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Neurocognitive performance in first-episode and chronic schizophrenic patients

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Abstract Previous research on neuropsychological disturbances in first-episode and chronic schizophrenic patients has provided mixed results which can be partially attributed to methodological inconsistencies. For the present study, 70 schizophrenic patients (40 with chronic and 30 with first-episode schizophrenia) were compared to 30 healthy controls on a large battery of neuropsychological tests. Special attention was paid to potential confounds such as differences in psychopathology, age and educational level between the schizophrenic sub-samples.

Healthy controls performed better than both first-episode and chronic patients in almost all cognitive domains ($P < 0.01$), while the patient samples did not differ in any of the tasks. Results were confirmed in a second series of analyses in which patient subgroups were equated for sociodemographic background variables. The present results confirm recent data collected in longitudinal studies, thus, lending further support for a neurodevelopmental model of schizophrenia. It is suggested that neuropsychological disturbances occur early in schizophrenia and do not worsen in the course beyond age-related decrement. Possible reasons why previous research has produced contradictory findings are discussed.

Key words Schizophrenia · neurocognition · neurodegeneration · neurodevelopment · neurocognitive deterioration

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Introduction

The concept of "dementia praecox" advocated by Kraepelin (1893) in the late 19th century has fueled an ongoing debate whether or not cognitive decline is an inevitable characteristic of schizophrenia (Rund 1998, Russell et al. 1997). Shedding light on this question is essential from the perspective of both pathogenetic research and sociotherapy/psychotherapy, since the prospect of progressive cognitive deterioration would necessitate a major re-definition of life plans for individuals experiencing their first episode.

Contradictory findings in the literature (Albus et al. 1996, Bilder et al. 1992, Gold et al. 1998, Hoff et al. 1999, Sobizack et al. 1999), which have been interpreted either in favor of a neurodegenerative or a neurodevelopmental model of schizophrenia, can be partially explained by methodological problems inherent in both longitudinal (see Rund 1998 for a review) and cross-sectional approaches. A major disadvantage of cross-sectional studies (i. e., a first-episode sample is compared with a chronic schizophrenic sample) is that differences in neurocognitive functioning may stem from incomplete sample matching. For example, chronic schizophrenic subjects in a study conducted by Bilder et al. (1992) had a significantly lower educational level than first-episode patients. As education is substantially correlated with most neurocognitive functions, differences between samples do not necessarily imply greater neurocognitive dysfunction in chronic schizophrenia indicative of cognitive deterioration. In another study published by Albus et al. (1996; see also Sobizack et al. 1999) chronic schizophrenic patients exhibited significantly higher BPRS scores than first-episode patients (negative symptomatology; scores for thought disturbance). An extensive literature (e. g., Liddle and Morris 1991, Moritz et al. 2001, Moritz et al. 2001 a) shows that disorganized and negative symptoms are correlated with deficits in memory, attention and executive functioning. Therefore, it cannot be ruled out that the re-

ported sample differences may reflect a mere by-product of illness severity. Equation for such potential moderators (including neuroleptic treatment) is essential in cross-sectional research in order to draw valid conclusions.

It has to be emphasized that cross-sectional studies can only serve as an indirect, confirmatory approach to address the question whether or not neurocognitive functioning declines in the course of the schizophrenic illness: only a longitudinal approach is able to *directly* shed light on this issue. A major short-coming of prospective follow-up studies, however, is that the time interval between initial assessment and re-test is often very short so that slow cognitive decline over time may remain undetected. Moreover, practice effects, lower psychopathology scores at follow-up, drop-out at re-test, and changes in medication regime are hard to control for in longitudinal studies and may mask cognitive decline (see Rund 1998, for a discussion). Therefore, given the shortcomings of both approaches, longitudinal and cross-sectional studies should be regarded as complementary methods.

The present study aims to confirm a previous finding collected in a different schizophrenic sample (Moritz et al. 2001 b). In that study, a self-report measure of cognitive functioning (Frankfurt Complaint Questionnaire) was administered in order to assess subjective cognitive complaints in everyday life. Chronic and first-episode patients were indistinguishable regarding subjective cognitive deficits, whereas healthy controls displayed significantly less mnestic, attentional and perceptual problems. In addition, samples did not differ on a lie scale assessing the tendency to conceal common somatic and cognitive dysfunctions.

In the present study, patient groups did not differ regarding psychopathology, neuroleptic dosage, gender distribution or education. Further, special care was taken regarding task selection: tests represented all major areas of cognitive functioning and included the digit-symbol test, which in the study conducted by Bilder et al. (1992) discriminated best first-episode from chronic patients.

Methods

Subjects

Seventy schizophrenic inpatients who were admitted consecutively to a psychiatric hospital following an acute episode participated in the study. Patients were categorized as chronic if first hospitalization was three or more years ago (n = 40). The first-episode patient group comprised subjects whose illness onset was, at most, half a year ago (n = 30). Mean length of illness in the chronic group was approximately 11 years. All available information, including medical records, patient interviews and interviews with relatives, was screened to ensure correct group assignment. The total sample consisted of 105 patients, including patients who withdrew consent and patients with unclear diagnoses or extensive missing data. The first-episode sample included patients who met DSM-IV diagnostic criteria for schizophreniform or schizophrenic disorder. These patients were usually followed-up six months later to confirm diagnoses. The chronic sample consisted solely of patients with a clear diagnosis of schizophrenia according to DSM-IV diagnostic criteria. Substance abuse or significant neurological disturbances not commonly reported in schizophrenia led to subject exclusion.

Neurocognitive testing was carried out two weeks after admission. At the same time, psychopathological ratings (PANSS; Kay et al. 1989) were administered by clinicians blind to neurocognitive status. The PANSS rating relied on a semi-structured interview with raters being trained for objectivity. Patients were on atypical neuroleptics (either clozapine, risperidone, sertindole, olanzapine, zotepine or amisulpride) for approximately two weeks following a wash-out period of at least three days. Patients were presently not taking any other concomitant anticholinergic, antidepressant or neuroleptic agents.

Diagnoses were confirmed using a semi-structured interview. Thirty healthy controls were recruited from different sources including hospital staff and members of the German army forces. Controls were screened for possible psychiatric disorders. Written informed consent was obtained from all subjects after the procedures had been fully explained.

Neurocognitive assessment

Executive functioning:

Wisconsin Card Sorting Test (WCST). A computerized WCST version was applied according to the standard procedure (Heaton 1981). Categories completed (0–6) and perseverative errors served as dependent variables.

Trail-Making Test. The standard procedure (Reitan 1992) was administered. Errors were corrected by the experimenter. Reaction times to complete both subtasks (A and B) served as dependent variables. Further, a difference score was computed as follows: reaction time Trail B minus reaction time Trail A.

Verbal fluency (LPS subtask 6; Horn 1962). The subject was instructed to write down as many words as possible starting with the letters F, K, R (1 minute per letter) and S (20 seconds). The number of correct words was taken as the dependent variable (if words were misspelled but semantically correct they were judged as correct, e. g., *ret* instead of *red*).

Creative verbal fluency (Schoppe 1975). The subject had 4 minutes to write down as many alternate uses as possible for an empty can and a piece of string (2 minutes per object). The number of non-trivial responses served as the dependent variable.

Memory

Digit span (taken from the German version of the WAIS-R, Tewes 1991). The standard procedure for administering WAIS-R was applied. Scores for digit span forward (short-term memory) and digit span backward (working memory) are reported independently.

Rey-Auditory Verbal Learning task (RAVLT, see Lezak 1995). A list consisting of 15 words (list A) was read to the subject five times. After each presentation the subject had to recall as many words as possible in any order. After that, a second list (list B) was introduced which had to be repeated directly thereafter. Then, list A had to be repeated immediately (trial 6) and 20 minutes later (trial 7) without prior presentation. Recall of list A after trial 1, 5 and 7 served as dependent variables.

Visuoperception

Mental rotation (LPS subtask 9; Horn 1962). The subject had to decide of how many sides a perceptually drawn body consisted (including those sides which were hidden due to the two-dimensional display). The subject had three minutes to solve as many items as possible.

Selective attention/concentration

Digit-symbol test (Tewes 1991). The test followed the WAIS-R standard procedure.

Stroop-task (Stroop 1935). The interference (Stroop) score was computed as the reaction time difference between two lists consisting of 72 colored bars and two lists consisting of Stroop words (i. e., color words written in an incongruent ink color, e. g., RED written in blue ink). Time was taken after each list.

Unlike previous studies we did not collapse neuropsychological test scores into single values, as decreased composite scores may be caused by one extreme outlying result which cannot be detected when only omnibus information is presented. For example, Albus et al. (1996) report that the greatest difference between first-episode and chronic patients was detected for a function called "visual-motor processing and selective attention". This index incorporated scores from the Stroop task, the digit-symbol task and the Trail-Making Test. However, closer inspection reveals that chronic and first-episode patients performed almost identical in the digit-symbol test and Stroop task. Substantial differences were only evident for the Trail-Making Test.

Results

Differences regarding sociodemographic and psychopathological background variables

Sociodemographic, psychopathological and neuropsychological characteristics of the samples are displayed in Table 1. Samples did not differ regarding gender distribution, years of education or verbal intelligence as assessed with a vocabulary test (Mehrfachwahl-Wortschatztest, Lehrl 1995). The chronic sample, however, was significantly older than the first-episode sample. The schizophrenic subsamples were comparable regarding PANSS subscores and neuroleptic dosage.

Differences regarding neuropsychological variables

Schizophrenic samples did not differ in any of the neurocognitive parameters even before an alpha correction for testing multiple comparisons was applied. Healthy controls displayed significantly better scores in all neurocognitive areas relative to both subgroups except for the Stroop-difference score, where post-hoc differences achieved significance only for first-episode patients and healthy controls.

Except for digit symbol substitution ($r = 0.22$; $P = 0.06$), mental rotation ($r = 0.19$; NS) and Trail-Making Test A ($r = -0.08$; NS), all other neurocognitive parameters were significantly correlated with years of education ($r > 0.26$; $P \leq 0.01$). Negative symptomatology (conventional PANSS subscore) was correlated with greater dysfunction in the verbal creativity task ($r = -0.25$; $P = 0.05$), the Trail-Making Test B ($r = 0.23$; $P = 0.07$) and the Trail-Making Test difference score ($r = 0.26$; $P = 0.05$).

Table 1 Sociodemographic, psychopathological and neuropsychological characteristics of the samples (mean and standard deviations)

	Healthy controls (H)	First-episode patients (FE)	Chronic sample (C)	Statistics (post-hocs were conducted using the Bonferroni procedure)
age	32.30 (10.68)	28.60 (9.61)	35.78 (9.94)	F=4.37; P ≤ 0.02; FE < C
Male/female	20/10	21/9	21/19	$Chi^2 = 2.66$; P > 0.25
Years of education	11.90 (1.30)	11.41 (1.64)	11.40 (1.74)	F=1.10; P > 0.3
Admissions	–	–	4.38 (4.12)	–
Chlorpromazine equivalents in mg*	–	333.33 (198.58)	357.95 (224.85)	T=0.34; P > 0.5
Length of illness	–	0.13 (0.21)	10.71 (7.01)	T=9.54; P ≤ 0.001
PANSS-Positive	–	14.42 (5.67)	16.03 (6.93)	T=0.97; P > 0.3
PANSS-Negative	–	16.62 (7.67)	17.94 (8.18)	T=0.65; P > 0.5
PANSS-Global	–	29.46 (9.31)	32.03 (10.81)	T=0.98; P > 0.3
PANSS-Total	–	60.50 (20.87)	66.00 (23.45)	T=0.95; P > 0.3
Vocabulary (MWT-B)	31.03 (3.50)	28.41 (4.90)	29.24 (5.82)	F=2.21; P > 0.1
TMT-A (s)	27.34 (10.14)	39.24 (14.46)	43.23 (21.17)	F=8.22; P ≤ 0.0005; H < FE/C
TMT-B (s)	56.95 (16.80)	102.13 (62.72)	106.35 (62.65)	F=8.43; P ≤ 0.0005; H < FE/C
TMT B-A (s)	29.61 (15.18)	62.90 (55.37)	63.12 (49.79)	F=5.91: P ≤ 0.005; H < FE/C
Verbal creativity	17.70 (5.95)	12.79 (5.19)	11.18 (5.68)	F=11.72; P ≤ 0.0001; H > FE/C
WCST-CC	5.30 (1.44)	3.37 (2.10)	3.14 (2.26)	F=10.19; P ≤ 0.0001; H > FE/C
WCST-PE	12.15 (8.21)	27.17 (13.87)	25.54 (17.16)	F=9.65; P ≤ 0.0005; H < C/FE
Verbal fluency (LPS)	44.77 (11.19)	35.21 (11.80)	33.80 (11.69)	F=8.52; P ≤ 0.0005; H > FE/C
Mental rotation (LPS)	26.00 (7.83)	20.16 (5.43)	20.71 (7.89)	F=5.10; P ≤ 0.01; H > C/FE
RAVLT trial 1	8.67 (1.75)	6.31 (2.02)	5.95 (2.22)	F=16.99; P ≤ 0.0001; H > FE/C
RAVLT trial 5	13.50 (1.55)	11.27 (2.35)	10.56 (2.81)	F=13.89; P ≤ 0.0001; H > FE/C
RAVLT trial 7	12.47 (2.40)	8.21 (4.25)	7.82 (3.25)	F=18.50; P ≤ 0.0001; H > FE/C
Stroop (s)	41.14 (14.96)	74.10 (37.42)	63.91 (52.00)	F=5.22; P ≤ 0.01; H < FE
Digit span forward	8.90 (1.77)	6.73 (2.15)	7.05 (2.12)	F=10.24; P ≤ 0.0001; H > C/FE
Digit span backward	8.03 (2.28)	5.53 (2.03)	6.00 (2.36)	F=10.77; P ≤ 0.0001; H > C/FE
Digit-symbol	59.43 (7.30)	42.60 (12.03)	39.37 (11.80)	F=29.74; P ≤ 0.0001; H > FE/C

RAVLT trial number of the Rev-Auditory Verbal Learning Test; *MWT-B* Mehrfachwahl-Wortschatztest; *PANSS* Positive and Negative Syndrome Scale; *TMT* Trail-Making Test; *WCST* Wisconsin Card Sorting Test Categories Completed (CC) and number of perseverative errors (PE); *s* seconds
* determined according to Dietmaier & Laux (1998)

Group comparisons with schizophrenic groups equated for background variables

As the chronic group was significantly older than the first-episode patients and contained considerably more women, samples were equated in a second analysis according to age and gender. For this purpose, 10 women with the highest age were dropped from the chronic schizophrenic group blind to neurocognitive results [first-episode sample: sex: 21 male/9 female, age: 32.67 years (SD: 7.92), years of education: 11.47 (SD: 1.66), Chlorpromazine equivalent dosage: 334.37 mg (SD: 172.69), all comparisons with chronic patient and healthy controls were not significant]. Group comparisons revealed that healthy controls performed significantly better than both groups (including the Stroop task), while the schizophrenic subsamples were again indistinguishable regarding any neuropsychological test (before and after alpha correction). Application of a more rigid criterion of chronicity (at least three psychotic episodes) again did not reveal any significant performance differences.

Discussion

The present study confirms previous research (e. g., Moritz et al. 2001 b) indicating that cognitive dysfunction is already present at the beginning of the schizophrenic disorder. Both chronic and first-episode schizophrenic patients performed 1–2 standard deviations lower than controls in most parameters. However, no patient displayed any dysfunctions suggestive of dementia. Therefore, whereas the "dementia praecox" concept is partly confirmed, as cognitive dysfunctions indeed appear early in the course of schizophrenia, the present data together with previous longitudinal and cross-sectional research suggest that Kraepelin's concept (1893) largely exaggerates the degree of cognitive dysfunctions in schizophrenia. In line with this, Heaton et al. (1994) showed that schizophrenic patients perform superior to patients with Alzheimer dementia. Heaton et al. also report that neither length of illness nor age of onset did relate to cognitive dysfunction.

The present study did not reveal any differences between first-episode and chronic patients even before correction for multiple comparisons. Results are thus in accordance with recent longitudinal studies conducted by Gold et al. (1998), Hoff et al. (1999) and Russell et al. (1997) but differ somewhat from results obtained in recent cross-sectional studies (see Bilder et al. 1992, Sobizack et al. 1999, Saykin et al. 1994). Although Rund (1998) criticizes cross-sectional designs as being generally inadequate to address the question whether schizophrenia is a neurodevelopmental or neurodegenerative disorder, inconsistencies among studies following either approach call for an explanation. As previously mentioned, longitudinal studies may obscure possible progradient cognitive decline through re-test familiarity with tasks, short re-test interval and decline in psychopathology. Cross-sectional studies on the other hand may over-estimate cognitive dysfunctions in chronic patients when samples are not equated for variables containing a major impact on cognition (especially age). The present study found no group differences between first-episode and chronic patients, which is attributed to the fact that, unlike the study of Bilder et al. (1992) and Sobizack et al. (1999), samples were equated regarding various possible contributors of task differences such as psychopathology, neuroleptic dosage, gender and education. In line with this, education (not equated in Bilder et al. 1992) was significantly correlated with task performance and negative symptomatology (not equated in the Sobizack et al. study) was accompanied by greater neurocognitive dysfunction in several tasks. It is also important to note that we did not find any difference in the digit-symbol task between chronic and first-episode patients, whereas Bilder et al. (1992) reported the strongest group differences for this task.

Since the chronic group in our first analyses was significantly older than the first-episode sample and contained (insignificantly) more females (for the impact of gender effects see Lewine et al., 1997), we computed another series of ANOVAs in which both schizophrenic samples were equated for gender distribution, age, education and neuroleptic dosage. Again, both groups performed equally impaired on all neuropsychological tests. Further, a more rigid criterion for chronicity (at least three hospitalizations) did not discriminate groups.

Although not directly tested, we do not think that neuroleptic treatment has led to additional cognitive impairment in either schizophrenic subsample, since a recent literature review reveals that the application of atypical neuroleptics (the sole neuroleptic medication in the present study) does not worsen but rather ameliorates cognitive dysfunction in schizophrenia (Keefe et al. 1999).

Results support conclusions drawn by Gold et al. (1998) that clinicians should adopt an attitude of guarded optimism towards the cognitive prognosis of schizophrenia. A patient with sufficient cognitive abilities, good compliance and stress resistance should not be discouraged from beginning or continuing post-secondary education or from performing a responsible job since there is increasing empirical evidence that cognitive functions do not decline in schizophrenic patients beyond age-related decrement. With regard to the growing body of literature showing that cognitive deficits predict both worse symptomatic (Moritz et al. 2000) and functional outcome (Green 1996), more efforts are required to treat neuropsychological dysfunctions in those schizophrenic patients suffering from severe cognitive disturbances.

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