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Executive functioning in obsessive–compulsive disorder, unipolar depression, and schizophrenia

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Abstract

The present study investigated whether schizophrenic, unipolar depressive, and obsessive–compulsive psychiatric patients show a distinguishable profile in tasks considered sensitive to frontal lobe functioning. Three psychiatric samples, each comprising 25 patients with little symptomatic overlap, were compared to 70 healthy controls. Participants completed several executive tasks (Wisconsin Card Sorting Test (WCST), verbal fluency, digit span, Stroop, and Trail-Making). Except for age, which was entered as a covariate, subjects did not differ in any sociodemographic background variable. Healthy controls showed superior performance relative to depressive and schizophrenic patients who exhibited comparable deficits in all tasks. Obsessive–compulsive disorder (OCD) patients revealed dysfunctions in the Trail-Making Tests A and B and in the fluency task. Dysfunctions in the domains of working memory, verbal fluency, distractibility, and concept formation were not confined to a specific psychiatric population. © 2002 National Academy of Neuropsychology. Published by Elsevier Science Ltd.

Keywords: Schizophrenia; Depression; Obsessive–compulsive disorder; Frontal lobe; Wisconsin Card Sorting Test; Verbal fluency

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1. Introduction

Within the last decade, evidence has accumulated that frontal lobe dysfunctions play a crucial role in the genesis of schizophrenia (Liddle, 1994), depression (Rogers, Bradshaw, Pantelis, & Phillips, 1998), and obsessive–compulsive disorder (OCD) (Purcell, Maruff, Kyrios, & Pantelis, 1998). Studies conducted with biological high-risk populations and healthy subjects scoring high on measures of OCD, schizotypy (considered to be a mild variant of schizophrenia), and depression suggest that neuropsychological dysfunctions are a risk factor for and not a mere consequence of these psychiatric disorders. However, the causative role of *circumscribed* prefrontal dysfunctions for the genesis of *specific* psychiatric disorders has not yet been convincingly established.

Neuropsychological studies with *depressive* and *schizophrenic* samples have revealed that dysfunctions in the anterior cingulate cortex (as indexed with the Stroop task; Peterson et al., 1999) and the dorsolateral cortex (as assessed with the Wisconsin Card Sorting Test (WCST); Weinberger, Berman, & Zec, 1986) are evident in both disorders compared to healthy controls (Franke et al., 1993; Moritz, 2000). However, most investigations conducted in the past have merely contrasted performance of a single psychiatric population with that of healthy controls without additionally investigating another psychiatric sample. Therefore, it has not yet been established whether executive deficits are equally pronounced in both disorders.

For *OCD patients*, a rather contradictory picture emerges regarding executive functioning (for reviews, see Cox, 1997; Tallis, 1997). Previous investigations have revealed mixed findings for various executive tasks. Whereas Alarcón, Libb, and Boll (1994) infer from previous research that OCD is associated with frontal impairments, in a more recent review Cox (1997) speculates that comorbid psychotic and depressive symptoms may have induced neuropsychological deficits, which were misattributed to OCD psychopathology. This hypothesis has been confirmed in a recent study: It was found that OCD patients exhibiting elevated depressive scores revealed cognitive deficits, whereas OCD patients with low depressive scores could not be distinguished from controls regarding executive functioning (Moritz et al., in press).

To analyze the executive performance profile of psychiatric patients, an exploratory study was performed with 25 subjects either suffering from OCD, schizophrenia, or unipolar depression. A total of 70 healthy subjects served as controls. It was hypothesized that executive deficits are not confined to any specific psychiatric group.

2. Methods

2.1. Subjects

All psychiatric subjects were inpatients at the psychiatric university hospital of Hamburg who were tested shortly after admission following an acute psychiatric episode. All patients gave informed consent to participate. Subjects with a current or previous history of alcohol or drug abuse, significant neurological disturbances (e.g. stroke), or a schizoaffective disorder

were not enrolled in the study. DSM-IV diagnoses were confirmed in extensive clinical interviews. Further, previous medical records were carefully screened. To minimize symptomatic overlap schizophrenic patients were excluded if their depression score as assessed with the Positive and Negative Syndrome Scale exceeded four points (i.e. moderate severity; 13 patients were excluded). No schizophrenic subject showed any obsessive–compulsive symptoms. Depressive subjects with obsessive–compulsive symptoms ($n=2$) and previous psychotic symptoms ($n=2$; the corresponding Hamilton Depression (HAM-D) scores must be zero) were not included in the present study. Patients suffering from OCD were excluded if their HAM-D score exceeded 17 points (17-item version, 11 patients were excluded). Besides, OCD subjects with psychotic symptoms or a previous diagnosis of major depression (even if the current HAM-D score was below the cut-off score) were excluded ($n=4$). Besides, depressive and OCD subjects were explicitly asked whether they had ever experienced hallucinations or delusions. The schizophrenic patients were treated with atypical neuroleptics for 2 weeks after a short wash-out phase. Most of the OCD ($n=14$) and depressive patients ($n=20$) were medicated with antidepressant agents. Benzodiazepines were administered in four schizophrenic, eight depressive, and two OCD patients.

Data was collected until 25 patients of each psychiatric group fulfilled the inclusion criteria. Finally, a healthy control group was recruited consisting of 70 subjects. The main sociodemographic characteristics are displayed in Table 1.

2.2. Instruments

Subjects were administered the following tasks thought to represent distinct aspects of executive functioning.

(a) WCST: A computerized version (Loong, 1990) was employed, which followed the procedure by Heaton (1981), i.e. category changes are not announced. None of the patients had any problems in using the computer keys.

(b) Stroop task: A German version of the Stroop task (Moritz, Mass, & Junk, 1998; Stroop, 1935) was administered, the Stroop effect was computed as the reaction time difference in the experimental condition (naming of the ink colour of incongruently coloured colour words) relative to a control condition (undistracted colour naming).

(c) Trail-Making Tests A and B (adult version): In Part A, the subject is required to quickly combine encircled numbers and in Part B to alternately combine numbers and characters in ascending order (Reitan, 1992).

(d) Digit span: In the first subtest, the participant has to repeat strings of numbers in the order presented by the experimenter (short-term memory). In the second subtest, the subject is instructed to repeat the presented numbers in backward order (working memory).

(e) (Creative) verbal fluency: The subject has 4 min to write down as many alternate uses he/she can think of for a can and a piece of string (e.g. to use a can as a football; Schoppe, 1975). Common or impossible uses (i.e. to make a knot with a piece of string or to use a can as a cushion) were not scored.

Clinical ratings were performed blind to neurocognitive results. The experimenter in the neuropsychological tests was never identical with the interviewer for the clinical assessment.

Table 1
Sociodemographic, psychopathological and neuropsychological characteristics of the samples

Variable	Healthy Ss ($n=70$)	Schizophrenic Ss ($n=25$)	Depressive Ss ($n=25$)	OCD Ss ($n=25$)	Statistics[a]
Background variables					
Male/female	40/30	14/11	11/14	11/14	$\chi^2(3)=2.18$; $P>.5$
Age (years)	33.1 (9.4)	34.7 (11.3)	41.0 (10.1)	31.6 (9.3)	$F(3,144)=4.71$; $P\leq.005$ O,H < D
Years of school (years)	11.6 (1.6)	11.6 (1.5)	11.0 (1.8)	11.4 (1.8)	$F(3,144)=0.84$; $P>.4$, NS
Length of illness	–	6.7 (7.5)	5.6 (7.0)	9.0 (7.8)	$F(2,70)=1.25$; $P>.2$, NS
PANSS global	–	62.5 (22.5)	–	–	–
Y-BOCS global	–	–	–	24.1 (5.5)	–
HAM-D global (17 items)	–	–	16.4 (5.2)	9.0 (4.0)	$t(48)=5.56$; $P\leq.001$
Verbal intelligence[b]	30.8 (2.2)	29.9 (3.8)	31.1 (4.0)	29.9 (3.6)	$F(3,143)=0.93$; $P>.4$, NS
Neuropsychological tasks					
TMT A (s)	25.7 (8.9)	38.2 (15.8)	37.3 (17.4)	38.0 (13.8)	$F(3,144)=10.80$; $P\leq.0001$ H < D,O,S [H < D,O,S]
TMT B (s)	56.8 (16.5)	95.9 (38.8)	96.3 (45.1)	83.8 (30.9)	$F(3,144)=17.57$; $P\leq.0001$ H < O,S,D [H < O,S,D]
TMT B – A	31.1 (14.4)	57.7 (37.0)	58.9 (35.2)	45.8 (26.2)	$F(3,144)=11.03$; $P\leq.0001$ H < O,S,D [H < S,D]
Stroop (s)	36.6 (15.3)	68.8 (36.3)	55.1 (31.8)	31.7 (23.1)	$F(3,142)=14.18$; $P\leq.0001$ O,H<D,S [O,H<D,S]
WCST categories	5.2 (1.6)	3.4 (2.4)	3.7 (2.7)	4.3 (1.6)	$F(3,144)=6.59$; $P\leq.0005$ H>S,D [H>S,D]
WCST perseveration	13.5 (8.6)	25.7 (18.7)	22.6 (18.2)	17.8 (10.1)	$F(3,144)=6.70$; $P\leq.0005$ H < D,S [H < D,S]
Digit span forward	8.5 (1.8)	7.5 (1.9)	7.6 (2.1)	8.1 (2.4)	$F(3,144)=2.37$; $P\leq.1$ NS [NS]
Digit span backward	7.8 (2.3)	6.1 (2.1)	6.4 (2.1)	8.1 (2.5)	$F(3,144)=6.14$; $P\leq.001$ O,H>D,S [O,H>S; O>D]
Verbal fluency	17.1 (6.1)	12.5 (5.1)	10.4 (5.2)	10.9 (5.2)	$F(3,141)=13.30$; $P\leq.0001$ H>S,O,D [H>S,O,D]

Abbreviations: H = healthy controls, S = schizophrenia, O = obsessive-compulsive disorder, D = depression HAM-D = Hamilton Depression Scale (17 items); PANSS = Positive and Negative Syndrome Scale; TMT = Trail-Making Test, WCST = Wisconsin Card Sorting Test; Y-BOCS = Yale–Brown Obsessive Compulsive Scale.

[a] Post hoc comparisons were conducted using Tukey's HSD before and after controlling for age (the latter analyses are displayed in square brackets); the significance level was set at $P\leq.05$.

[b] Mehrfachwahl-Wortschatztest (Lehrl, 1995) A score of 27 is equivalent to a verbal IQ of 100.

3. Results

Subjects did not differ regarding verbal intelligence and major sociodemographic parameters except for age. Length of illness was comparable among the psychiatric samples and did not correlate with any of the neurocognitive tasks ($P>.1$; correlations were performed separately for all groups).

Overall group differences were computed using an analysis of variance. In Table 1, post hoc group differences (Tukey's HSD) are displayed before and after entering age as a covariate (ANCOVA).

Schizophrenic subjects and depressive subjects did not differ in any neuropsychological parameter (regardless whether a correction for multiple comparisons was conducted or not). Healthy controls performed superior compared to schizophrenic and depressive subjects in all tests except for digit span forward (for digit span backward, depressives differed at trend level from controls ($P \leq .1$) when age was controlled for).

The OCD sample showed a more heterogeneous profile. While OCD patients performed normal in the WCST, the digit span, the Stroop task, and the Trail-Making Test difference score (after controlling for age), deficits occurred for the creative verbal fluency task and the Trail-Making Tests A and B. In the digit span backward and the Stroop task, OCD patients performed superior to schizophrenic and depressive patients.

Core clinical ratings were not significantly correlated with task performance (schizophrenia, PANSS: $|r|=.08-.35$; $P>.1$; OCD, Yale–Brown Obsessive–Compulsive Scale: $|r|=.03-.24$; $P>.3$; depression, HAM-D: $|r|=.07-.32$; $P>.1$). A significant correlation emerged between verbal fluency and HAM-D for the OCD sample ($r=-.42$; $P \leq .05$).

When patients medicated benzodiazepines were compared to nonreceivers, no significant difference occurred for any test even before correction for multiple comparisons. The effects of antidepressant medication could only be tested in OCD patients since most depressive patients were medicated with antidepressants. Again, no significant difference occurred even before correction for multiple comparisons.

4. Discussion

The present results obtained in samples of psychiatric patients with minimal psychopathological overlap suggest that depressive and schizophrenic patients do not exhibit a distinct executive performance profile. In line with several studies (e.g. Liddle, 1994; Rogers et al., 1998), both samples revealed disturbances in domains considered sensitive for the anterior cingulate (Stroop task) and the dorsolateral prefrontal cortex (WCST). Replicating previous findings that nondepressed patients with OCD have no dysfunctions in concept formation and verbal (working) memory (see Cox, 1997; Tallis, 1997), OCD patients performed normal on the WCST, the digit span, the Stroop task, and the Trail-Making Test differences score. Unexpected from own previous research (Moritz et al., in press), deficits were evident for a task tapping verbal fluency and the Trail-Making B. However, in a previous study, the OCD sample exhibited less depressive symptoms. As the HAM-D score in the current study was

highly correlated with verbal fluency performance ($r = -.42$; $P \leq .05$) and substantially correlated with slowing in the Trail-Making B ($r = .29$; NS), these dysfunctions may be attributed to residual depressive symptoms rather than to OCD symptomatology. Finally, it should be noted that we could not replicate a previous finding demonstrating enhanced Stroop interference in OCD patients (Moritz, Kloss, Katenkamp, Birkner, & Hand, 1999), which indicates normal anterior cingulate function.

The present findings suggest that causal neuropsychological models of schizophrenia, OCD, and unipolar depression, which in part rely on data obtained with the present tasks, need careful reconsideration. Deficits in verbal working memory (digit span backward), interference/distractibility (Stroop task), concept formation/problem solving (WCST), verbal fluency (creativity task), and set-shifting (Trail-Making Test), which all have been discussed as being sensitive to frontal functioning are not confined to any of the investigated psychiatric populations. Therefore, these deficits seem to represent a more general rather than a special vulnerability to psychiatric illness. Future studies should employ more fine-grained tasks adopted from cognitive psychology since the tasks administered in the present study were rather complex and presumably did not only tap deficits in the frontal lobes. In addition, greater attention should be paid to the heterogeneity of the psychiatric disorders. Factor analytic studies correlating task performance with psychopathological factor scores or studies dichotomizing samples according to the presence or absence of cardinal psychiatric symptoms may shed light on specific cognitive disturbances.

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