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# Neuropsychological functioning in youth with obsessive compulsive disorder: An examination of executive function and memory impairment

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ABSTRACT

Preliminary research suggests neuropsychological deficits in youth with obsessive-compulsive disorder (OCD) similar to those in adults; however, small samples and methodological confounds limit interpretation. We aimed to examine the rates and clinical correlates of cognitive sequelae in youth with OCD, focusing on executive functioning and memory abilities. Youth ages 7–17 years with OCD ($N=96$) completed a hypothesis-driven neuropsychological battery (including the Rey–Osterreith Complex Figure, California Verbal Learning Test, and subtests of the Delis–Kaplan Executive Function System and Wide Range Assessment of Memory and Learning) that primarily assessed executive functioning, memory and processing speed. Cognitive sequelae were identified in 65% of youth (37% using a more stringent definition of impairment). Magnitude of cognitive sequelae was not associated with OCD severity or age; however, greater neuropsychological impairments were found amongst youth prescribed atypical neuroleptics and those diagnosed with comorbid tic disorders. Comorbidity burden was associated with presence of neuropsychological impairment, but was not specific to any single test. Findings suggest that the presence of cognitive sequelae is prevalent amongst treatment-seeking youth with OCD. Deficits were found in executive functioning and non-verbal memory performance but these impairments were not associated with OCD severity.



## 1. Introduction

Obsessive-compulsive disorder (OCD) is a chronic and impairing neuropsychiatric disorder characterized by recurrent intrusive thoughts/impulses and contingent ritualized behavior. Converging findings from imaging studies implicate abnormalities in frontal-subcortical circuitry in the pathogenesis of OCD (Baxter et al., 1992; Saxena and Rauch, 2000) with most emphasis focused on anomalies in the basal ganglia, anterior cingulate cortex and orbitofrontal cortex (Bedard et al., 2009). Notably, disruption of the prefrontal-basal ganglia-thalamic-prefrontal loop (PFCT), a system that plays a critical role in conscious gating of cortical inputs, shifting and balancing of attention, and behavioral inhibition, is consistent with the clinical phenomenology of OCD (e.g., the impaired ability to inhibit repetitive behaviors and thought-intrusions) (Saxena and Rauch, 2000).

Given that neurocognitive impairments should mirror underlying brain dysfunction (Beers et al., 1999) there is traction for using neuropsychological testing as an endophenotypic marker of neuropsychiatric illnesses (Gottesman and Gould, 2003), including OCD (Penades et al., 2005; Rao et al., 2008), with data suggesting that neurocognitive impairment in individuals with OCD may differ from schizophrenia, depression, and other anxiety disorders (Purcell et al., 1998b; Moritz et al., 2002; Ornstein et al., 2010). Neuropsychological examinations of individuals with OCD have been largely limited to adult populations, but implicate impairment on tests of higher-order domains of cognition including planning, organizing, shifting, flexibility, response inhibition and coordinating more basic cognitive functions as well as non-verbal memory (Savage et al., 2000; Savage and Rauch, 2000; Kim et al., 2002; Nielen and Den Boer, 2003; Fontenelle et al., 2004; Penades et al., 2005; Roh et al., 2005; Andres et al., 2007; Olley et al., 2007; Rao et al., 2008). Models of neuropathology in OCD that emphasize frontostriatial dysfunction are consistent with these neuropsychological findings, suggesting deficits in these global domains of executive functioning and nonverbal memory (Kuelz et al., 2004).

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Unfortunately, no studies to date document the occurrences of neuropsychological impairments in large samples of youth with OCD (with only one extant study with > 30 youth) (Flessner et al., 2010).

Relative to adults, studies of cognitive dysfunction in youth with OCD are fewer and limited by small sample sizes, but findings generally suggest impairment in executive functions and non-verbal memory (Andres et al., 2007; Shin et al., 2008; Lewin et al., 2011). In 35 youth with OCD (ages 7–18), impairments in visual memory (but not verbal fluency or verbal memory) were noted (Andres et al., 2007). Although there was no relation between neurocognitive performance and OCD severity, age, and pharmacological treatment, depressive symptoms were linked to poorer neuropsychological performance. Visuospatial organization and memory deficits have been documented in other studies of youth with OCD. For example, a recent longitudinal study of 24 youth with OCD suggested that visuospatial organizational deficits predicted persistence of OCD symptoms into young adulthood (Bloch et al., 2011). Another study comparing 17 youth with OCD (without comorbidity) to 91 youth with other psychiatric disorders showed weaknesses in youth with OCD in the domains of set shifting and organization (Shin et al., 2008).

In contrast, a well-controlled study by Beers et al. (1999) did not identify marked cognitive impairment among youth with OCD ($n=21$). Savage and Rauch (2000) interpreted this null finding in the context of cognitive development in youth. Specifically, cognitive deficits likely emerge in parallel to normal prefrontal maturation and thus, youth with OCD may have less pervasive cognitive deficits (relative to adults) as a function of the relative immaturity of their prefrontal networks. Notably, the Beers et al. (1999) study did not account for comorbidity. Shin et al. (2008) further postulated on the null finding in Beers et al. by noting the absence of using the Rey Osterrieth Complex Figure (ROCF) (Rey, 1941), a task of visuospatial planning and memory commonly used on OCD-related neuropsychological evaluations. The role of executive dysfunction in OCD has been illustrated consistently with the ROCF (Savage and Rauch, 2000), which requires integration of executive and multiple additional PFCT-mediated functions (Shin et al., 2008). In fact, executive functioning deficits may drive other cognitive dysfunction in OCD (e.g., memory, fluency, error monitoring, switching) and potentially treatment response (Savage and Rauch, 2000; Shin et al., 2008; Flessner et al., 2010). For example, poor organizational/encoding strategy in youth with OCD may explain deficit memory encoding (Greisberg and McKay, 2003). In addition to executive impairments, lack of trust in one's memory may contribute to poor performance on tests of memory (Markarian et al., 2010).

Several studies document deficits in set-shifting, initiation tasks, planning/cognitive flexibility, inhibition and non-verbal memory in both adult and pediatric individuals with OCD (Head et al., 1989; Kuelz et al., 2004; Penades et al., 2005; Andres et al., 2008; Rao et al., 2008); however, small sample sizes of existing child studies (mean $N=33$ in five extant studies) may limit generalizations that can be made regarding neurocognitive impairment in youth with OCD given: (a) the wide range of normal cognitive development among children/adolescents (Savage and Rauch, 2000) and (b) variability in neuropsychological test batteries used, especially among small samples (Purcell et al., 1998a; Kim et al., 2002).

A number of factors may contribute to inconsistencies across existing studies, such as the severity of comorbidity, medication effects, symptom heterogeneity, OCD severity, and developmental differences (Penades et al., 2005; Andres et al., 2008). In a study comparing two groups of adults with OCD – 28 adults prescribed serotonin reuptake inhibitors (SRIs) and 24 SRI-free – no group performance differences were found on a comprehensive neuropsychological battery (Mataix-Cols et al., 2002). In contrast, a box-score review in adults suggested greater executive and processing speed impairment among medicated individuals (SSRIs) relative to those not taking medication (Kuelz et al., 2004). Although medication and comorbidity are likely to negatively influence performance on neuropsychological testing (Henin et al., 2009), further studies are needed to clarify the impact of medication or comorbidity on neurocognitive functioning in youth with OCD.

Nevertheless, a number of promising studies have examined neuroexecutive differences (inhibitory deficits in particular) between OCD, Attention Deficit Hyperactivity Disorder (ADHD) and Tourette Syndrome (TS) given the putative disruption in PFC-subcortical pathways inherent in each disorder (Gilbert et al., 2004). Comorbidity is common in childhood OCD (Lewin and Piacentini, 2009) with 16–20% having comorbid ADHD and 11–38% TS (AACAP, 2012). Generally studies converge, suggesting executive dysfunction (in particular, on assessments of response inhibition, e.g. sorting and Stroop tasks) among individuals with OCD and ADHD (Abramovitch et al., 2012). Although frontal-striatal dysfunction is implicated in both OCD and ADHD studies contrasting OCD and ADHD groups directly suggest neurocognitive deficits (and thus the underlying neural substrates) may be disorder-specific (Rubia et al., 2010, 2011) with some support for a direct relation between the severity of OCD symptoms and implicit learning problems (Vloet et al., 2010; Abramovitch et al., 2012). In addition, several studies suggest greater cognitive impairment among adults with OCD and depressive disorders (Moritz et al., 2001) as well as suggesting added executive functioning difficulties in adults with anxiety disorders (Basso et al., 2007). Other studies (in non-OCD youth) suggest that neurocognitive impairment may be compounded by the burden of multiple comorbidities (Sukhodolsky et al., 2010; Vloet et al., 2010).

Given the state of the current research on youth with OCD and questions remaining about prevalence of cognitive sequelae, the magnitude of cognitive sequelae, the influence of comorbidity, the impact of psychotropic medication, and the specific areas of deficit in executive and memory measures in youth with OCD, the current study had four primary aims. First, we aimed to determine the rates of neurocognitive sequelae, specifically executive- and memory-related deficits presumed to be associated with the PFCT loops, in youth with OCD. Second, we sought to determine the magnitude of neurocognitive sequelae and the relationship between the magnitude of neurocognitive impairment and the clinical phenomenology of pediatric OCD. Third, we aimed to examine differences in rates and magnitude of cognitive sequelae between individuals with and without comorbid conditions as well as individuals taking or not taking psychiatric medication. Fourth, we sought to determine the specific areas of memory and executive functions that were impaired in the youth with OCD relative to large, psychiatrically healthy, normative samples. Finally, in a series of *post-hoc* exploratory analyses, we explored the effects of comorbidity, medication status, and OCD severity on individual tests of neuropsychological functioning.

## 2. Methods

### 2.1. Participants

Participants included 96 consecutive treatment-seeking youth with OCD, who were recruited from normal clinic flow at an OCD specialty clinic. To be eligible for participation, youth had to be between 7 and 17 years of age and have a principle diagnosis of OCD. Psychotropic medication usage did not prohibit youth from participation. Children with a severe mental illness (e.g., mental retardation, autism, psychosis, bipolar disorder) or physical condition (e.g., visual impairment, hearing impairment, seizure disorder, traumatic brain injury [including concussion