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Invited Review

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

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ABSTRACT

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



**Contents**



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## 1. Introduction

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

## 2. Neurobiology of OCD

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

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

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

## 3. Neuropsychological performance across domains

### 3.1. Executive functions

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

#### 3.1.1. Response inhibition

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

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