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Neuropsychological functioning predicts community outcomes in affective and non-affective psychoses: A 6-month follow-up

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ABSTRACT

Introduction: Neurocognitive dysfunction is a major symptom feature of schizophrenia and bipolar disorder. A prognostic relationship between cognition and community outcomes is well-documented in schizophrenia and increasingly recognized in bipolar disorder. However, specific associations among neurocognition, diagnosis, state symptomatology, and community functioning are unclear, and few studies have compared these relationships among patients with affective and non-affective psychoses in the same study. We examined neurocognitive, clinical, and community functioning in a cross-diagnostic sample of patients with psychotic disorders over a 6-month follow-up interval.
Method: Neurocognitive, clinical and community functioning were assessed in participants with schizophrenia ($n = 13$), schizoaffective disorder ($n = 17$), or bipolar disorder with psychosis ($n = 18$), and healthy controls ($n = 18$) at baseline and 6 months later.
Results: Neurocognitive functioning was impaired in all diagnostic groups and, despite reductions in primary symptoms, did not recover on most measures over the follow-up period. Neurocognitive impairment was not associated with diagnosis or clinical improvement. Several neurocognitive scores at baseline (but not diagnosis or clinical baseline or follow-up scores) predicted community functioning at follow-up.
Discussion: In one of the few studies to longitudinally examine neurocognition in association with clinical and outcomes variables in a cross diagnostic sample of psychotic disorders patients, neurocognitive deficits were pronounced across diagnoses and did not recover on most measures despite significant reductions in clinical symptoms. Baseline neurocognitive functioning was the only significant predictor of patients' community functioning six months later. Efforts to recognize and address cognitive deficits, an approach that has shown promise in schizophrenia, should be extended to all patients with psychosis.

1. Introduction

Cognitive dysfunction is a core feature of schizophrenia (SZ), schizoaffective disorder (SZA), and bipolar disorder (BD) (Heinrichs and Zakzanis, 1998; Murphy and Sahakian, 2001; Mesholam-Gately et al., 2009). Some cross-sectional comparisons indicate that cognitive deficits are qualitatively similar but quantitatively different across diagnostic categories (typically: SZ < BD, SZA) (Altshuler et al., 2004; Krabbendam et al., 2005; Reichenberg et al., 2008). However, others report no differences between groups (Balanza-Martinez et al., 2005; Lewandowski et al., 2011a; Simonsen et al., 2011).

Cognitive impairment in adults with SZ appears to be significant but relatively stable (e.g. Hoff et al., 2005), although some cross-sectional evidence suggests that neurocognition may worsen with illness progression (Pukrop et al., 2006). Neurocognitive deficits in BD seem to present early in illness and remain largely stable (Mora et al., 2012; Torrent et al., 2012). A meta-analysis of neurocognition in euthymic BD showed deficits in most domains with medium to large effect sizes (Mann-Wrobel et al., 2011). However, some findings suggest that impairments are associated with illness duration and disease course (Denicoff et al., 1999; Robinson and Ferrier, 2006). Few studies have compared neuropsychological functioning across diagnostic groups longitudinally. A study of older adults with SZ or BD found similar trajectories of neurocognitive decline but greater variability in patients with BD (Depp et al., 2007). A three-year follow-up comparing patients with SZ and BD found that groups did not differ on most neuropsychological measures (Balanza-Martinez et al., 2005).

Cognition is associated with poorer functional outcomes in SZ, and an increasing literature supports the same relationship in BD (e.g. Green, 2006; Bowie et al., 2010; Mora et al., 2012). A recent meta-analysis reported that the strength of associations between cognition and community functioning was similar between patients with SZ or BD (Depp et al., 2012).

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We examined neurocognitive, clinical, and community functioning in patients with SZ, SZA, or bipolar disorder with psychosis (BDP) over time. We have previously reported that at baseline all three patient groups exhibited significant and similar neurocognitive deficits (Lewandowski et al., 2011a). We hypothesized that a) all patient groups would exhibit deficits in neurocognition at follow-up, which would not differ by diagnosis, b) any observed cognitive change would not be associated with diagnosis or clinical state, and c) cognitive deficits at baseline, but not diagnosis or clinical symptoms, would predict community functioning at follow-up.

2. Method

2.1. Participants

Participants with SZ ($n = 13$), SZA ($n = 17$) or BDP ($n = 18$), and healthy controls ($n = 18$) ages 18–55 were recruited through the Schizophrenia and Bipolar Disorder Program at McLean Hospital. All procedures were approved by the McLean IRB. The present sample includes only subjects who returned for follow-up (56% of the initial sample). Non-completion was due primarily to an inability to re-contact subjects (39%). Four subjects (5%) declined to participate in the follow-up. The present subsample did not differ from the baseline sample on any demographic characteristics or clinical measures; however, fewer patients with SZ were inpatients at baseline (68% vs. 38%).

2.2. Materials

Clinical assessment included the Young Mania Rating Scale (YMRS), the Montgomery–Asberg Depression Rating Scale (MADRS), and the Positive and Negative Syndrome Scale (PANSS). Cognitive measures included the Brief Visuospatial Memory Test-Revised (BVMT), Hopkins Verbal Learning Test (HVLT), Trails A and B, Stroop Color and Word Test (Stroop), and Category Fluency. Community functioning was assessed using the Multnomah Community Ability Scale (MCAS), which measures daily living, social involvement and interest, and occupational/other meaningful activity. We administered an abbreviated version, eliminating items that assessed clinical symptoms (M3, M4, M17), substance abuse (M16), and intellectual functioning (M2) so as to measure community functioning in a way that was less directly associated with clinical and cognitive symptoms. The final version included 11 items scored 1–5 (5 indicating highest functioning) for a total of 55 points.

2.3. Procedures

Ascertainment, diagnostic and baseline procedures are described in detail elsewhere (Lewandowski et al., 2011a). Briefly, participants completed assessments at baseline and approximately 6 months later. Diagnosis was established using the SCID interview. All participants were prescribed psychiatric medications at the time of assessment. Chlorpromazine (CPZ) equivalents were calculated based on the recommendations of Baldessarini (2012).

2.4. Statistical approach

Subjects were compared on demographic, clinical, and cognitive variables using ANOVA (continuous variables) or Chi-Square (categorical variables). Effect sizes were calculated (Cohen's d) for cognitive variables. Neuropsychological variables were normed using published data and converted to standard scores. A neuropsychological composite score was calculated for each subject and change scores were calculated for neuropsychological and clinical variables. Linear regressions were conducted predicting neuropsychological change using diagnosis or clinical change as predictors, accounting for number of lifetime hospitalizations, inpatient vs. outpatient status at baseline, and CPZ equivalents. To examine community outcomes at follow-up, linear regressions were conducted predicting MCAS using baseline cognition and baseline or follow-up clinical variables as predictors after accounting for diagnosis and the above confounders.

Table 1
Demographic variables by diagnosis.

	SZ ($n = 13$)	SZA ($n = 17$)	BDP ($n = 18$)	HC ($n = 18$)	Test statistic
Age	42.2 (8.9)	40.0 (8.4)	34.9 (12.8)	41.5 (8.5)	2.08*
Education[a]	4.0 (1.2)	4.6 (1.7)	4.7 (1.0)	6.3 (1.4)	1.01***
% Caucasian	69%	82%	89%	72%	1.94
% female	23%	53%	56%	39%	3.74
# Lifetime hosp.	4.8 (2.0)	4.8 (1.8)	3.8 (1.9)	n/a	1.52
% inpatient	38%	41%	75%	n/a	6.49*

Education: HC > SZ, SZA, BDP, $p < .01$.
Age: HC > BDP, $p < .05$.
Inpatient: BDP > SZ, SZA, $p < .05$.
[a] Education is coded based on the SCID Education and Work History scale: 1 = grade 6 or less; 2 = grade 7–12 (without graduating); 3 = high school grad or equivalent; 4 = part college; 5 = graduated 2 year college; 6 = graduated 4 year college; 7 = part graduate/professional school; 8 = completed graduate/professional school.
* $p < .05$.
*** $p < .001$.

3. Results

Patients did not differ by group on any demographic variable at baseline (Table 1). A greater proportion of patients with BDP were inpatients (BDP > SZ, SZA; $p < .05$). Groups differed on MCAS scores at baseline (BDP, SZA > SZ; $p < .05$) (Table 2). At follow-up, groups differed on PANSS P (SZ > SZA > BDP; $p < .05$) and MCAS (BDP, SZA > SZ; $p < .05$). Patients with BDP showed greater improvement on the YMRS ($p < .05$).

Cognitive data are presented in Fig. 1. At baseline and follow-up groups differed only on Trails B (BDP, SZA > SZ; $p < .05$). Groups differed in neurocognitive improvement over the follow-up only on the BVMT-R (BDP > SZA; $p < .05$) and Stroop Interference (BDP > SZA;

Table 2
Clinical variables by diagnosis.

	SZ ($n = 13$)	SZA ($n = 17$)	BDP ($n = 18$)	F-statistic
Baseline				
MCAS	39.3 (8.5)	45.1 (7.7)	47.7 (7.0)	4.21*
YMRS	11.5 (10.3)	13.1 (11.9)	20.7 (15.4)	2.28
MADRS	10.1 (7.6)	11.0 (8.8)	14.6 (11.7)	0.97
PANSS P	18.7 (8.6)	16.9 (8.3)	16.2 (7.1)	0.38
PANSS N	14.0 (3.9)	11.5 (4.4)	10.7 (7.1)	1.43
PANSS G	28.0 (8.4)	26.8 (9.2)	30.7 (11.4)	0.71
CPZE	534 (402)	476 (384)	294 (294)	1.62
Follow-up				
MCAS	42.6 (6.6)	46.4 (5.1)	48.6 (6.1)	3.80*
YMRS	9.3 (11.5)	8.1 (6.3)	6.9 (6.3)	0.35
MADRS	9.4 (8.4)	10.7 (8.3)	10.5 (12.0)	0.07
PANSS P	17.2 (8.0)	14.8 (5.7)	10.8 (3.5)	4.66*
PANSS N	14.0 (5.1)	13.6 (7.0)	10.7 (3.0)	1.98
PANSS G	28.8 (11.8)	24.9 (6.9)	23.5 (7.0)	1.54
Change scores				
MCAS	3.3 (4.6)	1.0 (9.4)	1.7 (5.4)	0.40
YMRS	−2.2 (11.3)	−5.1 (12.6)	−14.0 (15.7)	3.23*
MADRS	−0.8 (7.4)	−0.3 (8.1)	−4.1 (12.0)	0.80
PANSS P	−1.5 (6.0)	−2.1 (9.3)	−5.9 (6.7)	1.60
PANSS N	0.0 (6.0)	2.1 (6.2)	0.0 (7.6)	0.52
PANSS G	0.8 (6.8)	−1.9 (10.7)	−7.2 (11.3)	2.65

MCAS: Multnomah Community Ability Scale; YMRS: Young Mania Rating Scale; MADRS: Montgomery–Asberg Depression Rating Scale; PANSS: Positive and Negative Syndrome Scale; CPZE: CPZ Equivalents.
* $p < .05$.