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Bridging the gap between schizophrenia and psychotic mood disorders: Relating neurocognitive deficits to psychopathology

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ABSTRACT

Background: The neurobiological relationship between schizophrenia and psychotic mood disorders is not well understood. Neurocognitive deficits have been described in both types of disorders and have been proposed to reflect underlying neurobiological dysfunction. Examining the relationship between neurocognitive function and psychopathology could help illuminate the neurobiological relationship between schizophrenia and psychotic mood disorders.

Methods: Participants included 72 individuals with DSM-IV schizophrenia, 25 individuals with schizoaffective disorder or bipolar disorder with psychotic features, and 72 community controls. Standardized scores and correlations between four domains of neurocognition and psychopathology were examined.

Results: Individuals with schizophrenia and psychotic mood disorders scored similarly on several dimensions of neurocognitive function and psychopathology. The relationships between neurocognitive function and psychopathology were similar in the two groups.

Conclusions: Individuals with schizophrenia and psychotic mood disorders were similar in terms of both the level of impairment in neurocognitive function and psychopathology, as well as in the relationship between the two dimensions of illness. These results suggest that schizophrenia and psychotic mood disorders such as schizoaffective disorder and bipolar disorder with psychotic features are on a neurobiological continuum.

1. Introduction

A major issue facing the developers of the DSM-V and a topic of considerable debate in the literature is whether individuals with schizophrenia (SCZ) or psychotic mood disorders (PMD) are separate and distinct disorders, or whether they occur on a "psychosis" continuum (e.g., Allardyce et al., 2007). Some experts define this continuum as a spectrum of psychotic disorders with mood disorders, such as bipolar disorder with psychotic features (BDP), on one pole and schizophrenia (SCZ) on the other pole with schizoaffective disorder (SA) in the middle (e.g., Lake & Hurwitz, 2007; Kempf et al., 2005). Support for this view is based on reports of increased genetic risk for the continuum of disorders among first-degree relatives of SCZ, SA, and BDP. For instance, when compared to controls, the risk for bipolar disorder is higher in the relatives of SCZ and SA, the risk of schizophrenia is higher in the relatives of BDP and SA, and the risk of schizoaffective disorder is higher in the relatives of BDP and SCZ (Valles et al., 2000; Kendler et al., 1998; Rice et al., 1987; Tsuang et al., 1980). Linkage studies also suggest that the genes related to the risk of developing SCZ, SA, and BDP may be similar (e.g., Potash, 2006; Hamshere et al., 2005).

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The question of whether PMD, such as SA or BDP, are on the same continuum as schizophrenia has also been addressed by examining the severity of neurocognitive deficits and clinical symptoms across the disorders. Prior research has found several similarities between SCZ and PMD. For instance SA and SCZ had similar deficits in neurocognitive performance on individual tasks measuring working memory, executive functioning, and intelligence (e.g., Gooding & Tallent, 2002; Reichenberg et al., 2002). Similarly, SCZ and BDP had similar performance deficits on working memory tasks (Glahn et al., 2006). Also, although Heinrichs and colleagues (2008) found differences between SCZ and SA on specific neurocognitive tests, they concluded that these differences were of insufficient magnitudes to validate two distinguishable disorders. However, few studies compared neurocognitive domain scores between SCZ and PMD (Evans et al., 1999). Additional research is needed to compare the performance of PMD to SCZ with respect to neurocognitive domains, which may be more reliable and robust than scores from individual tests.

Studies comparing the psychopathology of SCZ to PMD indicated that the clinical boundaries between these disorders remain unclear. For instance, some studies found that SCZ, SA, and BDP had similar levels of negative symptoms (Evans et al., 1999; Cuesta & Peralta, 1995), while others did not share this finding (Peralta & Cuesta, 2008; Kendler et al., 1995). The extent of positive and disorganized symptoms among SCZ, SA, and BDP also remains unclear. For instance, Peralta and Cuesta (2008) found that measures of psychosis and disorganization in SA were intermediate between SCZ and BDP, while Evans and colleagues (1999) reported that SCZ scored higher than PMD on measures of positive symptoms. Others found them to have similar ratings on positive (Cuesta & Peralta, 1995; Kendler et al., 1995) and disorganized symptoms (Cuesta & Peralta, 1995). It is possible that these findings were mixed given that few groups used the same methods to assess psychopathology. Thus, our study will use standardized measures to assess psychopathology in SCZ and PMD.

Although existing research has examined the relationships between neurocognitive deficits and psychopathology within SCZ, research has yet to examine how these relationships might vary across different psychotic disorders. Prior research suggests that the domains of psychopathology in SCZ show various relationships to neurocognitive function (e.g., Basso et al., 1998). For example, research on SCZ suggests that positive symptoms show little relationship to neurocognitive function (e.g., Nieuwenstein et al., 2001), while disorganized symptoms have been consistently related to impairments in working memory, executive function and episodic memory (e.g., Bozikas et al., 2004; Cameron et al., 2002). Findings for negative symptoms are mixed, but a number of studies suggest a relationship between negative symptoms and deficits in working memory, executive function and episodic memory (e.g., Nieuwenstein et al., 2001; Palmer & Heaton, 2000). Understanding whether psychopathology and neurocognitive deficits are similarly related among SCZ and PMD will inform theories of diagnosis, psychopharmacologic treatment, and illness pathophysiology. Thus, it is necessary to examine whether the pattern of relationships between neurocognitive deficits and psychopathology is similar between SCZ and PMD.

In the present study, we examined whether individuals with schizophrenia differ from individuals with psychotic mood disorders with respect to [1] the type and severity of neurocognitive deficits, [2] level of psychopathology, and [3] the relationship between neurocognitive function and psychopathology.

2. Methods

2.1. Participants

Participants included 72 individuals with schizophrenia (SCZ), 72 community controls (CON), and 25 individuals with psychotic mood disorders (PMD), including schizoaffective disorder ($n=18$: bipolar subtype ($n=10$), depressive subtype ($n=8$)) and bipolar disorder with psychotic features ($n=7$). We recomputed all study analyses using the participants with schizoaffective disorder ($n=18$) for the comparison group (data not shown). All of the same findings were significant and in the same direction as the total PMD sample ($n=25$). Thus, we elected to use the larger sample for this study to maximize statistical power.

All groups were similar on age, gender, race, and parental socioeconomic status. SCZ and PMD were psychiatrically stable and recruited from a St. Louis metropolitan psychiatric center and its outpatient clinics. CON were recruited through local advertisements. All subjects gave written informed consent for participation after the study's risks and benefits were explained to them. Participants were excluded to avoid biasing results if they had: an unstable medical condition, a neurological disorder, a head injury with loss of consciousness (at any point in lifetime), or substance abuse or dependence in the three months preceding the study. CON were also excluded if they had a first-degree relative with a psychotic disorder.

2.2. Measures

All participants were assessed by Master's or Doctoral clinicians, blind to the diagnosis of the participant, who regularly participated in training and reliability sessions. DSM-IV Axis I diagnoses of each participant were determined by the consensus of a research psychiatrist and trained research clinicians who used the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) (First et al., 2002). The participants' age of illness onset (operationalized as first appearance of psychotic symptoms) was assessed using self-report.

Participants completed a battery of neuropsychological tests. Based on prior research (Nuechterlein et al., 2004), we converted raw scores from the neuropsychological tests into standardized scores (based on current sample) for four domains: crystallized IQ, working memory, episodic memory, and executive functioning. Crystallized IQ was based on a single scaled variable measuring vocabulary from the Wechsler Adult Intelligence Scale—Third Edition (WAIS-III) (Wechsler, 1997a). Working memory was a sum of scaled scores on letter-numbering sequencing, spatial span, and digit span, subtests from the Wechsler Memory Scales—Third edition (WMS-III) (Wechsler, 1997b), and the four-item d' score from the continuous performance task (Barch et al., 2004). Episodic memory was a sum of scaled scores from the WMS-III subtests