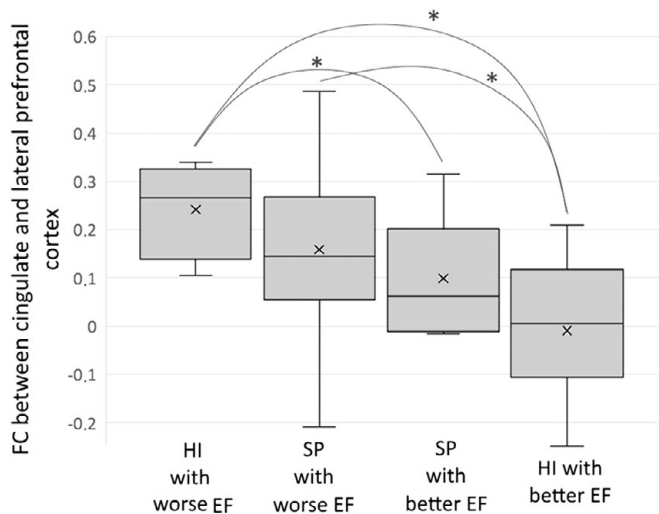


with the quality of outcome. However, neurobiological mechanisms of this heterogeneity are understudied.

Objectives: We aimed to identify features of resting-state functional connectivity (FC) within the frontoparietal network (FPN) that discriminate between SP and healthy individuals (HI) with better and worse EF.

Methods: Twenty-five SP (mean age 20.8 ± 3.23 , illness duration 1.3 ± 2.1 years, all males) and twenty-six HI (mean age 25.17 ± 3.46 , all males) underwent EF assessment (4 verbal fluency tests and a modified Stroop task) as well as resting-state fMRI (3T).

Results: We used *k*-means clustering based on EF scores to divide all participants into groups with worse (15 SP, 6 HI) and better EF (10 SP, 20 HI). These groups differed in productivity of all verbal fluency tasks and performance time of the Stroop task. Differences between four subgroups (HI/SP with worse/better EF) were revealed in FC between the cingulate and lateral prefrontal cortex in the left hemisphere (ANCOVA, *p*-uncorrected $< .005$, *p*[FDR] $< .05$; Fig. 1). SP and HI within each group demonstrated a similar FC pattern. SP with poorer EF had increased FC, compared to HI with higher EF. HI with poorer EF demonstrated increased FC, compared to HI and SP with better EF.



Conclusions: FC within FPN may be one of the neurophysiological underpinnings of EF heterogeneity in SP as well as in HI. Further machine learning fMRI studies are needed to clarify whether FC within FPN is a prognostic marker in schizophrenia.

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Keywords: resting-state fMRI; schizophrenia; frontoparietal network; Executive functions

Bipolar Disorders 01

EPP0087

Long-term brain changes in bipolar disorder

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Introduction: The term “neuroprogression” imply that bipolar disorder (BD) progressively worsens for some patients and accompanying neuroanatomical changes. BD has indeed been associated with cortical and subcortical brain abnormalities. But cross-sectional studies cannot determine whether the observed brain alterations reflect static premorbid traits or whether they result from progressive changes during the course of illness.

Objectives: The aims of this series of studies were to determine if progressive brain changes occur in bipolar disorder, and if so, what the drivers of these changes are.

Methods: We addressed these questions in the St. Göran cohort – a longitudinal study where patients and controls undergo structural magnetic resonance imaging (MRI) scans at baseline and after 7 years. We have also conducted a longitudinal multicenter study within the ENIGMA consortium including 307 patients and 925 healthy controls scanned at two time points.

Results: We addressed these questions in the St. Göran cohort – a longitudinal study where patients and controls undergo structural magnetic resonance imaging (MRI) scans at baseline and after 7 years. We have also conducted a longitudinal multicenter study within the ENIGMA consortium including 307 patients and 925 healthy controls scanned at two time points.

Conclusions: BD is associated with some (accelerated ventricular enlargement) but not global progressive brain changes (change in cortical structures do not differ from controls). Occurrence of manic episodes is, however, associated with accelerated cortical thinning over time. These results highlight the importance of preventing the potentially toxic effects of manic episodes and might explain why some patients experience worsening cognitive function.

Disclosure: ML has received lecture honoraria (unrelated to this topic) from Lundbeck pharmaceuticals.

Keywords: neuroprogression; longitudinal; Neuroimaging; bipolar disorder

EPP0089

The potential protein marker of bipolar disorder

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Introduction: Difficulties in the diagnosis of bipolar disorder (BD) are associated with a lack of understanding of the mechanisms of its pathogenesis. Identification of proteins involved in the pathogenesis of BD will bring us closer to an understanding of its mechanisms and can help in diagnosis.

Objectives: The search of proteomic biomarkers of bipolar disorder.

Methods: We performed a proteomic analysis of the serum of 16 healthy people and 33 patients with BD. Patients were