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Shared and distinct gray matter abnormalities in schizophrenia, schizophrenia relatives and bipolar disorder in association with cognitive impairment

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ABSTRACT

Cognitive impairments have been linked to structural and functional alterations in frontal and subcortical brain regions, ultimately leading to fronto-thalamic connectivity disturbances. We hypothesized that such neuronal disruptions in frontal and subcortical structures may account for neuropsychological deficits in schizophrenia (SZ), schizophrenia relatives and bipolar disorder (BD).

We acquired T1-weighted anatomical MRI sequences in 209 participants: 57 SZ patients, 47 first-degree relatives of SZ patients, 48 BD I patients and 57 healthy controls. We computed group comparisons of gray matter (GM) volume in frontal and basal ganglia regions-of-interest, followed by correlation analysis between psychomotor speed, executive functioning and learning and GM volumes in candidate regions.

Several frontal GM volume reductions as well as GM increases in the thalamus and the putamen were exhibited in SZ patients as compared to controls. The same finding was observed – less pronounced – when comparing SZ relatives and controls. BD patients presented GM volume increases in the basal ganglia in comparison to controls. In SZ patients, increases in bilateral thalamus GM volume and decreases in left middle and superior frontal gyrus volume were significantly associated with worse cognitive performance.

In summary, our results indicate distinct imbalances across frontal-subcortical circuits in BD, SZ relatives and SZ. The functional relevance of the findings were mainly limited to the SZ patients group: in this group, abnormalities were directly associated with cognitive performance. This result is in line with the finding that the volume alterations were strongest in SZ patients and followed by BD patients and SZ relatives.

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1. Introduction

Partly overlap in clinical and cognitive parameters of bipolar disorder (BD) and schizophrenia (SZ) (Mann-Wrobel et al., 2013; Schaefer et al., 2013), as well as recent neurophysiological and genetic findings suggesting shared heritability (Altshuler et al., 2000; Cross-Disorder Group of the Psychiatric Genomics et al., 2013; International Schizophrenia et al., 2009; O'Donnell et al., 2004; Sanchez-Morla et al., 2008; Smith et al., 2009), indicate a psychosis spectrum (Craddock and Owen, 2005, 2010). Furthermore, structural and functional neuroimaging findings suggest similarities in the distribution of volume decreases (see below) and functional networks, with widespread alterations in SZ

and mainly limbic, para-limbic and inter-hemispheric network abnormalities in BD (d'Albis and Houenou, 2015; Ivleva et al., 2012). A few studies observed that structural abnormalities go along with cognitive deficits in SZ and BD (Ehrlich et al., 2012; Gutierrez-Galve et al., 2011; Hartberg et al., 2010, 2011; Knöchel et al., 2015; Oertel-Knöchel et al., 2012) although these direct associations have not been reported by all authors for BD patients (see e.g. (Gutierrez-Galve et al., 2011)). (See Figs. 1–3.)

Direct comparisons between SZ and BD patients suggest that they share frontal and subcortical abnormalities, which supports the spectrum-hypothesis of psychotic disorders. However, SZ patients have marked fronto-temporal abnormalities compared to BD patients (Ellison-Wright and Bullmore, 2010; Friedman et al., 1999; Ivleva et al., 2012; Janssen et al., 2008; McIntosh et al., 2004; Yu et al., 2010). Moreover, studies on first-degree relatives of SZ patients indicate altered GM volumes in comparable regions – although less pronounced (Borgwardt et al., 2007; Honea et al., 2008; Job et al., 2005;

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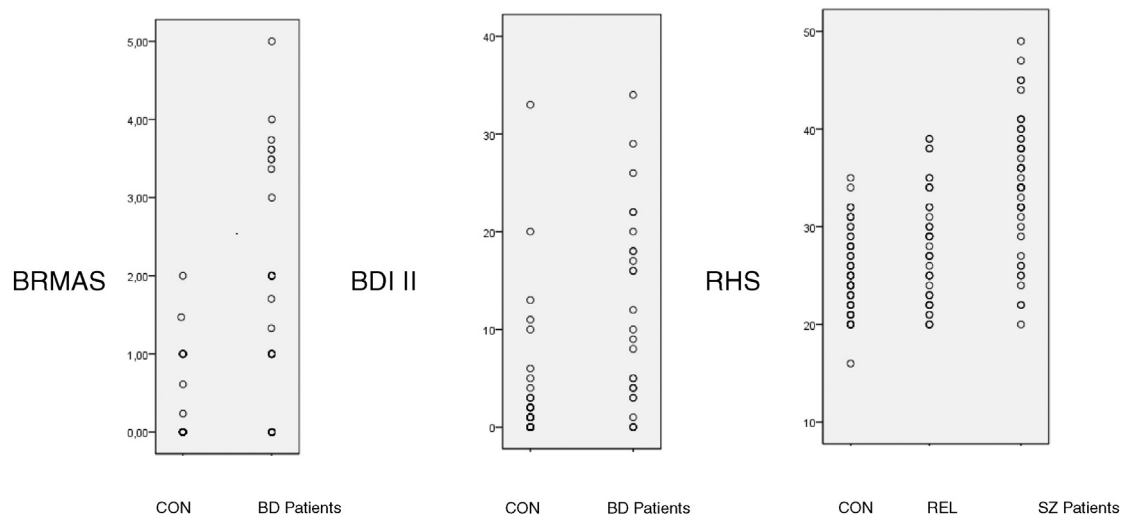


Fig. 1. Scatter plot of the results regarding the clinical scores (BRMAS, BDI II, RHS). Abbreviations: CON = controls, BD = bipolar, SZ = schizophrenia, REL = relatives, BRMAS = Bech Rafaelsen Mania Scale, BDI II = Beck Depression Inventory, RHS = Revised Hallucination Scale.

Oertel-Knochel et al., 2012). Alterations in cortical and subcortical regions (i.e., basal ganglia, thalamus, cingulate, insula, hippocampus) have been observed for both disorders (see for review (Ellison-Wright and Bullmore, 2010; Gupta et al., 2015; Shenton et al., 2001)). Our work group has recently demonstrated that subtle increases in volume of basal ganglia structures might be related to (an elevated) genetic risk for SZ (Oertel-Knochel et al., 2012). However, other studies have suggested that GM volumes reductions are unique to SZ and not present in BD (see i.e. (Altshuler et al., 2000; Farrow et al., 2005; Harvey et al.,

1994; Hirayasu et al., 2001; McDonald et al., 2005; Pearlson et al., 1997; Zipursky et al., 1997), underscoring Kraepelin's dichotomy concept (Kraepelin, 1919).

In this study, we addressed a potential overlap of structural abnormalities and clinical and cognitive symptoms in SZ, first-degree relatives of individuals with SZ and patients with type I BD. We hypothesized that individuals across the psychosis spectrum display shared as well as unique gray matter volume alterations in key brain regions underlying cognitive processing (e.g., frontal lobes, basal ganglia, thalamus). We

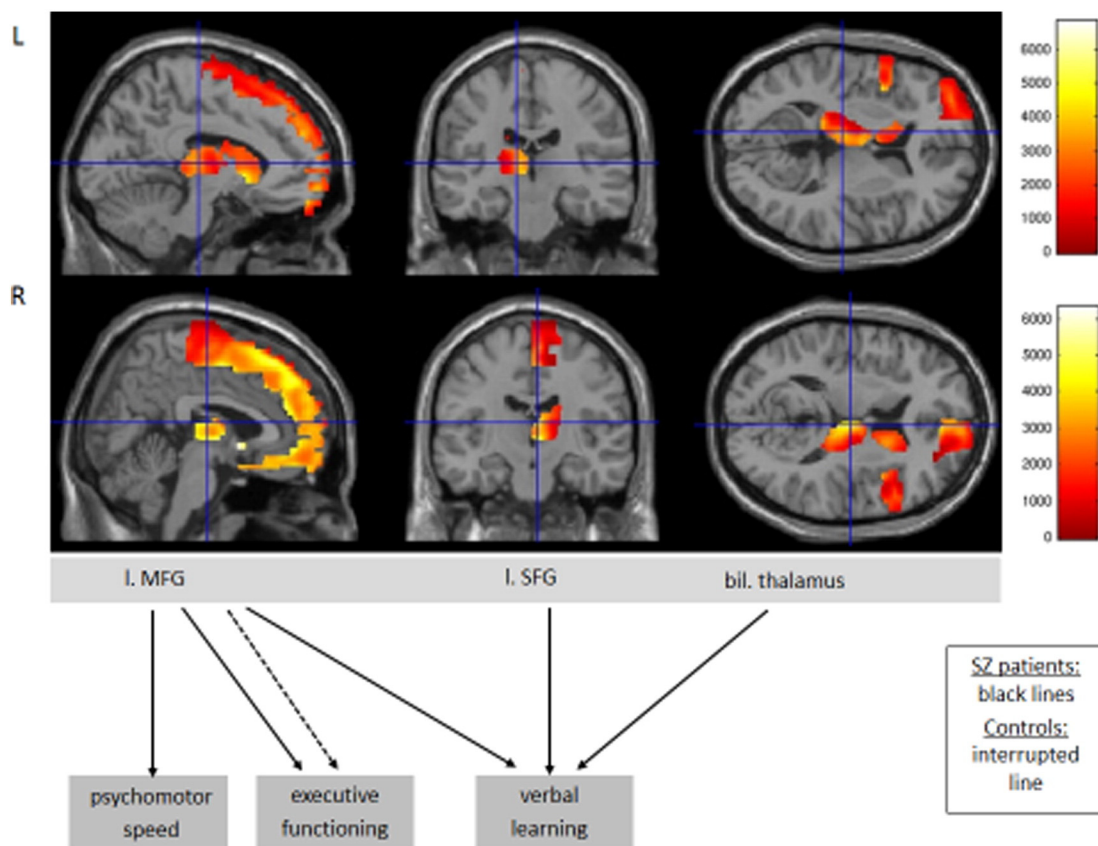


Fig. 2. GM group comparisons (SZ – BD – CON) in the ROIs. Furthermore, relevant correlations with cognitive variables in the SZ patient group (black lines) and the controls (interrupted line).

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