Cognitive Performance and Specific Deficits in OCD Symptom Dimensions:
II. Spatial Memory and Impaired Recognition of Visuospatial Object Locations

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Abstract

Objective: To investigate spatial recognition abilities and working memory in obsessive–compulsive disorder (OCD) patients grouped according to their primary symptom dimensions. Memory has always occupied a central position in OCD research, mainly because of the notion that faulty memory processes could mediate many of the repetitive compulsive rituals. However, the role of different memory systems is not yet fully understood.
Methods: There were 68 OCD patients and 65 healthy controls who completed two tasks from the Cambridge Neuropsychological Test Automated Battery (CANTAB); a) spatial recognition memory, b) spatial working memory. Standard clinical and psychological background measures were also employed.
Results: The OCD patients were impaired in spatial recognition memory whereas spatial working memory was spared regarding performance accuracy. Selective deficits in visuospatial recognition memory were associated with patients in all symptom dimensions while patients in the dimension contamination were the only ones impaired in both recognition accuracy and recognition time.
Conclusion: It is suggested that spatial memory impairments may be secondary to an inability to apply efficiently elaborated attentional and goal-oriented strategies as part of the executive system to support visuospatial recognition memory in addition to experiences of incompleteness. The clinical relevance of the findings is discussed (German J Psychiatry 2011; 14: 1-12).

Keywords: Obsessive–compulsive disorder, symptom dimensions, visuospatial recognition memory, cognitive dysfunctions, anxiety

Introduction

Obsessive–compulsive disorder (OCD) is a disabling mental disorder characterized by recurrent, intrusive thoughts and repetitive and stereotypical rituals either in response to the obsessions in order to reduce distress and anxiety or performed according to rigid rules (American Psychiatric Association, 2000). Memory was one of the first cognitive functions that were studied in OCD because of the assumption that impairments could explain why OCD patients had the urge to repeatedly carry out checking rituals in their own homes. It was generally found that memory for action was impaired (e.g., Sher et al., 1983)

and more recently it has similarly been suggested that epi-sodic memory, referring to personal recollection of events in the past, appears to be the type of memory process most relevant to OCD (Muller & Roberts, 2005). Episodic mem-ory can be broken down into verbal and non-verbal, work-ing, and visuospatial, and cognitive tasks typically assess recall and recognition performance. There are several impor-tant considerations to take into account when trying to make sense of how the obsessional thoughts and the repetitive nature of compulsions in OCD could be related to memory problems. First, many OCD rituals such as washing and checking are performed because patients feel they have not carried them out properly (Muller & Roberts, 2005) and this is linked to the 'just right' hypothesis (Leckman et al., 1994), premonitory urges (Leckman et al., 1993) 'not just right' experiences (Coles et al., 2005) and 'incompleteness' experi-ences (e.g., Ecker & Gönner, 2008). Second, a memory defi-cit and/or a lack of confidence in one's ability to remember can possibly account for checking behavior (e.g., Sher et al., 1983). Third, it has been documented that decreased mem-ory confidence may be triggered under conditions of high responsibility for the outcome of a check (e.g., Radomsky et al., 2001). Finally, obsessional doubt, linked to not having carried out a check correctly, and possibly sub-served by anxiety, could also account for repetitive compulsive actions (Muller & Roberts, 2005). On the contrary, reality monitor-ing, that is, the inability to distinguish between real and imag-ined events appears to be intact in OCD (Constans et al., 1995). Repeated checking has been found to make the actual checking event less vivid triggering repetitive compulsions to reach satisfaction, which may also implicate deficits in atten-tional processing (van den Hout & Kindt, 2003). The ab-sence of feelings of satisfaction can also be understood from an emotional point of view, that a check (turning off switches) or the perception of an event (arranging furniture in exact positions) were not satisfactorily carried out because of the urge to reach perfection or certain standards (Ecker & Gönner, 2008) and this is what has been labelled 'not just right' experiences (Coles et al., 2005) and triggers repetitive compulsive behavior to reach 'just right' experiences (Sum-merfeldt et al., 2004b). It is evident that memory impair-ments in isolation cannot account for many of the OCD rituals because the clinical picture appears very complex.

Neuropsychological performance in memory tasks do offer some insight into the processes that could account for the repetitive nature of compulsive behavior because findings do not point towards a memory impairment per se, but the problem in OCD appears to be the failure to adopt appro-priate organizational strategies to support memory, and impairments on tasks assessing verbal and non-verbal mem-ory have been postulated to involve deficits of the executive functions (e.g., Deckersbach et al., 2000; Moritz et al., 2003; Savage et al., 1999). On the other hand, verbal memory has been found to be unimpaired in OCD when spontaneous strategies are generally not required (Christensen et al., 1992; Martin et al., 1995; Mataix-Cols et al., 1999) and impaired when the presented stimuli are required to be semantically clustered to enhance encoding and later support the retrieval process, such as on the California Verbal Learning Test (CVLT; Delis et al., 1987) (Cabrera et al., 2001; Deckersbach et al., 2000; Savage et al., 2000). Similarly, performance on

the Rey-Osterrieth Complex Figure Test (RCFT; Osterrieth, 1944), probing visuospatial non-verbal memory skills, where complex line information must be organized from a stimulus card and later redrawn from memory, has also revealed defi-cits in OCD (Deckersbach et al., 2000; Lacerda et al., 2003; Savage et al., 1999). However, the results are inconsistent because intact performance in OCD has also been reported on the CVLT (Bédard et al., 2009; Burdick et al., 2008; De Geus et al., 2007) and the RCFT (Bédard et al., 2009; Simp-son et al., 2006).

The computerized memory tasks Spatial Recognition Mem-ory (SRM; Sahakian et al., 1988) and Spatial Working Mem-ory (SWM; Owen et al., 1990) were administered in the current study. They examine non-verbal memory processes and successful performance have been claimed to rely on the use of self-ordered strategies (Chamberlain et al., 2005; Ru-bies et al., 2001). On the SRM most studies have only re-ported recognition accuracy as a performance variable and findings point towards a reliable impairment in OCD (Bar-nett et al., 1999; Dittrich et al., 2010b; Nedeljkovic et al., 2009; Nielen & den Boer, 2003; Purcell et al., 1998a, b) while one study did not find a deficit (Watkins et al., 2005). Rec-ognition time has been found to be both impaired (Watkins et al., 2005) and intact (Purcell et al., 1998b). The perform-ance in OCD on the SWM is somewhat more mixed because OCD patients have been reported to make more errors when searching for tokens that are hidden in different spatial locations (Chamberlain et al., 2007; Dittrich et al., 2010b; Nedeljkovic et al., 2009; Purcell et al., 1998a, b) while similar performances to healthy controls have also been found (Barnett et al., 1999; Nielen & den Boer, 2003; Simpson et al., 2006). One study has systematically investigated the performance for patients in different symptom dimensions and found on the SRM that recognition accuracy was im-paired in obsessional patients while the error rates on the SWM was impaired in checkers and patients with a mixed obsessive-compulsive (OC) symptom profile (Nedeljkovic et al., 2009). The performances of OCD patients on these cognitive tasks have been inconsistent, which may be due to the heterogeneity of OC symptoms. Unfortunately, hetero-geneity has not been taken into account in Nedeljkovic et al.'s (2009) otherwise promising study but spoiled by over-lapping or unclear grouping criteria for OCD sub-typing. Therefore, in the current study Mataix-Cols et al.'s (2005) approach has been followed and only patients in clearly defined and temporally stable symptom dimensions have been included.

Similarly, behavioral treatment approaches to OCD have started to recognize the heterogeneity in symptoms and the importance of developing symptom specific techniques (e.g., McKay et al., 2004). Models to explain the underlying moti-vations for different OC dimensional symptomatology have been proposed (e.g., Summerfeldt et al., 2004a), which in turn can make useful recommendations to develop new rehabilitation techniques. In essence, two motivational fac-tors have been suggested to be involved in selective OC symptom dimensions (Ecker & Gönner, 2008; Summerfeldt et al., 2004a). They are 'harm avoidance' (e.g., locking the door to prevent a break-in) and 'incompleteness' (e.g., re-arranging books on a shelf). Research shows that symme-try/ordering symptoms are motivated by 'incompleteness'

while checking (safety, aggression) is associated with both 'incompleteness' and 'harm avoidance', but contamination symptoms with neither 'incompleteness' nor 'harm avoidance' (Ecker & Gönner, 2008). Feelings of 'incompleteness' appear to be most integral to patients in the dimension symmetry/order whereas 'harm avoidance', thought to trigger checks to prevent a fire or break-in, to be most relevant to patients in the dimensions safety and aggression. Moreover, subsequent checking carried out by patients in all three symptom dimensions can for different reasons be motivated by a sense of dissatisfaction. Although these are clinical approaches to understand and explain OC symptomatology, the neuropsychological approach has the ability to test whether these assumptions can be elucidated in cognitive task performances. For example, the notion that a sense of 'incompleteness' is associated with patients in the dimension symmetry/order can be tested with the SRM and the SWM because performances rely on the ability to integrate and organize visual stimuli. It has also been recognized that the SRM and SWM depend on processing in the dorsolateral prefrontal cortex (DLPFC) (Owen et al., 1996) and this study therefore examined specifically whether visuospatial memory is mediated by impairments in executive strategy failures.

It was hypothesized that the OCD patients compared to the healthy controls would be impaired in the number of spatial locations recognized on the SRM. For the SWM it was predicted that the OCD patients would make more between-search errors compared to the healthy controls. It was further predicted that patients in the symptom dimensions safety, aggression and symmetry/order would be most impaired in spatial locations recognized on the SRM and between-search errors on the SWM compared to patients in the dimensions contamination and sexual/religious and the healthy control group.

Methods

Participants

There were 68 OCD patients (43 female, 25 male) meeting criteria for a DSM-IV-TR (American Psychiatric Association, 2000) diagnosis and 65 healthy controls (44 female, 21 male) who participated. The OCD patients who volunteered to take part were recruited from a specialist out-patient mental health center at a hospital in the South-East of England. Mean age in the OCD group was 42.1 years (standard deviation (SD) = 12.8) compared to 38.0 years (SD = 14.6) in the healthy control group. At the time of testing 61 OCD patients received stable doses of selective serotonin reuptake inhibitor (SSRI) medication such as paroxetine, citalopram, fluvoxamine, fluoxetine and sertraline. These SSRIs are commonly prescribed for individuals who suffer from OCD. The remaining seven had been free from psychotropic medication for at least six months prior to taking part in the study. The OCD patients who presented with a co-morbid DSM-IV-TR Axis I diagnosis, current or history of alcohol or other substance abuse, neurological illness, head injury, Tourette's syndrome, tic-spectrum disorders, attention-deficit/hyperactivity disorder, and schizo-obsessive disorder were excluded.

The healthy control group was matched to the OCD group according to gender, handedness, age, years in formal education, and predicted verbal IQ. The healthy participants who volunteered to take part were recruited from the University of Hertfordshire, staff members of the hospital with the mental health center as one of its departments and the general Hertfordshire population by newspaper and posted advertisements. Exclusion criteria for the healthy control group constituted not having experienced current or past history of DSM-IV-TR Axis I disorders according to assessment with the Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998).

The OCD patients were grouped into their primary symptom dimension on the basis of their current primary obsessions and/or compulsions assessed by the Dimensional Yale-Brown Obsessive-Compulsive Scale (DY-BOCS; Rosario-Campos et al., 2006), which is a standardized instrument to evaluate the presence and severity of obsessions and compulsions for patients in different symptom dimensions. Detailed hospital case notes for each patient were also consulted to confirm or disconfirm the primary symptom dimension established by the DY-BOCS at the time of recruitment. If conflicts arose between the DY-BOCS grouping and the hospital case notes for a particular patient, the primary symptom dimension was decided upon after consulting the psychiatrist treating the patients. The OCD patients were categorized in five OC symptom dimensions according to the DY-BOCS interview. In this study the DY-BOCS symptom dimension 'obsessions about harm due to aggression/injury/violence/natural disasters and related compulsions' has been further divided into two sub-dimensions related mainly either to idiocentric (self-related safety) or allocentric (aggression) obsessions and compulsions. Aggression in the dimension aggression is used in the sense of directed to others, whereas aggression in the dimension safety is inferred through the concerns related to the protection of self. These two dimensions are what the OCD literature would normally label as classical 'checkers'. The following OC symptom dimensions were established from the patient sample:

1) obsessions about harm due to aggression/injury/violence/natural disasters predominantly to themselves including an urge to feel safe and protect the self and related compulsions (safety, n = 22)

2) obsessions about harm due to aggression/injury/violence/natural disasters predominantly to family members and others and related compulsions (aggression, n = 6)

3) obsessions about symmetry/'just-right' perceptions and compulsions to count or order/arrange (symmetry/order, n = 14)

4) contamination obsessions and cleaning compulsions (contamination, n = 21)

5) obsessions concerning sexual/moral/religious and related compulsions (sexual/religious, n = 5)

The study was approved by the Hertfordshire Partnership NHS Trust Local Research Ethics Committee, UK. Data in this manuscript were obtained according to the Helsinki Declaration.

Design

The experimental study used a mixed design, with the between-subjects factor group (OCD or OC symptom dimensions/healthy controls) and the within-subjects factor for the SWM task was difficulty level (4/6/8 box-search problems).

Materials

The clinical and psychological testing measures and the two neuropsychological tasks administered in the current study are separately described below.

Clinical and psychological testing

The severity of OCD was quantified with the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS; Goodman et al., 1989) and the extended clinical interview was supplemented with the MINI. Depression mood was quantified with the Montgomery-Åsberg Depression Rating Scale (MADRS; Montgomery & Åsberg, 1979) and anxiety was assessed with the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1983). Cognitive Assessment Instrument of Obsessions and Compulsions (CAIOC-18, 18-item version) evaluated the cognitive and executive impairments that are hypothesized to underpin the impact of OCD symptoms on functioning (Dittrich et al., 2010a, 2011a). Obsessive-compulsive personality symptoms were rated with the Compulsive Personality Assessment Scale (CPAS; Fineberg et al., 2007) and psychosocial impairment was quantified with the Sheehan Disability Scale (SDS; Sheehan et al., 1996). The Locus of Control (LoC; Rotter, 1966) scale assessed the extent to which individuals believe that they can control events that affect them. Predicted verbal IQ was estimated using the National Adult Reading Test (NART; Nelson, 1982). The healthy control group was administered the same clinical and psychological background measures as the patient group.

Neuropsychological tasks

The CANTAB (Cambridge Cognition, 2006) SRM task presents the participants with a forced-choice paradigm. A white square appears one at a time for three seconds on a computer screen in a sequence at five randomly different locations in the presentation phase. In the recognition phase the five squares are presented on the screen individually, but in the reversed order to the original presentation phase. This time, each square is paired with one which is in a place not previously seen in the presentation phase. The participants must touch the square that was in the same place as seen in the presentation phase, and avoid touching the incorrectly

positioned square. The task consists of four blocks each with five new locations. Dependent variables were mean percentage of correct spatial locations recognized for the 20 trials and mean correct latency (or recognition time) to choose the spatial locations in the recognition phase.

The CANTAB SWM task is a self-ordered test that requires the participants to search for blue tokens hidden inside an array of boxes. The participants use a touch screen to select the box they want to open and once a token is found, they place it in a column area called 'home' on the right hand side of the screen. Then a new token is hidden in a different box because the token never appears in the same box twice during the same search sequence and the number of tokens to be found corresponds to the number of boxes on the screen. The trial is completed when a token has been found in each box. Four practice trials were given each with three boxes and the assessed trials included four blocks of 4, 6 and 8 boxes. The total number of between-search errors (returning to a box where a token has already been found during the same trial) at the 4, 6 and 8 box difficulty levels were calculated. Strategy scores were also obtained indicating how often a search sequence was initiated from the same box in each trial and reflect the ability to use a systematic searching approach. Strategy scores ranged from 1-37 and lower scores indicate superior use of self-ordered strategies to optimize performance. Mean correct search latency was also assessed.

Procedure

On the day of testing during the clinical interview the patients were screened with the MINI to exclude past and present history of mental problems. During the same session, ratings of OCD severity (Y-BOCS), depression (MADRS), OC personality (CPAS), predicted verbal IQ (NART) and the primary OC dimensional symptom profiles of the patients (DY-BOCS) were established. The self-rated background questionnaires STAI, CAIOC, SDS and the LoC were either completed on the day of recruitment or at home and posted back using a pre-paid envelope. In total the clinical interview and the administration of the tasks took 1h and 30 minutes to complete. The healthy control participants were rated on clinical measures (Y-BOCS, MADRS, CPAS) and the NART. The self-rated clinical and psychological measures (STAI, CAIOC, SDS, LoC) were completed on the day of testing. The neuropsychological tasks were administered in a quiet room in the hospital clinic or at the University of Hertfordshire.

Data Analysis

The data were analyzed using the Statistical Package for the Social Sciences version 16.0 (SPSS Inc., 2008). The categorical variables gender and handedness were subject to Pearson chi-square analyses. The data from the clinical and psychological measures were analyzed with independent-samples t tests (OCD and healthy control group) and one-way analysis variance (ANOVA; patients in OC symptom dimensions and healthy control group). The data from the neuropsychologi-

Table 1. Gender and handedness characteristics in the OCD and healthy control group

	OCD (n = 68)	Healthy controls (n = 65)	
Variable	Frequency	Frequency	X^2-test
Gender (F : M)	43 : 25	44 : 21	n.s.
Hand (right : left)	57 : 11	59 : 6	n.s.

Note. OCD, Obsessive–Compulsive Disorder; Degrees of freedom (df; X^2-test) = 1

cal task performance were submitted to repeated-measures ANOVA and one-way ANOVAs. Post-hoc least significant difference tests were performed to follow up main effects. All statistical tests were two-tailed and the significance level alpha was set at 0.05. The partial eta squared (η^2_p) was used as an effect size measure, which indicates the proportion of total variability attributable to a factor. A η^2_p of .01 is considered a small effect size, .059 a medium effect size and ≥ .138 a large effect size (Cohen, 1988). In the OCD group, correlations between the Y-BOCS, MADRS, STAI-state, STAI-trait, CAIOC and the neuropsychological task measures were examined using Pearson product-moment correlation. A logarithmic (Base 10) transformation was performed for the SRM and SWM latency scores to reduce skewness and improve normality. For readability it was decided to display the untransformed scores in tables and figures while the statistics for transformed data were reported in the text because for all comparisons statistical analysis of both scores were consistent with each other.

Results

Demographic, clinical, and psychological background measures

The OCD and the healthy control group did not differ on the demographic variables gender and handedness (Table 1).

The two groups did not differ significantly in age, education, and predicted verbal IQ but the OCD patients scored significantly higher than the healthy controls on the clinical and psychological measures (Table 2).

The patients in the OC symptom dimensions and the healthy control group did not differ on the demographic variables gender and handedness (Table 3).

The patients in the symptom dimensions and the healthy control group did not differ in age, years in formal education, and predicted verbal IQ, while, as expected, patients in all symptom dimensions had significantly higher scores on the clinical measures compared to the healthy controls (p < .001 for all; Table 4). On the SDS, patients in the symptom dimensions scored significantly higher than the healthy control group (p < .01 for all) and in addition, patients in the dimension symmetry/order obtained significantly higher scores compared to patients in the dimension sexual/religious (p = .038). On the LoC, the healthy control group endorsed fewer external control of event statements compared to patients in the dimensions aggression (p = .032), symmetry/order (p = .042) and sexual/religious (p = .028).

Neuropsychological performance: SRM

Two independent-samples t tests were conducted to examine the performance in the OCD and healthy control group for mean percentage of correct locations recognized and mean

Table 2. Clinical and psychological background characteristics in the OCD and healthy control group

	OCD (n = 68)		Healthy controls (n = 65)		
Variable	Mean	SD	Mean	SD	t-test
Age (years)	42.1	12.8	38.0	14.6	n.s.
Education (years)	3.9	2.8	4.0	2.0	n.s.
Verbal IQ	115.3	5.1	115.8	5.9	n.s.
Y-BOCS (max 40)	18.8	7.2	2.4	2.1	17.636***
MADRS (max 60)	13.7	7.5	3.4	2.9	10.353***
STAI-state (max 40)	51.3	14.4	32.4	10.2	8.733***
STAI-trait (max 40)	56.2	11.5	36.8	9.5	10.568***
CAIOC (max 108)	61.7	19.7	28.1	14.5	11.140***
CPAS (max 32)	14.8	5.9	6.1	3.4	10.478***
SDS (max 30)	16.1	7.6	3.5	4.8	11.435***
LoC (max 23, external)	13.4	3.8	11.6	3.1	2.911**

Note. CAIOC-18, Cognitive Assessment Instrument of Obsessions and Compulsions; CPAS, Compulsive Personality Assessment Scale; LoC, Locus of Control; MADRS, Montgomery-Åsberg Depression Rating Scale; OCD, Obsessive–Compulsive Disorder; SD, Standard Deviation; SDS, Sheehan Disability Scale; STAI, State-Trait Anxiety Inventory; Y-BOCS, Yale-Brown Obsessive Compulsive Scale; df (t-test) = 131; ***p < .001; **p < .01

Table 3. Gender and handedness characteristics for patients in the OC symptom dimensions (SA, safety; AG, aggression; SO, symmetry/order; CO, contamination; SR, sexual/religious) and healthy control group (HC)

	SA (n = 22)	AG (n = 6)	SO (n = 14)	CO (n = 21)	SR (n = 5)	HC (n = 65)	
Variable	Frequency	Frequency	Frequency	Frequency	Frequency	Frequency	X^2-test
Gender (F : M)	12 : 10	3 : 3	10 : 4	16 : 5	2 : 3	44 : 21	n.s.
Hand (right : left)	19 : 3	11 : 3	6 : 0	17 : 4	4 : 1	59 : 6	n.s.

Note. df (X^2-test) = 5

correct latency. The OCD patients (Mean (M) = 77.8%, SD = 11.0) recognized significantly fewer correct locations compared to the healthy controls (M = 84.5%, SD = 9.1), t (131) = 3.811, p < .001. For latency, the OCD patients (M = 2942 milliseconds (ms), SD = 1044) took significantly longer to recognize the spatial locations than the healthy controls (M = 2281 ms, SD = 637), t (131) = 4.308, p < .001.

Two one-way ANOVAs were conducted to examine the performance for recognition accuracy and recognition time for patients in the symptom dimensions and the healthy controls. The one-way ANOVA for recognition accuracy (Figure 1) was significant, F (5, 127) = 3.446, p = .006, η^2_p = .119. Post hoc tests revealed that patients in the dimensions aggression (M = 75.8%, SD = 8.6; p = .048), contamination (M = 75.7%, SD = 13.0; p = .001) and sexual/religious (M = 75.0%, SD = 8.6; p = .046) performed worse than the healthy control group (M = 84.5%, SD = 9.1).

The one-way ANOVA for recognition time (Figure 2) was significant, F (5, 127) = 3.812, p = .003, η^2_p = .130. Post hoc tests indicated that patients in the dimensions safety (M = 3094 ms, SD = 1220; p < .001), symmetry/order (M = 3059 ms, SD = 1296; p = .007) and contamination (M = 2844 ms, SD = 799; p = .006) had significantly longer recognition times than the healthy controls (M = 2281 ms, SD = 637).

Neuropsychological performance: SWM

A repeated-measures ANOVA was conducted to compare the performance in the OCD and healthy control group in between-search errors at the 4, 6 and 8 box difficulty levels. Results revealed an expected main effect for difficulty level, F (2, 130) = 154.215, p < .001, η^2_p = .703, indicating that the participants made more errors when the level of difficulty increased. However, the level of difficulty did not interact with the group performances. The mean between-search errors in the OCD group (4 boxes: M = 1.2, SD = 2.0; 6 boxes: M = 7.4, SD = 7.2; 8 boxes; M = 18.6, SD = 12.2) and healthy control group (4 boxes: M = 0.8, SD = 1.7; 6 boxes: M = 5.9, SD = 6.3; 8 boxes; M = 18.3, SD = 12.2) were broadly similar. The mean correct search latency in the OCD patients (M = 937 ms, SD = 441) was significantly longer compared to the healthy controls (M = 780 ms, SD = 277), t (131) = 2.404, p = .018. An independent samples t-test confirmed that the strategy score for the OCD patients (M = 32.7, SD = 6.4) did not differ to that of the healthy control group (M = 32.8, SD = 5.4).

Table 4. Clinical and psychological characteristics for patients in the OC symptom dimensions (SA, safety; AG, aggression; SO, symmetry/order; CO, contamination; SR, sexual/religious) and healthy control group (HC)

	SA (n = 22)		AG (n = 6)		SO (n = 14)		CO (n = 21)		SR (n = 5)		HC (n = 65)		
Variable	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	F-value
Age	43.4	13.1	40.8	16.6	40.8	14.8	41.5	10.9	43.8	12.4	38.0	14.6	n.s.
Education	4.1	2.3	3.0	2.7	4.3	3.7	3.9	2.9	2.6	2.9	4.0	2.0	n.s.
Verbal IQ	114.1	2.8	115.2	4.3	115.0	6.3	116.9	6.5	114.6	4.2	115.8	5.9	n.s.
Y-BOCS	18.2	5.9	21.3	7.0	18.6	8.6	19.6	8.3	15.0	3.3	2.4	2.1	63.471***
MADRS	13.4	7.5	15.8	8.4	16.0	8.7	12.5	6.8	11.4	5.6	3.4	2.9	22.624***
STAI-state	51.7	15.6	52.2	15.2	50.1	15.7	51.2	13.7	52.6	11.7	32.4	10.2	14.860***
STAI-trait	56.0	11.8	59.2	12.1	55.3	15.2	56.2	10.1	55.8	3.6	36.8	9.5	21.869***
CAIOC	60.6	16.7	68.7	15.4	66.1	23.2	61.3	21.8	47.8	15.0	28.1	14.5	26.082***
CPAS	13.2	5.6	18.3	4.1	16.7	5.7	14.8	6.6	12.6	3.9	6.1	3.4	24.654***
SDS	15.8	7.0	16.0	7.9	18.1	8.6	16.3	8.2	11.2	3.5	3.5	4.8	27.142***
LoC	12.6	3.7	14.8	4.5	13.7	2.9	13.1	4.2	15.2	3.4	11.6	3.1	2.443*

Note. CAIOC-18, Cognitive Assessment Instrument of Obsessions and Compulsions; CPAS, Compulsive Personality Assessment Scale; LoC, Locus of Control; MADRS, Montgomery-Åsberg Depression Rating Scale; SD, Standard Deviation; SDS, Sheehan Disability Scale; STAI, State-Trait Anxiety Inventory; Y-BOCS, Yale-Brown Obsessive Compulsive Scale; df (one-way ANOVA) = 5,127; ***p < .001; *p < .05

Table 5. Means (M) and standard deviations (SD) of the SWM measures for patients in the OC symptom dimensions (SA, safety; AG, aggression; SO, symmetry/order; CO, contamination; SR, sexual/religious) and healthy control group (HC)

Variable	SA (n = 22)		AG (n = 6)		SO (n = 14)		CO (n = 21)		SR (n = 5)		HC (n = 65)		F-value	η^2_p
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD		
Errors 4 boxes	1.6	2.3	0.3	0.5	0.7	1.2	1.5	2.5	0.6	0.9	0.8	1.7	n.s.	.045
Errors 6 boxes	8.4	8.2	2.3	2.6	7.6	3.7	7.2	8.5	9.0	7.4	5.9	6.3	n.s.	.042
Errors 8 boxes	17.7	12.5	17.8	11.0	18.4	8.8	19.0	14.7	22.0	12.5	18.3	12.2	n.s.	.004
Strategy	33.0	7.0	33.0	4.5	33.4	5.8	31.2	7.0	35.0	5.1	32.8	5.4	n.s.	.018
Latency (ms)	1093	593	807	181	835	337	885	345	912	439	780	277	2.066	.075

Note. ms, milliseconds; df (one-way ANOVA) = 5,127

The means and standard deviations of the SWM task measures are displayed in Table 5. A repeated-measures ANOVA was conducted to examine the number of between-search errors for patients in the OC symptom dimensions and the healthy control group and results revealed the expected main effect for difficulty level, F (2, 126) = 78.935, p <.001, η^2_p = .556, but no group and difficulty level interaction was identified. A one-way ANOVA for the search latency was approaching significance, F (5, 127) = 2.066, p = .074, and further post hoc analysis revealed that patients in the dimension safety had longer latencies compared to the healthy controls (p = .002).

Correlation analysis

In the OCD group, the Y-BOCS, MADRS, STAI-state, STAI-trait and CAIOC did not correlate with any of the memory task characteristics. Within the SRM, a significant negative correlation was found between the transformed latency variable and recognition accuracy, r (68) = - .30, p = .013. A significant positive correlation was also found between the transformed latency scores on the SRM and SWM, r (68) = .55, p < .001.

Discussion

The results of this study confirmed that the OCD patients were impaired in both recognition accuracy and recognition time on the SRM and in search latency on the SWM. On the SRM, the performance for patients in the different symptoms dimensions revealed impairments in recognition accuracy for patients associated with the dimensions aggression, contamination, and sexual/religious, while patients associated with the dimensions safety, symmetry/order and contamination were impaired in recognition time. In contrast, patients in the different symptom dimensions performed to the same standard as healthy controls in terms of between-search errors on the SWM, while search latency in patients with safety concerns was impaired compared to the healthy controls. The hypothesis that the OCD patients would be impaired in remembering the spatial locations on the SRM was confirmed, but not the predicted impairment on the SWM. For the symptom dimensions, the hypothesis that patients in the dimension aggression would be impaired in recognition accuracy on the SRM was confirmed, but not the predicted impairment on the SWM.

The present results support a visuospatial memory impairment in OCD which appears to be mediated by executive function deficits (Hodgson et al., 1999), supported by the fact that performances on the SRM tap processing in the DLPFC, a brain area thought to reflect executive processing (Owen et al., 1996). Decision-making often depends on intact memory processes and it has been argued that working memory plays a crucial role insofar as representations of various options and scenarios in decision-making are held online over a period (Baddeley, 1992; Goldman-Rakic, 1992). Baddeley's influential model of working memory consists of three elements; the articulatory/phonological loop, the visuospatial sketch pad, and the executive control system (Baddeley, 1986). Rather than purely an immediate store for incoming information, working memory is seen as part of a modulated system, which is instead controlled by the central executive. It is suggested here that working memory as part of the executive system is necessary for decision-making (e.g., Dittrich et al., 2011b; Hodgson et al., 1999). In this sense, the link between memory impairments and purely executive dysfunctions is highly interesting as elucidated in the current study. More specifically, the difference between the SWM and the SRM tasks is that on the SWM the participants have to keep a representation of the spatial locations when they make decisions about which boxes to search in, while on the SRM the main task demand is to compare the stimuli locations during the presentation phase (i.e. memorized goal state) against those appearing in the recognition phase. Optimal performance on the SWM is achieved if the boxes found to be empty during the same search sequence are internally represented because it is assumed that working memory processes aid participants in making the correct search decisions. This updating of spatial locations is needed in order to avoid going back to previously empty boxes. The memory delays on the SWM are in the range of milliseconds to 15 seconds depending on the speed of the search by the participant. It is argued that in order to support intact per-
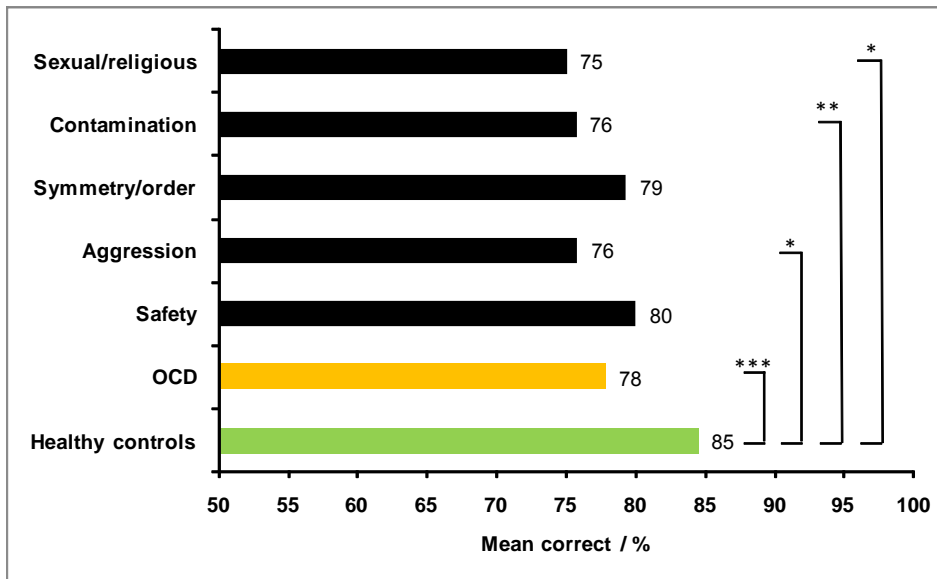
Figure 1. Mean correct recognition (%) of spatial locations in the healthy control group and patients in the OC symptom dimensions (*p < .05; **p < .01; ***p < .001)

working memory is continuously required while on the SRM short-term memory, which has components of working memory, is predominantly needed. The visuospatial sketch pad is assumed to be responsible for the short-term storage of visual and spatial information (Baddeley, 1992) and we argue that patients in selective symptom dimensions are impaired in the matching process on the SRM because the matching of internal spatial representations, most likely depending on attentional respectively executive functioning, to guide successful performance throughout the task seems to be impaired. Notwithstanding these considerations about differences in memorizing spatial information, task differences warrant further empirical testing as, in general, the role of different memory processes and modules in such tasks are not yet fully understood.

formance on the SRM a different kind of working memory process, not primarily related to spatial processing in working memory, but more related to decision-making and executive functions may be involved. Therefore, the working memory demands (i.e. attentional and goal-oriented processes) that support intact performance on the SRM seem different from SWM processing as simply demonstrated by task characteristics, for example, in SRM the memory time is defined externally by the task setting and at least, double the time as in SWM, up to a minute even (30-60 seconds). In order to make decisions in single cases about which squares have been identified in which locations, it is rather a question of comparing short term memory for old and new locations. However, it might be difficult in individual cases to dissociate between short-term memory and working memory processes. Consequently, on the SWM task

The current latency impairment on the SRM in the OCD group confirms a similar finding reported by Watkins et al. (2005). However, they argued that the optimal recognition accuracy revealed in OCD in their study may reflect intact DLPFC activity (Owen et al., 1996), but so far no study has supported this claim, including the present findings and earlier reports (Barnett et al., 1999; Nielen & den Boer, 2003; Purcell et al., 1998a, b). Furthermore, the brain imaging study in OCD by Mataix-Cols et al. (2004) needs to be considered following the findings in the present study. They found during symptom provocation that patients in the dimension c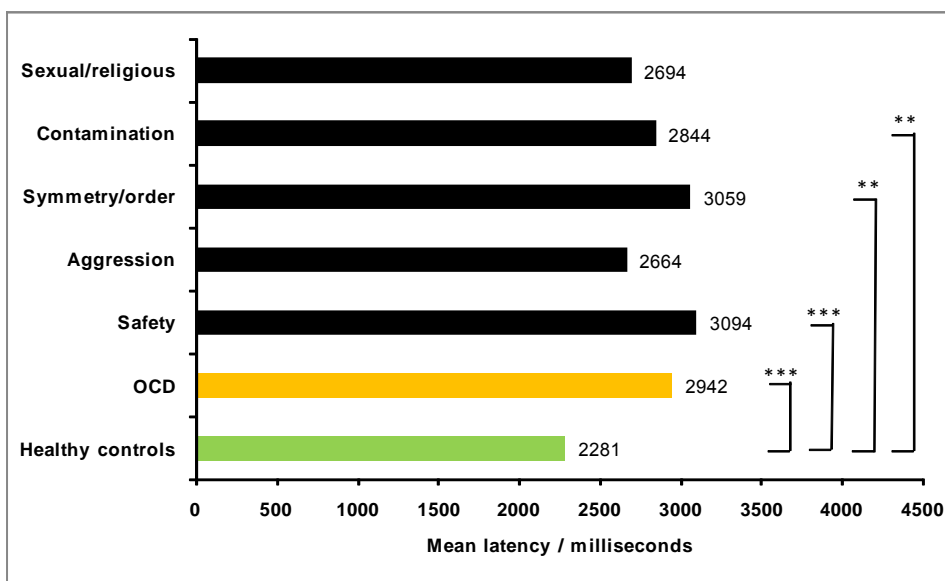ontamination were associated with abnormal activity in the ventromedial prefrontal cortex, whereas patients in the dimension checking/ obsession (safety, aggression) were associated with abnormal dorsal prefrontal cortical and striatal activity. These imaging results demonstrate that abnormal brain activity cannot be assumed to reflect deficits in cognitive information processing because on the SRM, which depends on DLPFC processing, patients in the dimension contamination were impaired in both recognition accuracy and latency, patients in the dimension aggression were impaired in recognition accuracy, and patients in the dimension safety showed

Figure 2. Mean correct latency (milliseconds) of spatial locations in the healthy control group and patients in the OC symptom dimensions (**p < .01; ***p < .001)

deficits in latency on both the SRM and SWM. Following the symptom provocation study it could be assumed that patients in the dimension contamination would be unimpaired on the SRM, which indicates that cognitive processing and symptom provocation processing may not directly complement each other. Furthermore, a different performance profile between checkers in the dimensions safety and aggression was found, which supports the notion that these patients should be considered separately in neuropsychological research.

It has been confirmed that symptom improvement in a group of OCD patients, as measured by reduced scores on the Y-BOCS following SSRI treatment, revealed that deficits on the SRM, but not the SWM ameliorated (Nielen & den Boer, 2003) implying that selective visuospatial deficits may remain despite symptomatic improvement. Here it was found that deficits in cognitive processing related to visuospatial working and short-term memory seem to have mediated the impaired performance, because no direct association between clinical symptoms and the SRM performance was found.

The present findings do not support the results in Nedeljkovic et al. (2009), who found checkers and patients with mixed symptoms to be impaired on the SWM (errors) and patients with obsessions only to be impaired on the SRM (recognition accuracy). Considering that both studies administered the same tasks, the different findings appear surprising, but could be explained by study recruitment and the basis for symptom dimension allocation of patients. The present study employed a standardized questionnaire (DY-BOCS) to group patients according to their primary symptom concerns whereas the Y-BOCS symptom checklist was used in Nedeljkovic et al. (2009). It is argued that the Y-BOCS symptom checklist may not reliably inform clinicians about predominant symptom dimensions in a patient sample because these are not systematically evaluated, which is in contrast to the DY-BOCS where the severity of each dimension is independently quantified. Moreover, the symptom dimensions appear on the face of it different, apart from the contamination dimension, labelled washers in Nedeljkovic et al.'s study. Their dimension labelled checkers was in the current study split into patients in the dimensions safety and aggression and their obsessional patient group did not constitute a dimension in the present study. The mixed symptom group in Nedeljkovic et al. included patients with washing, checking and symmetry/order symptoms. Therefore, comparison of results cannot be reliably attempted despite administering the same tasks. It is argued that a much more robust symptom dimensional approach (Henderson & Dittrich, 1993) is required, as adopted in the current study, in which patients showing the most temporally stable dimensions (checking, contamination and symmetry/order) as identified in factor analytic studies (e.g., Mataix-Cols et al., 2005) are recruited in order to reliably assess the neuropsychological performance.

The impairment found for patients in the dimension aggression in respect to recognition accuracy on the SRM would seem to support the association with experiences of 'incompleteness', that is, an inability to achieve closure for actions and perceptions, thereby confirming the suggestions of Ecker and Gönner (2008). However, this study also found evidence for an 'incompleteness' deficit for patients in the dimensions safety and symmetry/order, but these patients were in the current study only impaired in recognition latency on the SRM, which seems to reflect a speed trade-off strategy to avoid errors in order to verify their choices of square location in the recognition phase or it could be that these patients needed more time to integrate their perceptions, thus showing hesitancy in decision-making. In contrast to other cognitive operations such as motor inhibition, which has been suggested to be characterized by impulsivity in OCD (Chamberlain et al., 2005), it was revealed that patients in the dimension safety was characterized by the opposite. On the other hand, the speed trade-off strategy did not seem to support successful recognition performance for patients in the dimension contamination who displayed both recognition accuracy and recognition time deficits and is therefore in contrast to the suggestions by Ecker and Gönner (2008) that patients with contamination fear are not associated with experiences of 'incompleteness'. Hesitancy and impulsiveness seem contrasting cognitive operations, and should be investigated further by appropriate neuropsychological tasks.

The current study has documented that executive functions may mediate the SRM impairments in OCD and not working memory per se, confirming earlier suggestions (Hodgson et al., 1999; Savage et al., 1999). Moreover, the notions that OCD patients need things to be 'just right' (Leckman et al., 1994) and experience urges to carry out compulsive behaviors when things are 'not just right' (Coles et al., 2005) because actions and perceptions are experienced as incomplete (Ecker & Gönner, 2008) are also factors that are assumed to implicate the present findings through the interaction between cognition and emotion. The patients in the dimensions aggression, contamination and sexual/religious were found to be impaired in visuospatial recognition accuracy, which could stem from failures of different experiences strongly linked to dysfunctions in emotion perception and executive functions that negatively affected working and short-term memory to support abilities to structure information internally (Rabbitt, 1997). Although a breakdown in executive processes seems to mediate the SRM deficit, clinical symptomatology is strongly linked to emotion perception and should be taken into account for the cognitive deficit to produce symptom dimension relevance (Dittrich et al., 2010a). However, linking the sense of 'incompleteness' to neuropsychological findings must be done with some caution because it is a motivational factor derived from a questionnaire study (Ecker & Gönner, 2008) and do not fully support the present results. Nevertheless, assessing cognitive dysfunctions designing systematic neuropsychological, clinical, behavioral, and brain imaging studies is important in order to come closer to fully understand the heterogeneous nature of OCD (Henderson & Dittrich, 1993).

This study has provided new evidence for both selective and spared deficits for patients in different OC symptom dimensions related to spatial recognition and working memory. Spatial recognition memory was impaired while spatial working memory was spared. The patients in the symptom dimension contamination were the only patients impaired in both recognition accuracy and recognition time. It is sug-

gested that an inability to represent information internally following a deficient executive functioning related to decision-making and matching processes in short-term memory formation could contribute to explaining the impairments in addition to experiences of 'incompleteness'. In this sense, in OCD, spatial processing per se seems rather unaffected when using visual displays. Future research would benefit from further decomposing the component processes in cognitive tasks related to memory and executive functioning as well as emotional experience in order to refine our understanding of the basis for the apparent visuospatial mnemonic failures associated with patients in the different symptom dimension of OCD.

Acknowledgements

References

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders IV - Text Revision. Washington, DC: American Psychiatric Association 2000.

Baddeley A. Working memory. Oxford: Oxford University Press 1986.

Baddeley A. Working memory. Science 1992;255:556-559.

Barnett R, Maruff P, Purcell P, Wainwright K, Kyrios M, Brewer W, Pantelis C. Impairment of olfactory identification in obsessive–compulsive disorder. Psychological Medicine 1999;29:1227-1233.

Bédard M-J, Joyal CC, Godbout L, Chantal S. Executive functions and obsessive–compulsive disorder: on the importance of subclinical symptoms and other concomitant factors. Archives of Clinical Neuropsychology 2009;24:585-598.

Burdick KE, Robinson DG, Malhotra AK, Szeszko PR. Neurocognitive profile analysis in obsessive–compulsive disorder. Journal of the International Neuropsychological Society 2008;14:640-645.

Cabrera AR, McNally RJ, Savage C. Missing the forest for the trees? Deficient memory for linguistic gist in obsessive–compulsive disorder. Psychological Medicine 2001;31: 1089-1094.

Cambridge Cognition. CANTAB (www.camcog.com). Cambridge Neuropsychological Test Automated Battery. Cambridge, UK 2006.

Chamberlain SR, Blackwell AD, Fineberg NA, Robbins TW, Sahakian BJ. The neuropsychology of obsessive compulsive disorder: the importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. Neuroscience and Biobehavioral Reviews 2005;29:399-419.

Chamberlain SR, Fineberg NA, Blackwell AD, Clark L, Robbins TW, Sahakian BJ. A neuropsychological comparison of obsessive–compulsive disorder and trichotillomania. Neuropsychologia 2007;45:654-662.

Christensen KJ, Kim SW, Dysken MW, Hoover KM. Neuropsychological performance in obsessive–compulsive disorder. Biological Psychiatry 1992;31:4-18.

Cohen J. Statistical power analysis for the behavioral sciences (2nd ed.). Hillsdale, NJ: Erlbaum 1988.

Coles ME, Heimberg RG, Frost RO, Steketee G. Not just right experiences and obsessive-compulsive features: experimental and self-monitoring perspectives. Behaviour Research and Therapy 2005;43:153-167.

Constans JI, Foa EB, Franklin ME, Matthews A. Memory for actual and imagined events in OC checkers. Behaviour Research and Therapy 1995;33:665-671.

Deckersbach T, Otto MW, Savage CR, Baer L, Jenike MA. The relationship between semantic organization and memory in obsessive–compulsive disorder. Psychotherapy and Psychosomatics 2000;69:101-107.

De Geus F, Denys D, Sitskoorn MM, Westenberg HGM. Attention and cognition in patients with obsessive–compulsive disorder. Psychiatry and Clinical Neurosciences 2007;61:45-53.

Delis DC, Kramer JH, Kaplan E, Ober BA. California Verbal Learning Test: Adult Version Manual. San Antonio: The Psychological Corporation 1987.

Dittrich WH, Johansen T, Fineberg NA. Cognitive Assessment Instrument of Obsessions and Compulsions (CAIOC-13) – a new 13-item scale for evaluating functional impairment associated with OCD. Psychiatry Research 2011a;187:283-290.

Dittrich WH, Johansen T, Landrø NI, Fineberg NA. Cognitive performance and specific deficits in OCD symptom dimensions: III. Decision-making and impairments in risky choices. German Journal of Psychiatry 2011b;14:13-25.

Dittrich WH, Johansen T, Landrø NI, Fineberg NA, Goder YM. Cognitive performance and specific deficits in OCD symptom dimensions: I. Olfactory perception and impaired recognition of disgust. German Journal of Psychiatry 2010a;13:127-139.

Dittrich WH, Johansen T, Padhi AK, Smith IE, Chamberlain SR, Fineberg NA. Clinical and neurocognitive changes with modafinil in obsessive–compulsive disorder: a case report. Psychopharmacology 2010b;212:449-451.

Ecker W, Gönner S. Incompleteness and harm avoidance in OCD symptom dimensions. Behaviour Research and Therapy 2008;46:895-904.

Fineberg NA, Sharma P, Sivakumaran T, Sahakian BJ, Chamberlain SR. Does obsessive-compulsive personality disorder belong within the obsessive-compulsive spectrum? CNS Spectrum 2007;12:467-482.

Goldman-Rakic PS. Working memory and the mind. Scientific American 1992;9:111-117.

Goodman WK, Price LH, Rasmussen SA, Mazure C, Fleischmann RL, Hill CL, Heninger GR, Charney DS. The Yale-Brown Obsessive Compulsive Scale I: development, use, and reliability. Archives of General Psychiatry 1989;46:1006-1011.

Henderson L, Dittrich WH. Decomposing the corpus of neuropsychological tests. Psycoloquy 1993;4:Frontal Cortex (3).

Hodgson TL, Dittrich WH, Henderson L, Kennard C. Eye movements and spatial working memory in Parkinson's disease. Neuropsychologia 1999;37:927-938.

Lacerda ALT, Dalgalarrondo P, Caetano D, Haas GL, Camargo EE, Keshavan MS. Neuropsychological performance and regional cerebral blood flow in obsessive–compulsive disorder. Progress in Neuro-Psychopharmacology & Biological Psychiatry 2003;27:657-665.

Leckman JF, Walker DE, Cohen DJ. Premonitory urges in Tourette's syndrome. American Journal of Psychiatry 1993;150:98-102.

Leckman JF, Walker DE, Goodman WK, Pauls DL, Cohen DJ. "Just-Right" perceptions associated with compulsive behaviors in Tourettes's syndrome. American Journal of Psychiatry 1994;51:675-680.

Martin A, Wiggs CL, Altemus M, Rubenstein C, Murphy DL. Working memory as assessed by subject-ordered tasks in patients with obsessive–compulsive disorder. Journal of Clinical and Experimental Neuropsychology 1995;17:786-792.

Mataix-Cols D, Junque C, Sanchez-Turet M, Vallejo J, Verger K, Barrios M. Neuropsychological functioning in a subclinical obsessive–compulsive sample. Biological Psychiatry 1999;45:898-904.

Mataix-Cols D, Rosario-Campos MC, Leckman JF. A multidimensional model of obsessive- compulsive disorder. American Journal of Psychiatry 2005;162:228-238.

Mataix-Cols D, Wooderson S, Lawrence N, Brammer MJ, Speckens A, Phillips ML. Distinct neural correlates of washing, checking, and hoarding symptom dimensions in obsessive–compulsive disorder. Archives of General Psychiatry 2004;61:564-576.

McKay D, Abramowitz JS, Calamari JE, Kyrios M, Radomsky A, Sookman D, Taylor S, Wilhelm S. A critical evaluation of obsessive–compulsive disorder subtypes: symptoms versus mechanisms. Clinical Psychology Review 2004;24:283-313.

Montgomery SA, Åsberg M. A new depression scale designed to be sensitive to change. The British Journal of Psychiatry 1979;134:382-389.

Moritz S, Kloss M, Jahn H, Schick M, Hand I. Impact of comorbid depressive symptoms on nonverbal memory and visuospatial performance in obsessive–compulsive disorder. Cognitive Neuropsychiatry 2003;8:261-272.

Muller J, Roberts JE. Memory and attention in obsessive–compulsive disorder: a review. Journal of Anxiety Disorders 2005;19:1-28.

Nedeljkovic M, Kyrios M, Moulding R, Doron G, Wainwright K, Pantelis C, Purcell R, & Maruff P. Differences in neuropsychological performance between subtypes of obsessive–compulsive disorder. Australian and New Zealand Journal of Psychiatry 2009;43:216-226.

Nelson HE. National Adult Reading Test (NART): Test Manual. Windsor, England: NFER – Nelson 1982.

Nielen MMA, den Boer JA. Neuropsychological performance of OCD patients before and after treatment with fluoxetine: evidence for persistent cognitive deficits. Psychological Medicine 2003;33:917-925.

Osterrieth PA. Le test du copie d'une figure complex: Contribution à l'étude de la perception et de la memoire (The test of copying a complex figure: a contribution to the study of perception and memory). Archives of Psychology 1944;30:286-350.

Owen AM, Doyon J, Petrides M, Evans AC. Planning and spatial working memory: a positron emission tomography study in humans. European Journal of Neuroscience 1996;8: 353-364.

Owen AM, Downes JD, Sahakian BJ, Polkey CE, Robbins T. Planning and spatial working memory following frontal lobe lesions in man. Neuropsychologia 1990;28:1021-1034.

Purcell R, Maruff P, Kyrios M, Pantelis C. Cognitive deficits in obsessive compulsive disorder on tests of forntal-striatal function. Biological Psychiatry 1998a;43:348-357.

Purcell R, Maruff P, Kyrios M, Pantelis C. Neuropsychological deficits in obsessive compulsive disorder: a comparison with unipolar depression, panic disorder and normal controls. Archives of General Psychiatry 1998b;55:415-423.

Rabbitt P. Introduction: methodologies and models in the study of executive functions. In P. Rabbitt (Ed.), Methodology of Frontal and Executive Function (pp. 1-37). Hove: Psychology Press 1997.

Radomsky AS, Rachman S, Hammond D. Memory bias, confidence and responsibility in compulsive checking. Behaviour Research and Therapy 2001;39:813-822.

Rotter JB. Generalized expectancies for internal versus external control of reinforcement. Psychological Monographs: General and Applied 1966;80:1-28.

Rosario-Campos MC, Miguel EC, Quatrano S, Chacon P, Ferrao Y, Findley D, Katsovich L, Scahill L, King RA, Woody SR, Tolin D, Hollander E, Kano Y, Leckman JF. The Dimensional Yale-Brown Obsessive-Compulsive Scale (DY-BOCS): an instrument for assessing obsessive-compulsive symptom dimensions. Molecular Psychiatry 2006;11:495-504.

Rubies P, Fineberg NA, Simpson J, Dittrich WH. Deficits in visual memory and executive function in patients with obsessive compulsive disorders. Journal of Psychopharmacology 2001;14(Suppl.3):A15.

Sahakian BJ, Morris RG, Evenden JL, Heald A, Levy R, Philpot M, Robbins TW. A comparative study of visuospatial memory and learning in Alzheimer-type dementia and Parkinson's disease. Brain 1988;111:695-718.

Savage CR, Baer L, Keuthen NJ, Brown HD, Rauch SL, Jenike MA. Organizational strategies mediate nonverbal memory impairment in obsessive–compulsive disorder. Biological Psychiatry 1999;45:905-916.

Savage CR, Deckersbach T, Wilhelm S, Rauch SL, Baer L, Reid T, Jenike MA. Strategic processing and episodic memory impairment in obsessive–compulsive disorder. Neuropsychology 2000;14:141-151.

Sheehan DV, Harnett-Sheehan K, Raj BA. The measurement of disability. International Clinical Psychopharmacology 1996;11(Suppl.3):89-95.

Sheehan DV, Lecrubier Y, Harnett-Sheehan K, Amorim P, Janavs J, Weiller E, Hergueta T, Baker R, Dunbar GC. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. Journal of Clinical Psychiatry 1998;59(Suppl.20):22-33.

Sher KJ, Frost RO, Otto R. Cognitive deficits in compulsive checkers: an exploratory study. Behaviour Research and Therapy 1983;21:357-363.

Simpson HB, Rosen W, Huppert JD, Lin S-H, Foa EB, Liebowitz MR. Are there reliable neuropsychological deficits in obsessive–compulsive disorder? Journal of Psychiatric Research 2006;40:247-257.

Spielberger CD, Gorsuch RL, Lushene R, Vagg PR, Jacobs GA. Manual for the state-trait anxiety inventory. Palo Alto, CA: Consulting Psychologists Press 1983.

SPSS version 16.0. Statistical Package for the Social Sciences (www.spss.com). Chicago, IL: SPSS Inc. 2008.

Summerfeldt LJ, Hood K, Antony MM, Richter MA, Swinson RP. Impulsivity in obsessive-compulsive disorder: comparisons with other anxiety disorders and within tic-related subgroups. Personality and Individual Differences 2004a;36:539-553.

Summerfeldt LJ, Kloosterman PH, Antony MM, Richter MA, Swinson RM. The relationship between miscellaneous symptoms and major symptom factors in obsessive-compulsive disorder. Behaviour Research and Therapy 2004b;42:1453-1467.

van den Hout MA, Kindt M. Repeated checking causes memory distrust. Behaviour Research and Therapy 2003;41:301-316.

Watkins L, Sahakian BJ, Robertson MM, Veale DM, Rogers RD, Pickard KM, Aitken MRF, Robbins TW. Executive function in Tourette's syndrome and obsessive-compulsive disorder. Psychological Medicine 2005;35:571-582.