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Does impairment in neuropsychological tests equal neuropsychological impairment in obsessive-compulsive disorder (OCD)? Momentary influences, testing attitude, and motivation are related to neuropsychological performance in OCD

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ABSTRACT

Meta-analyses conclude that individuals with obsessive-compulsive disorder (OCD) share neurocognitive deficits. The aim of the present study was to examine the impact of attitude towards neuropsychological assessment, symptoms during assessment, and performance motivation on test results in OCD.

Thirty OCD and 30 nonclinical individuals were assessed with a comprehensive battery of neuropsychological tests. Before and after testing, participants completed the newly developed *Momentary Influences, Attitudes and Motivation Impact on Cognitive Performance Scale* (MIAMI). The experimenter rated the patients' momentary influences, attitude, motivation, and OC behavior during testing.

Patients with OCD performed worse than controls on five out of ten outcome measures. Patients were more fearful about the test outcome and complained about more negative influences during testing than controls did. The MIAMI total score mediated the relationship between group and speed. When the MIAMI score was entered as a covariate, group differences for speed were nonsignificant. No group differences emerged between patients high on motivation and those with few negative momentary influences (as assessed by the experimenter) compared to controls.

OCD patients and controls differ greatly with regard to attitude towards testing and momentary influences, which likely represents a substantial source of secondary malperformance in patients. Consequently, we call for greater caution when interpreting group differences in neuropsychological studies on OCD. Contextual and motivational variables need to be controlled for. It is also necessary to avoid general and potentially stigmatizing inferences if group differences are due to malperformance in only a subgroup of patients.

1. Introduction

The interest in the neuropsychological profile of patients with obsessive-compulsive disorder (OCD) remains strong. Although the vast majority of empirical studies have shown evidence of neuropsychological impairment in OCD (Abramovitch, Abramowitz, & Mittelman, 2013; Kuelz, Hohagen, & Voderholzer, 2004; Shin, Lee, Kim, & Kwon, 2014; Tallis, 1997), the literature is not fully consistent, and emerging evidence hints at a more complex picture.

The present study investigated the potential impact of motivation, momentary influences, and test anxiety on neurocognitive performance in OCD, following up on preliminary evidence related to the relevance of these factors. Before we lay out our specific hypotheses, we briefly review the literature on neuropsychological functioning in OCD, first presenting affirmative evidence of impaired neurocognitive performance in OCD (thesis) and then turning to a number of studies that failed to find impaired neurocognition in OCD (antithesis). Finally, we discuss previously studied moderators of malperformance in OCD and introduce preliminary evidence for the potentially important role of motivation, momentary influences, and test anxiety on neuropsychological performance in OCD (synthesis). Thus, with the present study we seek to shed light on the inconsistencies in the literature and would like to raise awareness for the specific conditions under which impairment in neuropsychological tests may not equal neuropsychological impairment in OCD.

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1.1. Evidence for neuropsychological impairment in OCD and potential consequences (thesis)

Early theorists, such as Sigmund Freud (1963, p. 68) claimed that patients with OCD had above average cognitive abilities. In contrast most studies and virtually all reviews and meta-analyses to date (Abramovitch et al., 2013; Benzina, Mallet, Burguière, N'Diaye, & Pelissolo, 2016; Kuelz et al., 2004; Shin et al., 2014; Snyder, Kaiser, Warren, & Heller, 2015; Tallis, 1997) converge on the assumption that patients with OCD share circumscribed neuropsychological deficits that are (allegedly) tied to deficient brain circuitries (Pauls, Abramovitch, Rauch, & Geller, 2014; Peng et al., 2012; Piras et al., 2015; Rotge et al., 2010). For approximately 20 years, it has been suspected that these deficits are not ubiquitous but are most prominent for nonverbal material (Tallis, 1997). Patients usually show better results for verbal compared to nonverbal tests (Abramovitch et al., 2013; Shin et al., 2014). The question of whether or not patients with OCD have neurocognitive deficits is of great interest for a number of reasons. First, if it is true, cognitive remediation may help to ameliorate these deficits and perhaps open an indirect therapeutic route for the alleviation of OCD and related disorders such as hoarding (DiMauro, Genova, Tolin, & Kurtz, 2014). Second, reports about neurocognitive impairments in OCD as well as other disorders may provide relief to some patients ("It is not me; it's my OCD") but may unsettle others, compromise self-esteem, and affect subjective illness models. A biogenetic explanation may alleviate blame, but a recent meta-analysis found that it induces pessimism and may compromise recovery from psychological problems (Kvaale, Haslam, & Gottdiener, 2013). Biogenetic models in general seem to promote the stereotype that patients with mental disorders are dangerous (Kvaale et al., 2013). Defeatist beliefs are likely fueled by reports tying cognitive deficits to brain abnormalities (Pauls et al., 2014) and may deter individuals with OCD from seeking therapy. To illustrate, a reanalysis of a recent study (Moritz et al., 2016) demonstrates that 36% of patients with OCD responded affirmatively to the question, "OCD is a brain disorder—so does that mean I can't do anything to change it?"

1.2. Some evidence against neuropsychological impairment in OCD (antithesis)

Not all studies have detected neurocognitive deficits in OCD (Abramovitch et al., 2013). For many domains, the evidence is mixed (for a critical review, see Abramovitch & Cooperman et al., 2015), and a number of studies even failed to find group differences for nonverbal functioning, for which the evidence is most solid. In one large study (Moritz et al., 2005), a comprehensive battery of tests measuring elementary nonverbal visuospatial functions was administered to 71 OCD patients as well as 30 healthy and 33 psychiatric controls. The authors aimed to elucidate which cognitive subcomponents are associated with malperformance in complex nonverbal tasks like Block Design, which encompasses a variety of spatial skills (e.g., matching patterns of different size under speed, handling three-dimensional material). No visuospatial component proved to be specifically impaired in OCD. While patients with OCD performed indeed more poorly than controls on Block Design, their scores were still in the normal range according to norm values (i.e., scaled scores), and group differences were largely owing to above-normal performance of controls. In another study, patients did not display impairment in a newly designed memory task, the Picture Word Memory Test (PWMT), which assesses verbal versus nonverbal memory (Moritz, Kloss, von Eckstaedt, & Jelinek, 2009). Finally, a study that administered a test battery encompassing everyday memory (verbal and nonverbal tasks assessing short-term, long-term and prospective memory), the Rivermead Behavioral Memory Task (RBMT), failed to discriminate between the patient and control groups (Jelinek, Moritz, Heeren, & Naber, 2006).

1.3. Moderators of malperformance in OCD (synthesis)

The aforementioned studies that failed to detect group differences must be considered outliers in light of the overarching bulk of evidence indicating neurocognitive deficits in OCD (Abramovitch et al., 2013; Kuelz et al., 2004; Shin et al., 2014; Tallis, 1997). Yet, because impairments are less consistently found in OCD compared to other psychiatric disorders, most notably schizophrenia (Fatouros-Bergman, Cervenka, Flyckt, Edman, & Farde, 2014; Keefe & Harvey, 2012), and because for some domains the evidence is in fact mixed (Abramovitch & Cooperman et al., 2015), researchers have increasingly embraced the possibility that deficits are perhaps confined to a subgroup of patients (e.g., Hwang et al., 2007) or represent epiphenomena of underlying factors. Although the review by Abramovitch et al. (2013) suggests large effect sizes for nonverbal memory impairment (which is possibly tied to executive dysfunction in OCD), the authors remain cautious in their inferences: "Furthermore, it is not clear whether these observed differences play any role in the development of OCD, or whether they are epiphenomena of having OCD" (p. 1169). The search for moderators has, however, produced largely equivocal results. An early claim that neuropsychological performance is only found in patients high on depression symptoms (Basso, Bornstein, Carona, & Morton, 2001; Moritz et al., 2001; Moritz, Kloss, Jahn, Schick, & Hand, 2003) has not been consistently replicated (Abramovitch et al., 2013). The impact of psychotropic medication, particularly antipsychotic agents, on performance has also been tested. These are increasingly prescribed in patients with OCD and may compromise performance in timed tests (Fervaha et al., 2015). Again, there is some evidence for this hypothesis, but it is not conclusive (Benzina et al., 2016; Kuelz et al., 2004). Abramovitch et al. (2013) write, "This indicated that in studies where more OCD participants were using neuroleptics, these participants performed more poorly relative to healthy controls. However, when a correction for multiple comparisons was applied (p = .01), these correlations were no longer significant" (p.1167).

Recently, studies have examined whether deficits in task performance could be owing to motivational deficits (Moritz, Hottenrott, Jelinek, Brooks, & Scheurich, 2012) or to distraction by the presence of OCD symptoms during assessment (see the executive overload model by Abramovitch, Dar, Hermesh, & Schweiger, 2012). In a recent study, a substantial subgroup of OCD patients affirmed OCD-related worries and motivational problems (Moritz et al., 2012), whereby motivation as well as checking were significantly associated with malperformance on neuropsychological tests. The relationship between checking and neurocognitive performance is particularly noteworthy as it provides a rather simple nonbiological explanation for nonverbal deficits in patients. Patients may have specific difficulties with material that involves order, symmetry (e.g., the Rey–Osterrieth Complex Figure Test), or touching (e.g., Block Design); such tasks may easily trigger and/or interfere with obsessive fears (e.g., contamination) and compulsions (checking). A methodological limitation of the study was that patients were asked to rate their level of motivation and attitude following assessment. Self-rating may have been biased by two confounding factors. First, patients may not be fully aware of their problems due to metacognitive deficits, and second, overtly poor performance may elicit self-serving tendencies to blame one's motivation and negative circumstances rather than one's own reduced cognitive capacity.

1.4. The present study

The present study aimed to address the apparent heterogeneity of findings across neuropsychological studies in OCD (Abramovitch & Cooperman et al., 2015). As noted above, the evidence in favor of neurocognitive deficits in OCD is overwhelming. Yet, there are several empirical exceptions, and some studies indicate that poor cognitive results partly reflect confounding and secondary conditions.