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No deficits in nonverbal memory, metamemory and internal as well as external source memory in obsessive-compulsive disorder (OCD)

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ABSTRACT

A large body of literature suggests that some symptoms of obsessive-compulsive disorder (OCD) result from mnemonic dysfunctions. The present study tested various formulations of the memory deficit hypothesis considering important moderators, such as depression and response slowing. Thirty-two OCD patients and 32 healthy controls were presented verbal or nonverbal instructions for actions (e.g. simple gestures). These actions should either be performed or imagined. For recognition, previously presented as well as novel actions were displayed. Decisions had to be made whether an action was previously displayed (verbally vs. nonverbally) or not and whether an action was performed or imagined (internal source memory). Moreover, both judgments required confidence ratings. Groups did not differ in memory accuracy and metamemory for verbally presented material. Patients displayed some impairment for nonverbally presented material and imagined instructions, which, however, could be fully accounted for by response slowing and depressive symptoms. The study challenges the view that primary memory deficits underlie OCD or any of its subtypes. We claim that research should move forward from the mere study of *objective* impairment to the assessment of cognitive performance *in conjunction with* personality traits such as inflated responsibility.

Introduction

Early theories on the pathogenesis of obsessive-compulsive disorder (OCD) have emphasized the role of hygiene education and childhood events while cognitive functions and intellect were considered as either spared or even higher-than-normal (Freud, 1963, p. 98). In contemporary theories this view has almost reversed. Today, many theoretical accounts focus on objective cognitive impairment in OCD and ascribe early life-events a somewhat negligible role. Apart from executive functioning, such as deficits in alternation learning (Abbruzzese, Bellodi, Ferri, & Scarone, 1995; Abbruzzese, Ferri, & Scarone, 1997; Cavedini, Ferri, Scarone, & Bellodi, 1998; Moritz, Fricke, Wagner, & Hand, 2001), memory dysfunction is targeted as a major cognitive mechanism of OCD symptoms (for reviews see Kuelz, Hohagen, & Voderholzer, 2004; Muller & Roberts, 2005; Olley, Malhi, & Sachdev, 2007; Woods, Vevea, Chambless, & Bayen, 2002), especially checking. In this view checking can be understood as an overcompensation of primary memory deficits, for example, arising from problems to mistake performed actions as only imagined (i.e. deficits in internal source memory according to the definition by Johnson, Hashtroudi, & Lindsay, 1993).

Since the start of systematic research in this area in the late 1970s and early 1980s of the last century (Reed, 1977; Sher, Frost, & Otto, 1983; Sher, Mann, & Frost, 1984) the memory deficit hypothesis has witnessed various variants. While some studies postulated a generalized memory deficit, a position soon rejected as untenable in view of uncompromised verbal memory in OCD (Tallis, 1997), subsequent research targeted single aspects of memory. A number of researchers have claimed primary problems with memory for actions/reality monitoring (Ecker & Engelkamp, 1995; Rubenstein, Peynircioglu, Chambless, & Pigott, 1993; Sher, Frost, Kushner, Crews, & Alexander, 1989; Zermatten, Van der Linden, Laroi, & Ceschi, 2006) which, however was not verified by all researcher (Brown, Kosslyn, Breiter, Baer, & Jenike, 1994; Constans, Foa, Franklin, & Mathews, 1995; Hermans, Martens, De Cort, Pieters, & Eelen, 2003; Merckelbach & Wessel, 2000). Others have highlighted nonverbal memory deficits (for a review see Muller & Roberts, 2005) and most recently metamemory (memory confidence and memory vividness), whereby evidence is again mixed for the latter domain (Cabrera, McNally, & Savage, 2001; Dar, 2004; Foa, Amir, Gershuny, Molnar, & Kozak, 1997; McNally & Kohlbeck, 1993; Moritz, Jacobsen, Willenborg, Jelinek, & Fricke,

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2006; Moritz, Kuelz, Jacobsen, Kloss, & Fricke, 2006; Moritz et al., 2007; Tekcan, Topcuoglu, & Kaya, 2007; Tolin et al., 2001; Tuna, Tekcan, & Topcuoglu, 2005). Others have attributed memory dysfunction to secondary influences such as executive impairment (Anderson & Savage, 2004), response slowing and depression (Basso, Bornstein, Carona, & Morton, 2001; Moritz et al., 2001; Moritz, Kloss, Jahn, Schick, & Hand, 2003). Despite many studies reporting no or only weak evidence for primary mnestic impairment in OCD, this line of research has remained influential (for reviews see Kuelz et al., 2004; Muller & Roberts, 2005; Olley et al., 2007).

Inconsistencies are presumably owing to a number of confounding factors. For example, inferences from many studies are limited by small sample sizes ($n < 20$) and differences in design. Further, few studies have addressed the various formulations of the memory deficit hypothesis concurrently, thus limiting the generalizibility of findings to other variants of the hypothesis and leaving the possibility that malperformance on some tasks may stem from generalized performance deficits or overall response slowing. In particular, in timed tests and paradigms where stimuli are presented for a limited duration, psychomotor retardation (i.e. response slowing) will compromise virtually all results, whether or not higher cortical functioning is affected.

In a recent study, we have looked at several of the aforementioned memory aspects concurrently (Moritz, Kloss, Vitzthum von Eckstaedt, & Jelinek, in press). In this study, we presented participants with OCD and healthy controls verbal (words difficult to visualize) and nonverbal items (drawings difficult to verbalize) and asked them to reproduce these items over three leaning trials. Without prior presentation, a final recall was requested after 20 min followed by a final recognition task. For the latter, participants made old–new judgments graded for confidence. Participants with OCD performed equally well on all parameters including memory confidence and nonverbal memory. Small effect sizes assert that huge sample sizes ($N > 350$) would have been needed to show significant effects. However, this study did not address whether patients share problems with action memory. As stated above, Sher et al. among others propose that checkers may be biased to misattribute performed actions as imagined (Sher et al., 1983, 1984), that is a problem with internal source memory (for conflicting findings on source memory see Brown et al., 1994; Constans et al., 1995; Hermans et al., 2003; Merckelbach & Wessel, 2000; Moritz, Jacobsen, et al., 2006). The present study aimed to fill this gap. Participants either had to perform or to imagine verbally or nonverbally presented actions, such as to form two fingers to a victory sign. For recognition, we presented old instructions with unprecedented ones. For memory accuracy, participants made judgments for the presentation mode (verbal vs. nonverbal) and internal source memory (i.e. whether an action was performed and imagined), both graded for response confidence. As results in the literature are inconsistent possibly owing to problems with power in some studies and to guard against false-negative findings, a large sample was recruited and exploratory contrasts were conducted even if ANOVA models failed to reach significance. If group differences emerged, we expected that these could be accommodated for by secondary influences such as comorbid depression or response slowing but not OCD symptoms.

Methods

Participants

We recruited 32 in- and outpatients diagnosed with obsessive-compulsive disorder (OCD) from the Department of Psychiatry and Psychotherapy of the University Medical Center Hamburg-Eppendorf (9 male/23 female; age: 34.00 [$SD = 10.88$]; years of formal school education: 11.56 [$SD = 1.68$]). Clinical diagnoses relied on structured interviews with the Neuropsychiatric Interview (MINI, Sheehan et al., 1998). Exclusion criteria for the OCD group were presence of any psychotic symptoms (hallucinations, delusions, mania), current alcohol or substance dependence, and macroscopic neurological disorders including OCD spectrum disorders such as Tourette's syndrome. Twenty patients fulfilled diagnostic criteria for either a current major depressive episode or dysthymia. Thirty-two healthy participants served as controls who were recruited by word-of-mouth and advertisements (11 male/21 female; age: 31.78 [$SD = 11.67$]; years of formal school education: 12.00 [$SD = 1.57$]). The MINI interview verified absence of lifetime psychiatric diagnosis in healthy participants.

The Yale-Brown Obsessive-Compulsive Scale (Y-BOCS, Goodman et al., 1989; Hand & Büttner-Westphal, 1991; Jacobsen, Kloss, Fricke, Hand, & Moritz, 2003) and the Hamilton Depression Rating Scale (HDRS, Hamilton, 1960) were employed to assess the severity of obsessive-compulsive ($M = 25.19$, $SD = 6.94$) and depressive symptoms ($M = 11.72$, $SD = 6.01$). Following a previous factor analytic study (Moritz et al., 2002), we segregated the Y-BOCS into three dimensions (obsessions, compulsions, resistance). For later subsidiary analyses relating to the effects of depression, we split the OCD group in the median of the HDRS total score ($M = 12$). Fifteen patients scored below the median. All participants gave written informed consent to participate.

Severity of contamination ($M = 5.43$, $SD = 4.88$), checking ($M = 6.04$, $SD = 4.54$) and overall symptom severity (total score, $M = 30.61$, $SD = 13.32$) was determined with the Obsessive-Compulsive Inventory Revised (OCI-R, Foa et al., 2002). According to the Y-BOCS checklist, 18 of the patients were both washers and checkers, 6 were pure checkers and 4 were pure washers.

Action memory task

Participants were individually tested in a quiet room by the second author. For the learning phase, participants were presented either verbal instructions or nonverbal pictograms for actions as displayed in Fig. 1. Instructions set in a green frame (presented as black in the figure) had to be performed by the participant (actions involving one extremity could be performed with either the left or right arm/leg/hand/foot), whereas action instructions set in a red frame (presented as gray in the figure) had to be imagined but not performed. In a pilot study, gestures and actions were compiled to achieve a similar level of complexity (i.e. both verbal and nonverbal information comparably often referred to the same body parts and comparably often involved symbolic gestures).

Following a short practice trial to acquaint participants with the task requirements, 18 verbal and 18 nonverbal action instructions were presented, with each half required to perform or image, respectively (9 items each). To avoid circular inferences, none of the actions was typical for OCD rituals (e.g. movements such as in washing hands or counting were not required). The computer screen displayed each instruction only once which lasted for exactly 10 s. Comprehension problems for individual items were recorded. Before recognition, we administered a filler task that took 10 min. Then, the 36 verbal instructions for the studied items were presented along with 20 novel action instructions (the recognition items were presented in a different font than the encoding items to prevent physical matching). Three responses were required: 1. corresponding instruction appeared either as text (verbal), pictogram (nonverbal) or was novel (external source memory)? 2. confidence rating on a 4-point scale ranging from 100% certain (= 1) to extremely uncertain (= 4) for the latter assessment. 3. In case, the participant thought that the action was presented, they had to judge whether it was performed or imagined (internal