly did not meet the inclusion criteria. Next, the abstracts of all remaining articles were examined, and if an article appeared likely to meet the inclusion criteria, the full text was obtained and checked for inclusion criteria. In addition, the reference lists of included articles, and articles citing included articles, were screened for any studies missed in the database search process. Publication bias was assessed using the trim-and-fill method (Duval & Tweedie, 2000).

Coding procedures

Tasks. The types of tasks used in the included studies determined the specific aspects of EF covered in the present meta-analysis. Tasks were coded as tapping one of the following EF components, as detailed later: shifting, inhibition, updating, verbal and visuospatial WM, planning, and verbal fluency. This list is not meant to be exhaustive of all EF abilities but, rather, includes all the EF tasks commonly included in the OCD literature. The first author, who is highly experienced with EF research, coded all studies. In addition, for 25% of studies, the coauthors, who are also highly experienced with EF research, coded the EF component measured by each EF task, blind to the first author's coding. Intercoder agreement for the included EF tasks was high (99%); thus, the first author's coding was used in all analyses.[2] Descriptions of the included tasks tapping each EF construct, their dependent measures, and the number of studies reporting each are provided in Table 1. For each construct, all tasks were included in a composite measure analysis. All tasks and measures reported by at least three studies were also analyzed individually in separate analyses.

In addition, two types of motor-speed measures reported by studies in the meta-analysis were included to determine whether there is general motor slowing associated with OCD, as proposed by Hypothesis 3. The Trail Making Test Part A (TMT-A; $k = 32$) shares the motor-speed and sequencing demands of the Trail Making Test Part B (TMT-B) but does not require shifting like the TMT-B. In addition, 10 studies reported one or more general motor-speed measures, including simple RT ($k = 4$), choice RT ($k = 2$), finger tapping (tap fingers as quickly as possible; $k = 1$), and grooved pegboard (put pegs into holes in a board as quickly as possible; $k = 4$). These tasks were included in a general motor-speed composite score.

Moderator variables. Information was coded on current OCD symptom severity, age, gender, psychotropic medication use, and co-occurring depression.

Symptom severity. Total Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989) scores were reported by 81% of studies. The Y-BOCS is the most frequently used questionnaire to assess OCD symptom severity and has good reliability and validity (Goodman, Price, Rasmussen, Mazure, Delgado, et al., 1989; Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989). It is a clinician-rated 10-item scale, with each item rated from 0 (*no symptoms*) to 4 (*severe symptoms*), that provides a total range of 0 to 40.

Age. The mean age of the OCD group was included as a continuous variable in metaregression analyses. Age was reported by all studies.

Gender. The percentage of females in the OCD group was included as a continuous variable in metaregression analyses. Gender was reported by 96% of studies.

Medication. The percentage of the OCD group currently taking psychotropic medications was coded for each sample. Medication usage was reported by 81% of studies. Many studies reported only the total number of participants using medication; thus, a more detailed analysis of the types or duration of medication use could not be conducted. However, when type was reported, medications were generally antidepressants.

Co-occurring depression. Because of the diversity of depression measures reported and the lack of detailed depression reporting in many studies, continuous measures of co-occurring depressive symptoms could not be analyzed. Instead, the presence of co-occurring depression or depressive symptoms in the sample was coded as a categorical variable. The sample was coded as containing individuals with co-occurring depression or depressive symptoms if (a) any OCD participants were reported to have a comorbid diagnosis of a depressive disorder or (b) mean depressive symptoms on a standard depression questionnaire were reported in the clinical range. The sample was coded as containing participants without co-occurring depression or depressive symptoms only if neither of the prerequisites just noted were met (given that the absence of diagnosed depression does not preclude clinically significant levels of depressive symptoms). The clinical range was defined as follows, using published

Table 1. EF Tasks Included in the Meta-Analysis

Construct and task	Description	Outcome measure	Studies (*n*)
Shifting			
Intradimensional/ extradimensional shift	Learn from feedback to select a stimulus based on one dimension, switch to the previously nonrewarded stimulus (intradimensional shift), then switch to a different stimulus dimension (extradimensional shift).	†1. Perseverative errors in intradimensional and extradimensional shifts 2. Number of shifts achieved	7^
Trail Making Test Part B	Alternately connect letters and numbers in sequence (A-1-B-2 etc.). Often compared with Trail Making Test Part A (connect letters or numbers only, does not require shifting).	†1. Trail Making Test Part B – Trail Making Test Part A time 2. Time to complete B	37^
Object Alternation Test/ Delayed Object Alternation Test	Find object hidden alternately under two different cups immediately or after short delay.	Errors	17^
Wisconsin Card Sorting Test	Learn from feedback to sort cards by one dimension (e.g., color) and then switch to a different dimension (e.g., shape) when given negative feedback on the first dimension (repeats with multiple sorting rules).	†1. Perseverative errors 2. Number of rules achieved	42^
Cued-task switching	Perform one of two tasks depending on cue before each trial (e.g., color/shape).	Switch cost (switch – repeat RT)	3^
Inhibition			
Color-word Stroop	Identify the ink color a colored word is printed in. Trials are incongruent (e.g., "red" written in blue ink) or neutral (noncolor word or color patches).	†1. Stroop interference (incongruent – neutral RT) 2. Incongruent condition time 3. Accuracy	28^
Stop signal	Quickly categorize and respond to stimuli (e.g., left- and right-pointing arrows) while withholding responses when a stop signal is presented.	Stop signal RT (time needed to stop a response)	6^
Go/no-go	Quickly categorize and respond to stimuli (e.g., left- and right-pointing arrows) while withholding responses to another stimulus.	Commission (no-go) errors	11^
Antisaccade	Look in the opposite direction of visual cue.	Errors (prosaccades)	2
Hayling	Read sentences in which the final word is omitted but highly predictable. First complete sentences correctly (Part A), then complete with an unrelated word (Part B).	Part B – Part A RT	2
Updating			
n-Back	Indicate whether the stimulus (usually a letter) matches the stimulus *n* (e.g., 3) items back.	Accuracy	4^
Visuospatial working memory			
Block span (spatial span, Corsi block tapping)	Tap irregularly arranged blocks/squares in the same order as experimenter (Corsi blocks) or computer (spatial span).	Span (maximum length of sequence correctly performed)	14^
Self-ordered pointing	Search an array of boxes for hidden tokens. Token is in each location only once.	†1. Within-search errors (return to previous location) 2. Strategy score (how often search is initiated from same starting box)	11^
Delayed match-to-sample	Maintain a complex spatial pattern across a delay and indicate whether a probe matches it.	Accuracy	3^

(continued)

Table 1. (continued)

Construct and task	Description	Outcome measure	Studies (*n*)
Verbal working memory			
Digit-span forward	Repeat sequence of numbers in forward order.	Span (maximum length of sequence correctly performed)	19^
Digit-span backward	Repeat sequence of numbers in reverse order.	Span (maximum length of sequence correctly performed)	11^
Letter-number sequencing	Repeat list of alternating letters and numbers, resequenced into numbers first, then letters.	Span (maximum length of sequence correctly performed)	2
Reading span	Read a series of sentences while remembering the last word in each sentence. Some versions also require verifying whether each sentence is true or false.	Number of correctly recalled words	2
Operation span	Solve a series of math operations while trying to remember a series of unrelated words.	Number of correctly recalled words	1
Self-ordered pointing with words	Point to a different word in an array on each trial.	Between-search errors	1
Verbal fluency			
Semantic verbal fluency	Say as many words from a semantic category (e.g., animals) as possible in 1 (or 3) min.	Number of words	17^
Phonemic verbal fluency/ Controlled Oral Word Association	Say as many items starting with a certain letter (usually F, A, S) as possible in 1 (or 3) min.	Number of words	35^
Planning			
Tower of London/Tower of Hanoi	Move rings on pegs from a starting position to a target position in as few moves as possible following a set of rules.	†1. Number of moves in excess of minimum 2. Number of problems solved in minimum moves	28^

Note: Daggers indicate preferred measure if reported. Carets indicate analyzed individually as well as part of composite measure. RT = reaction time.

cut-point norms: Hamilton Depression Rating Scale (> 7; Kearns et al., 1982), Montgomery–Asberg Depression Rating Scale (> 7; Kearns et al., 1982), Beck Depression Inventory (> 9; Beck, 1978), Beck Depression Inventory-II (> 13; Beck, Steer, & Brown, 1996), and Children's Depression Inventory (> 12; Kovacs, 1983). Applying both criteria, 55% of samples were coded as having co-occurring depression/depressive symptoms and 18% as having no co-occurring depression/depressive symptoms; 27% of samples did not report enough information to allow for coding. Co-occurring depression was included as a categorical variable in meta-analyses of variance whenever there were at least three studies in the smaller category. In addition, samples with no co-occurring depression/depressive symptoms were analyzed separately to provide a conservative test of the hypothesis that EF deficits in OCD are driven by co-occurring depression.

Statistical methods

For each study, effect sizes comparing the performance of the OCD and control groups on each EF measure were calculated as Cohen's *d*; the sign was set such that a positive value always indicated poorer performance for the OCD group relative to the control group (e.g., lower accuracy, higher error rates, or longer RTs). Before analyses were conducted, effect sizes were adjusted, weighted, and screened for outliers. First, given that it has been demonstrated that Cohen's *d* is slightly overestimated when sample sizes are small, Hedges's (1980) small-sample-bias correction was applied: $d_{adj} = d[1 - (3/4N) - 9]$, where N is the number of participants in both samples combined. Second, because sampling error is also higher for smaller sample sizes, effect sizes were weighted by sample size using inverse variance weights: $w = [2(n_1 + n_2)n_1n_2]/[2(n_1 + n_2)^2 + n_1n_2d^2]$, where n_1 and n_2 are the number of participants in the OCD and control groups, respectively. Finally, analyses were screened for outliers with effect sizes 3 *SD* above and below the mean effect size in each analysis. Only three such outliers were detected: One was excluded from the WCST and shifting composite analysis, one from the digit-span-forward and verbal-WM composite analyses, and one from the phonemic VF analysis.

Only one effect size from each sample comparison was included in each analysis to avoid statistical dependence.

If three or more studies reported a particular task, individual tasks were analyzed separately, as described in the Coding Procedures section. In addition, composite effect sizes were calculated by averaging effect sizes within a construct (e.g., all inhibition measures). In addition, if individuals were tested more than once (e.g., at different points in treatment), only task performance at the first testing time was analyzed, given that practice effects may diminish the EF demands of tasks.

Random-effects meta-analytic models were used for all analyses. Although many meta-analyses have used fixed-effects models in the past, random-effects models are now considered more appropriate because there are likely to be many sources of variability between study samples beyond sampling error, which violates the assumptions of fixed-effects models (Raudenbush, 2009). It is important that random-effects models allow inferences to be drawn about a broader population of studies rather than just about the samples tested.

Analyses were conducted using the SPSS meta-analysis macro developed by David B. Wilson (2006). For each analysis, weighted mean effect sizes with 95% confidence intervals were calculated. The null hypothesis that the mean effect size is 0 was tested with the z statistic at the alpha significance level of .05. Heterogeneity in effect sizes was tested with the Q_t statistic (Hedges & Olkin, 1985). Q_t quantifies the degree to which the studies contributing to each weighted mean effect size can be considered homogeneous. If Q_t is significant, it suggests that there are substantive differences between the studies in that analysis. Publication bias was assessed with the trim-and-fill method (Duval & Tweedie, 2000). In addition, separate analyses were conducted including only published studies to determine whether the inclusion of unpublished studies affected the results. To ensure that effects were not driven by failure to match OCD and control samples on IQ, we conducted separate analyses including only those studies that reported that IQ did not significantly differ between groups.

Moderator analyses were conducted via mixed-effects models with method-of-moments estimation. Current symptom severity (Y-BOCS), OCD group age, and medication status (percentage receiving psychotropic medications) were included as continuous variables in separate and combined metaregression analyses. Co-occurring depression (individuals with clinically significant levels of depressive symptoms or depressive disorder diagnosis) was included as a categorical variable in meta-analyses of variance if there were at least three studies in the smaller category. Moderator analyses were conducted only for measures with 20 or more effect sizes, given that analyses with fewer studies have inadequate power and may produce unstable estimates (Marín-Martínez & Sánchez-Meca, 1998; Sánchez-Meca & Marín-Martínez, 1998).

Results

The search process identified 110 studies for inclusion, 104 of which were published in peer-reviewed journals and 6 of which were unpublished. An additional 20 studies were screened but were excluded from analysis because they did not report sufficient information to calculate effect sizes (k = 10), did not include a healthy control group (k = 4), included the same data as another article in the meta-analysis (k = 3), prematched/pretrained participants on task performance (k = 2), or used tasks not clearly classifiable into an EF component (k = 1; see Table S1 in the Supplemental Material available online). Included studies are marked with an asterisk in the reference list. The full data sets used in the analyses are available from the authors on request.

In total, the included studies comprised 6,315 participants (3,162 individuals with OCD and 3,153 healthy control participants). On average, individuals with OCD in the included studies were 31.36 years old (SD = 7.16, range = 12–49),[3] and there were equal numbers of males (49.86%) and females (50.14%) with OCD (SD = 20.40, range = 0–100%). Across studies, the average Y-BOCS (Goodman, Price, Rasmussen, Mazure, Fleischmann, et al., 1989) score was 23.07 (SD = 4.32, range = 3–30), which corresponds to a moderate level of symptom severity, and 38.90% of individuals with OCD were currently taking psychotropic medications (SD = 37.35, range = 0–100%).

Weighted mean effect sizes

Individuals with OCD performed more poorly than did healthy control participants on most EF tasks, but the magnitude of these impairments varied somewhat depending on the aspect of EF and the task. Table 2 reports the effect size (d) for each measure, along with the 95% confidence intervals and significance test; these effect sizes, with 95% confidence intervals, are also plotted in Figure 1. Table 2 also provides the test for homogeneity of effect sizes across samples (Q) and tests for sensitivity of the results to publication bias, inclusion of unpublished studies, and IQ matching, as detailed in the following sections.

Inhibition. There were significant small-to-medium effects of OCD groups compared with healthy groups for the inhibition composite measure (d = 0.37), Stroop incongruent condition time (d = 0.55), interference (d = 0.36) and accuracy (d = 0.39), and stop signal RTs (d = 0.62) but only a small and nonsignificant effect for accuracy on the go/no-go task (d = 0.24).

Shifting. There was a moderate and significant effect of group for shifting composite scores (d = 0.50). Examining

Table 2. Mean Effect Size Analyses

Measure	*N*	*k*	*d*	95% CI		*SE*	*z*	*p*	Homogeneity test				Publication bias		
				LL	UL				*v*	*Q*	*df*	*p*	*d* Trimmed and filled (*k* trimmed)	*d* Published only (*k*)	*d* IQ matched only (*k*)
Inhibition															
Composite score	2,753	50	0.37*	0.24	0.51	0.07	5.35	< .001	0.15	141.01	49	< .001	0.37* (0)	0.40* (46)	0.43* (24)
Stroop (incongruent time)	861	16	0.55*	0.21	0.90	0.18	3.14	.002	0.39	85.36	15	< .001	0.55* (0)	0.55* (16)	0.99 (4)
Stroop (interference)	1,326	18	0.36*	0.17	0.56	0.10	3.72	.002	0.10	44.89	17	< .001	0.36* (0)	0.40* (17)	0.53* (7)
Stroop (accuracy)	240	6	0.39*	0.11	0.67	0.14	2.73	.006	0.00	3.62	5	.606	0.39* (0)	0.39* (6)	0.38 (3)
Stop signal (reaction time)	248	6	0.62*	0.23	1.01	0.20	3.14	.002	0.13	10.80	5	.055	0.62* (0)	0.60* (5)	0.62* (6)
Go/no-go (no-go accuracy)	438	11	0.24	–0.07	0.55	0.16	1.51	.132	0.16	25.14	10	.005	0.24 (0)	0.18 (10)	0.41 (5)
Shifting															
Composite score[a]	4,762	74	0.50*	0.42	0.58	0.04	12.23	< .001	0.05	122.35	73	< .001	0.36* (19)	0.49* (68)	0.53* (36)
ID/ED shift (accuracy)	435	7	0.50*	0.31	0.70	0.10	5.13	< .001	0.00	1.99	6	.921	0.48* (1)	0.49* (6)	0.53* (5)
OAT/DAT (accuracy)	1,131	17	0.32*	0.15	0.49	0.09	3.72	< .001	0.05	29.07	16	.023	0.23* (4)	0.29* (15)	0.30* (9)
TMT-B (time)	2,659	37	0.54*	0.42	0.66	0.06	8.67	< .001	0.07	76.69	36	< .001	0.54* (0)	0.53* (33)	0.59* (16)
WCST (perseverative errors)[b]	2,706	42	0.44*	0.33	0.55	0.06	7.82	< .001	0.06	75.80	41	< .001	0.29* (12)	0.45* (39)	0.46* (20)
Cued-task switching (switch cost)	102	3	0.35	–0.05	0.76	0.21	1.70	.089	0.00	0.53	2	.767	0.35 (0)	0.35 (3)	—
Updating															
n-Back	375	4	0.71*	0.26	1.15	0.23	3.13	.002	0.12	8.08	3	.044	0.61* (1)	0.71* (4)	—
Verbal WM															
Composite score	1,673	24	0.22*	0.08	0.36	0.07	3.06	.002	0.06	46.30	24	.004	0.22* (0)	0.22* (22)	0.21 (11)
Manipulation composite score	942	14	0.31*	0.10	0.52	0.11	2.87	.004	0.09	31.22	13	.003	0.26* (1)	0.31* (12)	0.22 (8)
Digit-span backward (span)	771	11	0.21	0.00	0.42	0.11	1.95	.052	0.06	20.01	10	.029	0.21 (0)	0.20 (9)	0.11 (7)
Maintenance composite score[c]	1,206	19	0.07	–0.06	0.20	0.07	1.02	.310	0.02	25.74	18	.106	0.06 (1)	0.07 (17)	0.01 (8)
Digit-span forward (span)[c]	1,206	19	0.08	–0.05	0.20	0.06	1.21	.225	0.02	24.29	18	.146	–0.07 (7)	0.07 (17)	0.03 (8)

(continued)

Table 2. (Continued)

Measure	*N*	*k*	*d*	95% CI		*SE*	*z*	*p*	Homogeneity test				Publication bias		
				LL	UL				*v*	*Q*	*df*	*p*	*d* Trimmed and filled (*k* trimmed)	*d* Published only (*k*)	*d* IQ matched only (*k*)
Visuospatial WM															
Composite score	1,483	24	0.47*	0.31	0.62	0.08	6.01	< .001	0.07	44.10	23	.005	0.47* (0)	0.48* (21)	0.47* (12)
Block span (span)	1,107	14	0.43*	0.27	0.59	0.08	5.43	< .001	0.03	18.89	13	.13	0.33* (3)	0.43* (12)	0.41* (8)
Self-ordered pointing (accuracy)	596	11	0.62*	0.37	0.87	0.13	4.93	< .001	0.09	20.89	10	.022	0.56* (1)	0.72* (9)	0.74* (8)
DMTS (accuracy)	122	3	0.49	–0.10	1.09	0.30	1.62	.106	0.16	4.68	2	.096	0.49 (0)	0.49 (3)	—
VF															
Composite score	2,681	40	0.36*	0.26	0.47	0.05	6.79	< .001	0.04	62.91	39	.009	0.29* (7)	0.37* (37)	0.42* (19)
Phonemic (words)[d]	2,462	35	0.39*	0.31	0.47	0.04	9.17	< .001	0.00	30.39	34	.645	0.39* (0)	0.38* (33)	0.41* (18)
Semantic (words)	1,088	17	0.34*	0.11	0.57	0.12	2.96	< .001	0.15	50.77	16	< .001	0.26* (2)	0.34* (17)	0.42* (8)
Planning															
Composite score	2,017	28	0.44*	0.31	0.57	0.07	6.42	< .001	0.06	55.05	27	.001	0.35* (6)	0.42* (26)	0.50* (17)
TOL/TOH (accuracy)	1,853	25	0.44*	0.27	0.61	0.09	5.11	< .001	0.12	71.85	24	< .001	0.36* (4)	0.41* (23)	0.53* (17)
TOL/TOH (movement latency)	850	12	0.42*	0.23	0.62	0.10	4.17	< .001	0.05	20.03	11	.045	0.29* (3)	0.44* (11)	0.50* (6)
Motor-speed task															
TMT-A (time)	2,297	32	0.57*	0.45	0.68	0.06	9.74	< .001	0.04	48.79	31	.022	0.49* (5)	0.55* (30)	0.60* (22)
General motor-speed composite score	514	10	0.34*	0.05	0.63	0.15	2.30	.021	0.13	23.12	9	.006	0.34* (0)	0.39* (8)	0.08 (3)

Note: Dashes indicate too few IQ-matched studies to conduct analysis ($k < 3$). N = number of participants; k = number of studies; d = weighted mean Cohen's d effect size; CI = confidence interval; LL = lower limit; UL = upper limit; v = random-effects variance component; Q = heterogeneity; ID/ED = intradimensional/extradimensional; OAT/DAT = Object Alternation Test/Delayed Object Alternation Test; TMT-B = Trail Making Test Part B; WCST = Wisconsin Card Sorting Test; WM = working memory; DMTS = delayed match-to-sample; VF = verbal fluency; TOL/TOH = Tower of London/Tower of Hanoi; TMT-A = Trail Making Test Part A.

[a]One outlier excluded ($d = 2.66$). With this outlier included, the weighted mean Cohen's d effect size is 0.53.

[b]One outlier excluded ($d = 2.66$). With this outlier included, the weighted mean Cohen's d effect size is 0.47.

[c]One outlier excluded ($d = 1.96$). With this outlier included, the weighted mean Cohen's d effect size is 0.14.

[d]One outlier excluded ($d = –0.58$). With this outlier included, the weighted mean Cohen's d effect size is 0.37.

*$p < .05$.

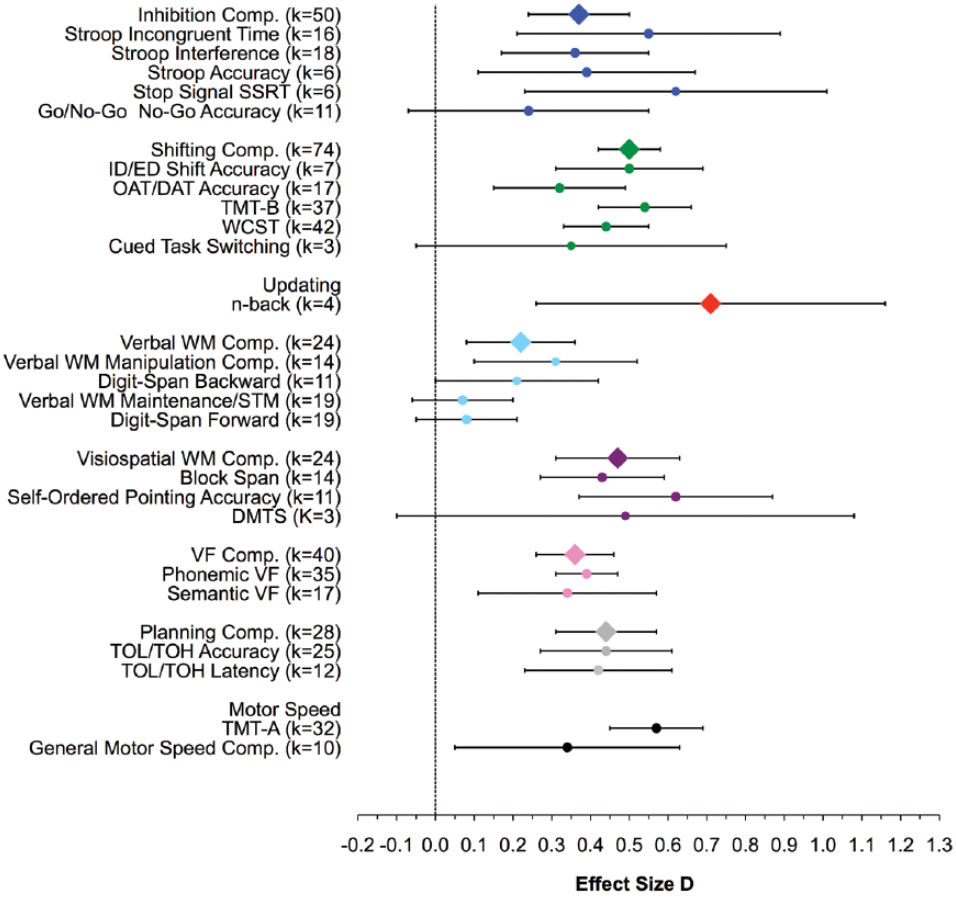

Fig. 1. Weighted mean effect sizes for all analyses. Error bars represent 95% confidence intervals. Measures for which the lower border of an error bar does not pass the vertical line are significantly greater than 0. Executive function (EF) composite measures are indicated with diamond symbols, and individual measures within each EF component are indicated by circle symbols in the same color. Black circles indicate non-EF comparison measures. *k* = number of studies; Comp. = composite score; ID/ED = intradimensional/extradimensional; OAT/DAT = Object Alternation Test/Delayed Object Alternation Test; TMT-B = Trail Making Test Part B; WCST = Wisconsin Card Sorting Test; WM = working memory; STM = short-term memory; DMTS = delayed match-to-sample; VF = verbal fluency; TOL/TOH = Tower of London, Tower of Hanoi; TMT-A = Trail Making Test Part A.

the individual shifting tasks, we found the largest effect size was for TMT-B (d = 0.54). However, the effect size was as large for the TMT-A, which does not require shifting (d = 0.57), which suggests that this effect may be primarily driven by slowed general motor speed or sequencing and not shifting per se. Thus, it may be more informative to focus on shifting tasks that are not confounded by general motor-speed demands because they are self-paced and have accuracy, rather than RT, outcome measures (see Table 1). These all have somewhat smaller but significant effects: intradimensional/extradimensional shift, d = 0.50; Object Alternation Test/Delayed Object Alternation Test, d = 0.32; and Wisconsin Card Sorting Test, d = 0.44. The only shifting task that did not show a significant effect was cued-task switching (d = 0.35), which was marginal; however, this should be interpreted with caution because there were few studies using this task (k = 3).

Updating. Only four studies tested updating, all with an n-back test. Nonetheless, there was a large effect size (d = 0.71), although confidence intervals are wide due to the small number of studies.

Verbal WM. There was a small but significant effect of group on overall verbal-WM composite scores (d = 0.22). Separating measures into those that require manipulation of information in WM versus simple WM maintenance, we found a small but significant effect of group on WM-manipulation composite scores (d = 0.31), with a marginal effect for digit-span backward (d = 0.21). There was no evidence for meaningful verbal-WM-maintenance

impairments in individuals with OCD. There were very small and nonsignificant effects for verbal-WM-maintenance composite scores (d = 0.07) and digit-span forward (d = 0.08).

Visuospatial WM. There were significant effects of OCD groups compared with healthy control groups for visuospatial-WM composite scores (d = 0.47), block span (d = 0.43), and self-ordered pointing (d = 0.62). The effect size for the delayed match-to-sample task (DMTS) was comparable in magnitude with those for block span and composite scores (d = 0.49) but did not reach significance because of the small number of studies using this task (k = 3).

Verbal fluency. There was a small but significant effect of group for verbal-fluency composite scores (d = 0.36), with comparable and significant effects for phonemic (d = 0.39) and semantic (d = 0.34) verbal fluency.

Planning. There was a significant effect for planning composite scores (d = 0.44). Given that some studies used latency measures (i.e., time until first move), which may be influenced by overall slowing, latency and accuracy measures were also analyzed separately. There were similar and significant effects for accuracy measures (d = 0.44) and latency measures (d = 0.42).

General motor speed. There was a significant effect of OCD groups compared with healthy groups for general motor-speed measures (d = 0.34), although a great deal of variability between studies led to a wide confidence interval (d = 0.05–0.63). As noted earlier, there was also a significant effect for the TMT-A—the comparison task for the TMT-B—which requires both general motor speed and sequencing (d = 0.57).

Heterogeneity analyses

There was significant heterogeneity among effect sizes for all measures except Stroop accuracy, stop signal (marginal), intradimensional/extradimensional shift, cued-task switching, verbal-WM maintenance, digit-span forward, block span, DMTS (marginal), and phonemic verbal fluency. There may be multiple sources of this variability. First, some variability is likely due to differences in methodology across studies. In composite score analyses, tasks are likely to vary in sensitivity (e.g., standard neuropsychological tests are less sensitive to subtle impairments than are those designed to assess individual differences in the normal range). Even in analyses of single tasks and measures, task versions may vary in sensitivity (e.g., the standard neuropsychological version of the Stroop task, with separate blocks of neutral and incongruent stimuli, is easier than versions in which trial types are intermixed). Given the myriad variations in tasks, it is not possible to account for this variation. Second, some variability is due to differences in the demographic characteristics of the patient groups included in each study (see the Moderator Analyses section). Finally, there are likely additional unmeasured moderators, given the known heterogeneity of clinical profiles, genetics, and neurobiology in all diagnostic categories, including OCD.

Sensitivity analyses

Publication bias. Effect sizes for Duval and Tweedie's (2000) trim-and-fill analyses are reported in Table 2. Overall, there was little evidence of publication bias. Across analyses, effects were very robust to the trim-and-fill procedure: On average, the weighted mean effect size (d) was only 0.05 lower than for untrimmed analyses, and no significant measures became nonsignificant.

IQ matching. Effect sizes for IQ-matched samples only are reported in Table 2. Across analyses, effects were very robust to IQ matching: On average, for studies that matched groups on IQ, weighted mean effect size (d) was 0.04 higher than those for all samples combined. Nearly all analyses that were significant for all samples remained significant when restricted to IQ-matched samples. Two significant effects, for the verbal-WM overall and verbal-WM-manipulation composite scores, had slightly reduced effect sizes and became nonsignificant as a result of low power, given that there were few IQ-matched samples (verbal-WM overall: k = 11; verbal-WM manipulation: k = 8). In addition, the effect size for Stroop accuracy was higher for IQ-matched samples, but the effect became nonsignificant because of low power (k = 4). IQ-matched samples could not be analyzed for cued-task switching, n-back, and DMTS because there were fewer than three IQ-matched samples.

Moderator analyses

Effect sizes were largely stable across variation in demographic and clinical characteristics of the samples, although age and gender did moderate some effects. Specifically, there was some evidence for increasing effect sizes with increasing age and increasing percentage of female participants, whereas symptom severity did not moderate effect sizes. It is important that moderator analyses indicated that deficits in EF associated with OCD are not driven by co-occurring depression or medication use.

Depression. Comparisons of samples with and without co-occurring diagnosis of a depressive disorder or elevated depressive symptoms are reported in Table 3. Table 3

Table 3. Depression Moderator Analyses of Variance

Dependent variable	Depression[a]	*d*	95% CL LL	95% CL UL	*SE*	*z*	*p*	*k*	*v*	*Q* Between (*df*)	*p*	*Q* Within (*df*)	*p*
Inhibition composite score	Absent	0.47*	0.18	0.76	0.15	3.14	.002	10	0.14	0.02 (1)	.894	31.41 (30)	.396
	Possible	0.49*	0.29	0.69	0.10	4.86	< .001	22					
Stroop interference	Absent	0.72*	0.34	1.09	0.19	3.71	< .001	5	0.11	3.88 (1)	.048*	11.84 (11)	.376
	Possible	0.23	–0.06	0.53	0.15	1.57	.117	8					
Stroop incongruent time	Absent	1.24*	0.46	2.02	0.40	3.12	.002	3	0.36	1.56 (1)	.211	7.36 (5)	.195
	Possible	0.59	–0.06	1.24	0.33	1.79	.073	4					
Stop signal (reaction time)	Absent	0.39	–0.17	0.95	0.28	1.38	.169	3	0.13	1.28 (1)	.257	3.80 (4)	.434
	Possible	0.84*	0.30	1.39	0.28	3.03	.003	3					
Go/no-go (accuracy)	Absent	0.43	–0.21	1.06	0.32	1.32	.186	3	0.23	0.16 (1)	.691	4.75 (5)	.447
	Possible	0.26	–0.32	0.83	0.26	0.87	.384	3					
Shifting composite score	Absent	0.55*	0.34	0.76	0.11	5.14	< .001	10	0.02	0.48 (1)	.487	52.03 (52)	.473
	Possible	0.47*	0.38	0.56	0.05	10.19	< .001	44					
OAT/DAT	Absent	0.47*	0.13	0.82	0.18	2.70	.007	3	0.00	1.88 (1)	.170	8.03 (9)	.531
	Possible	0.21*	0.05	0.37	0.08	2.63	.009	8					
TMT-B	Absent	0.58*	0.25	0.91	0.17	3.44	.001	4	0.05	0.02 (1)	.884	25.64 (25)	.427
	Possible	0.55*	0.41	0.69	0.08	7.74	< .001	23					
WCST	Absent	0.29*	0.09	0.49	0.10	2.86	.004	9	0.01	0.73 (1)	.392	25.70 (26)	.480
	Possible	0.39*	0.27	0.51	0.06	6.61	< .001	19					
Verbal-WM composite score	Absent	0.31#	–0.01	0.62	0.16	1.90	.058	5	0.05	0.30 (1)	.584	17.05 (15)	.316
	Possible	0.20	0.01	0.40	0.10	2.05	.040	12					
Verbal-WM-manipulation composite score	Absent	0.24	–0.22	0.70	0.23	1.01	.310	3	0.10	0.58 (1)	.448	6.74 (7)	.456
	Possible	0.46*	0.12	0.79	0.17	2.69	.007	6					
Verbal-WM-maintenance composite score	Absent	0.20	–0.16	0.56	0.18	1.10	.273	5	0.11	0.05 (1)	0.822	15.70 (12)	.206
	Possible	0.25#	–0.02	0.53	0.14	1.79	.074	9					
Digit-span forward	Absent	0.20	–0.16	0.56	0.18	1.09	.274	5	0.11	0.05 (1)	.825	15.71 (12)	.205
	Possible	0.25#	–0.03	0.53	0.14	1.78	.076	9					
Visuospatial-WM composite score	Absent	0.41*	0.06	0.76	0.18	2.27	.023	6	0.08	0.10 (1)	.747	20.76 (18)	.292
	Possible	0.48*	0.27	0.68	0.11	4.51	< .001	14					
Block span	Absent	0.57*	0.16	0.99	0.21	2.72	.001	3	0.05	0.47 (1)	.493	10.04 (9)	.347
	Possible	0.41*	0.19	0.63	0.11	3.62	< .001	8					

(continued)

Table 3. (Continued)

Dependent variable	Depression[a]	*d*	95% CL		*SE*	*z*	*p*	*k*	*v*	*Q* Between (*df*)	*p*	*Q* Within (*df*)	*p*
			LL	UL									
VF composite score	Absent	0.37*	0.12	0.61	0.13	2.87	.004	6	0.02	0.02 (1)	.875	26.17 (25)	.400
	Possible	0.39*	0.27	0.51	0.06	6.26	< .001	21					
Phonemic VF	Absent	0.37*	0.13	0.61	0.12	2.99	.003	6	.01	0.03 (1)	.870	25.83 (25)	.417
	Possible	0.39*	0.27	0.51	0.06	6.58	< .001	21					
Semantic VF	Absent	0.28	–0.23	0.79	0.26	1.09	.278	3	0.12	0.46 (1)	.498	9.05 (9)	.432
	Possible	0.49*	0.18	0.79	0.16	3.11	.002	8					
Planning composite score	Absent	0.40*	0.09	0.72	0.16	2.56	.010	6	0.07	0.02 (1)	.898	18.72 (20)	.540
	Possible	0.42*	0.24	0.61	0.09	4.55	< .001	16					
TOL/TOH accuracy	Absent	0.41*	0.07	0.75	0.18	2.34	.019	6	0.11	0.03 (1)	.874	14.30 (18)	.709
	Possible	0.38*	0.16	0.59	0.11	3.40	< .001	14					
TMT-A	Absent	0.45*	0.21	0.69	0.12	3.69	< .001	4	0.00	2.75 (1)	.097#	21.19 (21)	.448
	Possible	0.68*	0.56	0.80	0.06	11.36	< .001	19					

Note: *d* = weighted mean Cohen's *d* effect size; CI = confidence interval; LL = lower limit; UL = upper limit; *k* = number of studies; *v* = random-effects variance component; *Q* Between = between-group (moderator) heterogeneity; *Q* Within = within-group (residual) heterogeneity; OAT/DAT = Object Alternation Test/Delayed Object Alternation Test; TMT-B = Trail Making Test Part B; WCST = Wisconsin Card Sorting Test; WM = working memory; VF = verbal fluency; TOL/TOH = Tower of London/Tower of Hanoi; TMT-A = Trail Making Test Part A.

[a]Depression possible indicates average depressive symptom questionnaire scores in clinical range or individuals with diagnosis of any depressive disorder. Depression absent indicates average depressive symptom questionnaire scores below clinical range and no patients with a diagnosed depressive disorder.

*$p < .05$. #$p < .10$ (marginal).

shows effect sizes for samples without co-occurring depression (absent) and samples with possible co-occurring depression (possible), along with the confidence interval and significance test for each group, and the test for the significance of the difference in effect sizes between the groups (*Q* Between). Across analyses, effects were very robust to depression. Depression was a significant moderator for only one task measure, Stroop interference, with larger effect sizes for samples without co-occurring depression diagnosis or elevated depressive symptoms. However, this finding should be interpreted with caution, given that there were few Stroop interference studies in participants without depression ($k = 5$). In addition, for the TMT-A comparison measure, effects were marginally larger for samples with possible co-occurring depression.

On average, across the 20 EF analyses for which there were enough studies to conduct a meta-analysis of variance, effect sizes were very similar for those with and without co-occurring diagnosis of a depressive disorder or elevated depressive symptoms ($\Delta d = 0.04$). Nearly all EF analyses that were significant with all samples included remained significant when restricted to samples without co-occurring depression diagnosis or elevated depressive symptoms, with the exception of stop signal RT, verbal-WM overall and verbal-WM-manipulation composite scores, and semantic verbal fluency, all of which had low power due to having few samples without co-occurring depression ($k = 3$–5).

Continuous moderators. Metaregression analyses for continuous moderators are reported in Table 4. For each measure with sufficient studies to conduct metaregression analyses, Table 4 provides the regression coefficients for each moderator, with the associated 95% confidence intervals and significance test. Age significantly moderated visuospatial-WM composite scores and marginally moderated shifting composite scores, verbal-fluency composite scores, phonemic verbal fluency, and planning composite scores such that effect sizes were larger for older samples. These effects remained significant or marginal when controlling for gender—visuospatial-WM composite: $z = 2.15$, $p = .032$; shifting composite: $z = 2.53$, $p = .011$; verbal-fluency composite: $z = 2.62$, $p = .009$; phonemic verbal fluency: $z = 1.97$, $p = .049$; planning composite: $z = 1.91$, $p = .056$. When we controlled for medication use, the effect of age remained significant or marginal for visuospatial-WM composite scores ($z = 1.82$, $p = .069$), verbal-fluency composite scores ($z = 2.00$, $p = .046$), and planning composite scores ($z = 1.71$, $p = .087$), whereas the effect of age became nonsignificant on shifting composite scores ($z = 1.31$, $p = .189$) and phonemic verbal fluency ($z = 1.15$, $p = .250$). When we controlled for symptom severity, the effect of age remained significant or marginal on visuospatial-WM composite scores ($z = 2.09$, $p = .037$), shifting composite scores ($z = 1.70$, $p = .089$), and planning composite scores ($z = 1.97$, $p = .049$), whereas the effects on verbal-fluency composite scores ($z = 1.09$, $p = .275$) and phonemic verbal fluency ($z = 1.40$, $p = .162$) became nonsignificant.

There was a significant effect of gender on shifting composite scores and marginal effects of gender on TMT-B and verbal-WM composite scores such that samples with more female participants had worse performance. When we controlled for age, the effect of gender remained significant on shifting composite scores ($z = 2.48$, $p = .013$) and verbal-WM composite scores ($z = 2.02$, $p = .044$) but became nonsignificant on TMT-B ($z = 1.63$, $p = .104$). When we controlled for medication, the effect of gender remained significant for shifting composite scores ($z = 2.53$, $p = .011$) but became nonsignificant on TMT-B ($z = 1.54$, $p = .123$) and verbal-WM composite scores ($z = 1.26$, $p = .207$). When we controlled for symptom severity, the effect of gender remained significant for shifting composite scores ($z = 2.25$, $p = .025$) but became nonsignificant for TMT-B ($z = 1.21$, $p = .228$) and verbal-WM composite scores ($z = 1.30$, $p = .192$).

The percentage of individuals with OCD taking psychotropic medication marginally moderated inhibition composite scores such that samples with a higher percentage of medicated participants exhibited worse performance. This effect remained marginal after we controlled for age ($z = 1.76$, $p = .078$) and gender ($z = 1.68$, $p = .092$) but not symptom severity ($z = 1.30$, $p = .194$). OCD symptom severity, as assessed by the Y-BOCs, did not significantly moderate any analyses.

Discussion

Evaluating hypotheses: EF is broadly impaired in OCD

In sum, the current meta-analysis shows that in comparison with their healthy peers, individuals with OCD exhibited significantly impaired performance on tasks measuring most aspects of EF, with most effect sizes (*d*) in the 0.3 to 0.5 range.[4] These effects were not due to failure to match groups on IQ or to publication bias. The results are consistent with Hypothesis 1, which posits a broad impairment across multiple aspects of EF that may be driven by dysfunction in prefrontal-striatal circuits (e.g., Kuelz et al., 2004; Menzies, Chamberlain, et al., 2008). The exception was verbal-WM maintenance, in which task performance for individuals with OCD was comparable with control participants. However, this finding is not incompatible with Hypothesis 1, given that simple maintenance of information in WM (as opposed to manipulation) is not strongly linked to other aspects of EF (e.g., Engle et al., 1999). Hypothesis 2, which

Table 4. Moderator Regression Analyses

Dependent and independent variable	β	*b*	95% CI LL	95% CI UL	*SE*	*z*	*p*	*k*	*v*	*Q* Model (*df*)	*p*	*Q* Within (*df*)	*p*
Inhibition comp.													
Severity (Y-BOCS)	0.132	0.000	–0.001	0.001	0.001	0.83	.407	39	0.157	0.69 (1)	.407	38.95 (37)	.382
Age	0.099	0.007	–0.012	0.025	0.010	0.70	.486	49	0.153	0.49 (1)	.486	48.93 (47)	.340
Gender	–0.109	–0.003	–0.002	0.004	0.003	–0.75	.451	48	0.142	0.57 (1)	.451	47.77 (46)	.401
Medication	0.288	0.004	–0.000	0.008	0.002	1.90	.058#	40	0.130	3.59 (1)	.058	39.72 (38)	.393
Shifting comp.													
Severity (Y-BOCS)	0.179	0.015	–0.006	0.036	0.011	1.40	.162	61	0.06	1.96 (1)	.162	59.46 (59)	.459
Age	0.201	0.011	–0.001	0.023	0.006	1.76	.078#	74	0.04	3.11 (1)	.078	73.49 (72)	.429
Gender	0.297	0.001	0.000	0.002	0.000	2.63	.009*	73	0.04	6.93 (1)	.009	71.84 (71)	.450
Medication	0.022	0.000	–0.002	.002	0.001	0.16	.869	58	0.06	0.03 (1)	.869	56.74 (56)	.447
TMT-B													
Severity (Y-BOCS)	0.099	0.008	–0.020	0.036	0.014	0.56	.573	33	0.08	0.32 (1)	.573	32.07 (31)	.414
Age	0.226	0.016	–0.006	0.037	0.011	1.40	.160	37	0.07	1.97 (1)	.160	36.76 (35)	.387
Gender	0.273	0.005	–0.001	0.011	0.003	1.66	.097#	35	0.05	2.76 (1)	.097	34.16 (33)	.412
Medication	0.177	0.002	–0.002	0.006	0.002	0.95	.342	30	0.08	0.90 (1)	.342	37.94 (28)	.473
WCST													
Severity (Y-BOCS)	0.106	0.009	–0.019	0.036	0.014	0.60	.547	34	0.07	0.36 (1)	.547	31.83 (32)	.475
Age	0.067	0.004	–0.014	0.022	0.009	0.42	.673	42	0.06	0.18 (1)	.673	40.04 (40)	.467
Gender	0.026	0.001	–0.006	0.007	0.003	0.17	.868	42	0.06	0.03 (1)	.868	40.19 (40)	.462
Medication	–0.002	–0.000	–0.003	0.003	0.002	–0.01	.990	33	0.08	0.00 (1)	.990	31.96 (32)	.469
Verbal-WM comp.													
Severity (Y-BOCS)	0.227	0.014	–0.013	0.041	0.014	1.01	.311	19	0.05	1.03 (1)	.311	18.87 (17)	.336
Age	0.180	0.010	–0.011	0.031	0.011	0.92	.358	25	0.06	0.84 (1)	.358	25.24 (23)	.338
Gender	0.338	0.010	–0.001	0.020	0.005	1.80	.072#	25	0.05	3.23 (1)	.072	25.13 (23)	.344
Medication	0.311	0.003	–0.002	0.008	0.002	1.30	.194	17	0.07	1.69 (1)	.194	15.80 (15)	.396
Visuospatial-WM comp.													
Severity (Y-BOCS)	0.315	0.025	–0.007	0.056	0.016	1.54	.122	22	0.06	2.39 (1)	.122	21.60 (20)	.263
Age	0.424	0.026	0.004	0.047	0.011	2.30	.021*	24	0.04	5.31 (1)	.021	24.25 (22)	.334
Gender	0.088	0.003	–0.009	0.014	0.006	0.43	.665	23	0.07	0.19 (1)	.665	23.90 (21)	.298
Medication	0.334	0.003	–0.001	0.008	0.002	1.57	.118	21	0.06	2.45 (1)	.118	19.58 (19)	.420

(continued)

Table 4. (Continued)

Dependent and independent variable	β	*b*	95% CI LL	95% CI UL	*SE*	*z*	*p*	*k*	*v*	*Q* Model (*df*)	*p*	*Q* Within (*df*)	*p*
VF comp.													
Severity (Y-BOCS)	0.083	0.006	–0.019	0.031	0.013	0.46	.646	31	0.05	0.21 (1)	.646	30.72 (29)	.379
Age	0.287	0.014	–0.001	0.030	0.008	1.87	.061#	40	0.04	3.51 (1)	.061	39.23 (38)	.414
Gender	0.123	0.002	–0.004	0.008	0.003	0.76	.448	39	0.03	0.58 (1)	.448	37.54 (37)	.444
Medication	0.145	0.001	–0.002	0.004	0.001	0.78	.434	31	0.03	0.61 (1)	.434	28.69 (29)	.481
Phonemic VF													
Severity (Y-BOCS)	0.321	0.014	–0.004	0.033	0.010	1.49	.136	28	0.00	2.23 (1)	.136	19.29 (26)	.824
Age	0.334	0.013	–0.001	0.026	0.007	1.84	.065#	35	0.00	3.39 (1)	.065	27.00 (33)	.760
Gender	–0.025	–0.000	–0.006	0.005	0.003	–0.14	.892	35	0.00	0.02 (1)	.892	30.37 (33)	.599
Medication	–0.036	–0.000	–0.002	0.002	0.001	–0.15	.878	28	0.00	0.02 (1)	.877	18.82 (26)	.844
Planning comp.													
Severity (Y-BOCS)	–0.134	–0.010	–0.039	0.020	0.015	–0.64	.523	25	0.07	0.41 (1)	.523	22.40 (23)	.497
Age	0.358	0.017	–0.002	0.034	0.009	1.93	.053#	28	0.05	3.73 (1)	.053	25.35 (26)	.499
Gender	0.174	0.004	–0.005	0.012	0.004	0.87	.383	27	0.06	0.76 (1)	.383	24.55 (25)	.488
Medication	0.106	0.001	–0.003	0.005	0.002	0.48	.640	24	0.08	0.23 (1)	.630	20.37 (22)	.560
Planning accuracy													
Severity (Y-BOCS)	–0.336	–0.026	–0.059	0.007	0.017	–1.52	.129	23	0.10	2.30 (1)	.129	18.12 (21)	.641
Age	0.160	0.009	–0.015	0.032	0.009	0.72	.468	25	0.11	0.53 (1)	.469	19.89 (23)	.648
Gender	0.137	0.003	–0.006	0.011	0.004	0.61	.545	25	0.12	0.37 (1)	.545	19.10 (23)	.488
Medication	–0.031	–0.000	–0.005	0.005	0.003	–0.13	.899	22	0.12	0.02 (1)	.899	16.79 (20)	.666
TMT-A													
Severity (Y-BOCS)	0.117	0.008	–0.017	0.032	0.013	0.60	.549	27	0.04	0.36 (1)	.549	25.98 (25)	.409
Age	0.138	0.008	–0.012	0.029	0.011	0.78	.436	32	0.04	0.61 (1)	.436	31.02 (30)	.414
Gender	0.133	0.002	–0.004	0.004	0.003	0.71	.475	30	0.03	0.51 (1)	.475	28.44 (28)	.441
Medication	–0.156	–0.000	–0.001	0.000	0.000	–0.80	.426	26	0.04	0.63 (1)	.426	24.55 (24)	.430

Note: *d* = weighted mean Cohen's *d* effect size; CI = confidence interval; LL = lower limit; UL = upper limit; *k* = number of studies; *v* = random-effects variance component; *Q* Model = moderator heterogeneity; *Q* Within = within-group (residual) heterogeneity; Comp. = composite score; Y-BOCS = Yale-Brown Obsessive Compulsive Scale; TMT-B = Trail Making Test Part B; WCST = Wisconsin Card Sorting Test; WM = working memory; VF = verbal fluency; TMT-A = Trail Making Test Part A.

*$p < .05$. #$p < .10$ (marginal).

posits a specific impairment in shifting or inhibition, was not supported because effect sizes in other EF domains were equivalent to those for shifting and inhibition. The results are thus consistent with the theory that individuals with OCD have impairments in the unitary component of EF (i.e., common EF), which is posited to be the ability to actively maintain task goals and use this information to effectively bias lower-level processes (Friedman et al., 2008; Miyake & Friedman, 2012). Although other explanations are also possible (e.g., multiple specific aspects of EF could be independently impaired in OCD), impairment in common EF is the most parsimonious interpretation.

It is also possible that individuals with OCD have deficits in common EF as well as processing-specific impairments in shifting or updating (recall that there is no inhibition-specific component; e.g., Friedman et al., 2008). Indeed, the largest effect size in the meta-analysis was for updating WM (n-back), which is believed to depend critically on striatal gating of information into PFC (e.g., Chatham et al., 2011; Frank, Loughry, & O'Reilly, 2001; Hazy, Frank, & O'Reilly, 2007). This suggests that individuals with OCD might have specific updating impairments in addition to common EF impairments, given that both striatal and prefrontal dysfunction may contribute to deficits on updating tasks. Future research using a latent-variable approach is needed to address these possibilities, as discussed in the Future Directions section.

Hypothesis 3, which posits that apparent EF deficits are due to general motor-response slowing, was not supported. The current analysis revealed that individuals with OCD do exhibit significant general motor slowing and are especially slowed on the TMT-A, which requires both motor speed and sequencing. However, significant impairments were also detected on accuracy measures from self-paced EF tasks, with effect sizes as large as or larger than many of the response time tasks. Thus, although individuals with OCD do have slowed responses even on simple general motor-speed tasks, these deficits cannot fully account for deficits on EF tasks. (However, as discussed in the introduction, it is impossible to rule out a deficit in general processing speed—as opposed to general motor speed—that could potentially reduce accuracy.)

Finally, co-occurring depression does not account for EF deficits in OCD as posited by Hypothesis 4. OCD samples with no depression diagnoses and low levels of depressive symptoms were significantly and equivalently impaired on almost all measures of EF. This raises the question of how EF deficits associated with OCD and MDD (e.g., Snyder, 2013) are related to one another. One possibility is that prefrontal abnormalities that lead to impairments in EF may be transdiagnostic risk factors for psychopathology, including OCD and depressive disorders including MDD (e.g., Nolen-Hoeksema & Watkins, 2011; see the Relating Deficits Across Cognitive Domains and Disorders section). It is also possible that OCD and MDD have independent effects on EF that might be detected with more sensitive continuous analyses of depressive symptoms, which were not possible here because of the wide variety of depression measures reported in the primary literature.

Effect sizes were largely stable across variation in demographic characteristics of the samples, although there was some evidence for larger deficits for older OCD groups (for shifting, visuospatial WM, verbal fluency, and planning). This finding warrants further research, given that empirical studies have not investigated age effects. In addition, medication use was associated with larger impairments on inhibition composite scores, and a higher percentage of female participants was associated with larger impairments in shifting and verbal WM. Although these effects were not found for any other measures, they may warrant further empirical study both because some medications may have cognitive side effects and because one previous study showed larger EF impairments for women with OCD on some measures (Mataix-Cols et al., 2006). The fact that symptom severity did not moderate effect sizes suggests that EF impairment may be a stable trait associated with OCD rather than fluctuating with current symptoms. However, this finding must be interpreted with caution, given the relatively narrow range of severity levels in the included studies.

Limitations

There are several limitations in the conclusions that can be drawn from the current meta-analysis as a result of limitations in the primary literature. First, co-occurring depression was coded as a categorical variable. This was necessary because the primary literature reports a wide variety of depression measures, which cannot easily be converted into a single continuous measure. The categorical depression measure (no co-occurring depression vs. any amount of co-occurring depression) provides a conservative test that demonstrates that EF deficits are present in nondepressed individuals with OCD. However, this categorical measure limits the ability to detect the extent to which co-occurring depression might contribute to larger EF deficits in individuals with OCD. Future research could address this issue in several ways: Researchers in individual studies could examine correlations between depression and EF performance in samples with OCD, and increased reporting of a common set of depression measures across studies, or psychometric studies to allow conversion of different measures to a common scale, would allow for the use of continuous depression measures in future meta-analyses.

Second, the current meta-analysis is limited in its ability to determine to what extent EF deficits are related to specific OCD symptoms versus anxiety more broadly. Addressing this issue requires increased reporting and analyzing of more detailed information about co-occurring anxiety disorders and anxiety symptoms, as well as more specific sets of OCD symptoms (e.g., compulsions and obsessions separately). Moving toward this more dimensional approach holds promise for uncovering mechanisms of psychopathology that may be obscured by heterogeneous diagnostic categories (e.g., Insel et al., 2010). Finally, as discussed in the Future Directions section, the current meta-analysis is limited by the types of EF tasks included in the primary literature. Specifically, many of these tasks are too broad to answer fine-grained questions about specific aspects of EF.

Implications for the frontal-striatal model

Consistent with the EF deficits reviewed here, structural and functional abnormalities in PFC have been shown in individuals with OCD (for reviews, see Menzies, Chamberlain, et al., 2008; Nitschke & Heller, 2005). Earlier versions of the frontal-striatal model posited a specific deficit in orbitofrontal function (e.g., Graybiel & Rauch, 2000). However, both the results of the current meta-analysis (which shows deficits in EF tasks known to depend primarily on other areas of PFC; e.g., Nee et al., 2007; Wager et al., 2004) and the more recent versions of the frontal-striatal model based on neuroimaging evidence (Menzies, Chamberlain, et al., 2008) suggest that function is disrupted in a wider PFC network not limited to orbitofrontal cortex.

Functional neuroimaging during EF tasks has revealed activation differences between individuals with OCD and healthy control individuals across a wide PFC network, including anterior cingulate (e.g., Fitzgerald et al., 2005; Maltby, Tolin, Worhunsky, O'Keefe, & Kiehl, 2005; Yucel et al., 2007) and dorsolateral and ventrolateral PFC (Maltby et al., 2005; Roth et al., 2007; van den Heuvel, Veltman, Groenewegen, Cath, et al., 2005), in addition to orbitofrontal cortex (Maltby et al., 2005; Roth et al., 2007). However, both hyperactivation and hypoactivation have been found across studies. Thus, although there is strong evidence for differences in PFC function in individuals with OCD compared with control participants, the direction of these differences is unclear and may depend on task or individual characteristics yet to be differentiated.

Future directions

Given the compelling evidence that individuals with OCD are impaired on most EF tasks, we argue that there is no longer a need for further case-control studies of performance on standard neuropsychological measures of EF. Rather, the opportunity exists to build on the foundation of such previous studies to better understand the specific mechanisms and causal processes contributing to EF deficits in OCD and to move toward translational applications. To do so, we advocate for (a) improving assessment of EF by using multiple, specific measures of different EF components based on well-established EF models; (b) probing deficits at multiple levels of analysis; (c) investigating how EF deficits are related across disorders and how EF deficits are related to deficits in other cognitive domains; and (d) using longitudinal, mediational, and behavior-genetic approaches to probe possible causal links between EF deficits and OCD.

More precise assessment of EF deficits. Future research would benefit from the use of more sensitive and specific measures of each aspect of EF and by the use of multiple measures. We argue that investigating how specific aspects of OCD are related to specific EF components is critical for elucidating the cognitive, neural, and genetic mechanisms involved. Many previous studies, including many in the current meta-analysis, have used EF measures that are too broad to answer these fine-grained questions. For example, verbal-fluency tasks have been a perennial favorite for assessing EF. However, they and other complex neuropsychological tests tap a wide variety of cognitive processes, including not only aspects of EF but also nonexecutive abilities (Miyake et al., 2000). As a result, impairments on such measures are difficult to interpret. This concern can be addressed by using tasks designed to specifically place demands on one aspect of EF while keeping other demands minimal (e.g., Aron, 2008; Miyake et al., 2000).

In addition, the inclusion of multiple measures of each aspect of EF would allow for construction of latent or composite measures, which greatly increase construct validity and power (e.g., Miyake et al., 2000). Although the current meta-analysis shows broad impairment in EF, which is most parsimoniously explained by a deficit in common EF, latent-variable approaches are also needed to answer the key question whether impairments across multiple aspects of EF are due to impairment of the common EF component, to multiple separate impairments, or to both. For example, such a design could address the question whether deficits on EF tasks are fully accounted for by deficits in common EF (i.e., once common EF is accounted for, there is no longer any evidence of process-specific impairments that are applicable only to individual aspects of EF) or whether there are processing-specific deficits not fully accounted for by common EF impairments (e.g., an updating-specific impairment related to striatal dysfunction). Research designs needed to test these models pose logistical challenges for research

in clinical populations, given that they require longer testing sessions to collect multiple measures and large (more than 200 participants) sample sizes. However, overcoming these challenges—for example, by administering tasks in several shorter sessions to reduce fatigue and collaborating across sites to increase sample size—may pay large dividends for determining the specific cognitive and neural mechanisms affected in individuals with OCD.

Understanding deficits at multiple levels of analysis. Although the current meta-analysis demonstrates impairments in EF at the level of behavioral-task performance, future research is needed to investigate the specific neural mechanisms contributing to EF deficits in OCD. For example, human genetic studies and nonhuman animal models have suggested a role for abnormalities in the dopamine, serotonin, and glutamate systems in OCD (e.g., Albelda & Joel, 2012; Pauls, 2008; Rolls, Loh, & Deco, 2008). A promising area of research is the integration of what is known about the role of these neurotransmitter systems in PFC networks with behavioral and neuroimaging evidence for EF impairments in individuals with OCD, thereby building a more detailed model of the neurobiology of OCD that spans multiple levels of analysis.

Relating deficits across cognitive domains and disorders. Although the current meta-analysis focused on EF, cognitive deficits associated with OCD are not restricted to EF, with meta-analytic evidence for deficits of a similar magnitude in processing speed, episodic memory, and attention (Abramovitch et al., 2013; N. Y. Shin et al., 2014; Woods, Vevea, Chambless, & Bayen, 2002). An important question thus concerns how these deficits are related to one another. Some may be independent, for example, general motor slowing may be related to dysfunction in premotor-striatal loops, which are adjacent to, but separate from, the PFC-striatal loops involved in EF (e.g., Haber & Calzavara, 2009). In other cases, a common deficit may lead to impairments across domains. For example, some researchers have argued that poor performance on memory tasks by individuals with OCD is largely attributable to EF deficits, which affect the ability to generate and implement organizational strategies (e.g., grouping words semantically) during encoding and retrieval (e.g., Olley et al., 2007). Investigation of these possibilities requires that future empirical research test relations among performance in different domains (e.g., whether memory impairments are fully mediated by EF impairments).

In addition, OCD is far from the only psychiatric disorder associated with EF deficits, with similar effect sizes to those in OCD for MDD (d = 0.3–0.7; e.g., Rock, Roiser, Riedel, & Blackwell, 2013; Snyder, 2013) and attention-deficit/ hyperactivity disorder (d = 0.3–0.7; e.g., Frazier, Demaree, & Youngstrom, 2004; Walshaw, Alloy, & Sabb, 2010) and somewhat larger deficits in schizophrenia (d = 0.7–1.3; e.g., Forbes, Carrick, McIntosh, & Lawrie, 2009; Mesholam-Gately, Giuliano, Goff, Faraone, & Seidman, 2009) and bipolar disorders (d = 0.4–0.8; e.g., Kurtz & Gerraty, 2009; Mann-Wrobel, Carreno, & Dickinson, 2011). Thus, a second important question is the extent to which EF deficits are shared, or differ, across disorders.

It is important that although effect sizes differ somewhat across disorders, the same broad pattern of impairment across multiple aspects of EF is found in each, which suggests that PFC abnormalities that lead to impairments in EF may be transdiagnostic risk factors for psychopathology (e.g., Buckholtz & Meyer-Lindenberg, 2012; Nolen-Hoeksema & Watkins, 2011). This general vulnerability may combine with unique genetic, neurobiological, and environmental factors to produce divergent trajectories, thereby leading to different disorders. However, if more detailed measures at multiple levels of analysis are considered, in some cases, these shared behavioral deficits may arise from distinct neural mechanisms (e.g., perturbations in different neurotransmitter systems; e.g., Gigante et al., 2012; Luykx et al., 2012). Future studies in which researchers systematically investigate relations between EF impairments, risk factors, and psychopathology are needed to refine understanding of how such broad EF deficits arise across disorders and which aspects of these deficits are transdiagnostic versus disorder specific.

Possible causal links between OCD and EF. Previous research has left the question of how OCD and EF impairments are causally related largely unaddressed. For example, the vast majority of studies, including in the current meta-analysis, have used cross-sectional case-control designs that do not provide any information about temporal precedence that could contribute to understanding causal links. There are (at least) three non–mutually exclusive possibilities for these causal relationships. First, individual differences in neurobiology could both confer risk for OCD and lead to EF deficits. Specifically, alterations in PFC function may cause impaired EF as well as contribute to OCD symptoms, such as perseverative behaviors and the inability to inhibit compulsions (e.g., Menzies, Chamberlain, et al., 2008). In support of this model, differences in PFC and impairments in EF are present in nonaffected relatives of individuals with OCD (e.g., Cavedini, Zorzi, Piccinni, Cavallini, & Bellodi, 2010; Menzies, Williams, et al., 2008; Rajender et al., 2011), which suggests that they may represent endophenotypes for OCD. This possibility can be further tested with future behavior-genetic research

testing for shared genetic influence on EF and OCD, as well as by longitudinal studies tracking children at risk for OCD (e.g., due to family history) before onset.

The hypothesis that PFC and basal ganglia dysfunction may play a causal role in OCD is also supported by evidence that acquired brain damage to these areas can cause obsessions and compulsions along with EF impairments (e.g., see Coetzer, 2004, for review). Experimentally induced lesions to PFC and basal ganglia structures in animal models thus present an obvious target for testing causal models of OCD. However, such animal models have been difficult to develop and validate, potentially as a result of the large differences in prefrontal structure and function between rodents and humans (e.g., see Albelda & Joel, 2012, for review). As an alternative approach, studies in which researchers carefully evaluate the location of lesions associated with the onset of OCD symptoms (e.g., through voxel-based lesion-symptom mapping), and associated EF deficits, may help to shed light on possible causal associations between dysfunction in particular brain areas and OCD.

Second, OCD could directly cause EF deficits. For example, OCD-related intrusive thoughts may consume cognitive resources and interfere with the ability to maintain task goals. This possibility could be tested experimentally (e.g., by triggering intrusive thoughts prior to tasks). However, this direct effect is unlikely to be the only causal path, given that there is some evidence that EF deficits remain stable after OCD symptoms have remitted (Bannon, Gonsalvez, Croft, & Boyce, 2006; Roh et al., 2005). Further research with individuals in remission is needed to confirm this possibility. If true, this finding suggests that even when treatment is considered successful from the perspective of eliminating affective symptoms and behaviors, persistent EF deficits may continue to undermine daily functioning and quality of life. It may therefore be useful to make deficits in EF as a focus for intervention a topic for future translational research.

Third, poor EF could contribute to the development or maintenance of OCD. Current models of anxiety suggest individuals with poor emotion-regulation abilities engage in frequent and excessive worrying (Borkovec & Roemer, 1995; Mennin, Heimberg, & Turk, 2002). Meanwhile, better EF is linked to more effective emotion-regulation strategies (e.g., Ochsner & Gross, 2005). Thus, it may be that poor EF leads individuals to develop alternative, but less adaptive or efficient, strategies for coping with emotional challenges that ultimately increase levels of OCD symptoms. For example, the anxiety-reduction hypothesis posits that compulsions are a maladaptive strategy for reducing anxiety associated with intrusive thoughts and, thus, are reinforced through avoidance learning (e.g., see Clark, 2004, for review). Thus, reduced ability to use EF to engage more adaptive anxiety-regulation strategies (e.g., shifting attention away from the anxiety-provoking thought) could contribute to the development or maintenance of compulsive behaviors. Because this path is speculative, longitudinal research is necessary to explore EF, emotion regulation, and the onset of OCD or escalation of OCD symptoms.

Understanding causal links between EF deficits and OCD will be critical for developing strategies for prevention and remediation. For example, if EF deficits precede and contribute to the development of OCD, those individuals at risk (e.g., as a result of family history) may benefit from early intervention to train EF or teach compensatory strategies. In addition, regardless of the initial direction of the causal arrow, poor EF may reduce the effectiveness of therapeutic interventions. For example, deficits in EF could interfere with cognitive-behavioral therapy (e.g., Mohlman & Gorman, 2005), particularly with techniques such as exposure and response prevention, which require top-down processes such as shifting (e.g., from elaboration of automatic, catastrophic thoughts to metacognitive awareness of thoughts), inhibitory control (e.g., resisting engaging in compulsive behaviors), and updating (e.g., replacing maladaptive beliefs and behaviors with adaptive ones). EF deficits may therefore present a major barrier to successful completion of treatment.

Conclusions

In sum, OCD is associated with broad EF impairment, and these deficits cannot be accounted for by co-occurring depression or general motor slowing. These results are consistent with theories that posit that prefrontal dysfunction is a contributing factor in OCD, but additional research is needed to determine the causal links between EF impairments and OCD and to build a more detailed model of the neurobiology of these impairments. A better understanding of when and how EF impairments arise for individuals with OCD may have important implications for treatment, such as pharmacological interventions that target specific aspects of prefrontal function or training programs to improve EF or teach compensatory strategies to mitigate the effects of EF impairments. Given the centrality of EF to our ability to successfully navigate daily life, such research has the potential to improve outcomes for many individuals affected by OCD.

Author Contributions

H. R. Snyder, R. H. Kaiser, S. L. Warren, and W. Heller developed the meta-analysis concept and design. H. R. Snyder performed the literature search, coding, and meta-analyses. All authors contributed to coding reliability checks. H. R. Snyder drafted the initial version of the manuscript, which was revised by all the authors. All authors approved the final version of the manuscript for submission.

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The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Supplemental Material

Additional supporting information may be found at http://cpx.sagepub.com/content/by/supplemental-data

Notes

1. Although updating and WM manipulation are clearly related, and some researchers may consider updating to be a subtype of WM-manipulation processes, updating specifically requires adding and removing information to and from WM, whereas WM manipulation often involves reorganizing (e.g., reordering) information already held in WM. Thus, there are unique aspects of updating not shared with other types of WM manipulation. For example, the basal ganglia are thought to play a key role in gating information into and out of WM during updating (e.g., Chatham et al., 2011; Frank, Loughry, & O'Reilly, 2001), which is of strong interest in regard to OCD, given evidence of basal ganglia dysfunction.
2. In addition, the following tasks were nominated for inclusion by one author each but were excluded because there was not agreement that the tasks clearly assessed a single, specific aspect of EF: (a) verbal-fluency switching (excluded because it mixes verbal-fluency and shifting demands), (b) Rey Complex Figure immediate recall (excluded because it mixes WM and episodic/incidental memory demands), and (c) delayed-response task (excluded because WM demands appear to be minimal).
3. Six studies had adolescent participants (ages 12–14). Given that adult and nonadult studies could potentially differ, supplementary analyses were conducted with studies of adult participants (ages 18 and older) only. Effect sizes were virtually identical when the adolescent samples were excluded compared with when they were included. Thus, all studies are included in the analyses reported here.
4. Comparing the results of the current meta-analysis with those of the previous meta-analyses that included some EF measures, we found that effect sizes for individual tasks reported by N. Y. Shin, Lee, Kim, and Kwon (2014) were fairly consistent with the current results for analyses in which Shin et al. included 10 or more studies, whereas analyses with only 5 to 6 studies both over- and underestimated effects compared with the current study, which likely reflects imprecision in these estimates due to the small number of studies. Comparison of the current results with those of Abramovitch, Abramowitz, and Mittelman (2013) is not straightforward because Abramovitch et al. did not report analyses of individual measures, and they grouped measures into composites that do not always align with the theoretically motivated grouping of measures used in the current meta-analysis. For the most comparable composite measures across meta-analyses, planning effect sizes were identical (d = 0.44), and inhibition effect sizes were somewhat smaller in the current study (d = 0.37 vs. 0.49). For the less comparable composites, the shifting composite measure effect size in the current study was similar to that of the shifting/fluency/Wechsler Adult Intelligence Scale similarities measure in Abramovitch et al. (d = 0.50 vs. 0.55), the verbal-WM/updating composite (d = 0.34) was between those for verbal WM (d = 0.22) and updating (d = 0.71) in the current study, and the spatial-WM/updating composite (d = 0.37) was lower than both the visuospatial-WM (d = 0.47) and the updating effect sizes in the current study (potentially due to the small number of studies included in the Abramovitch et al. analysis and differences in the included tasks).

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