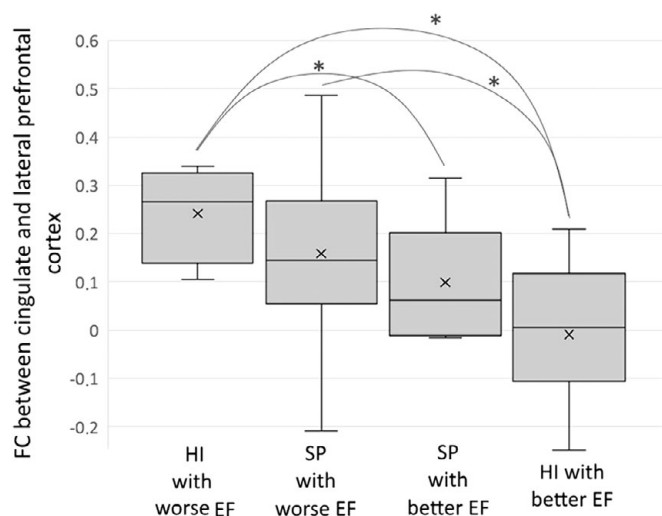


with the quality of outcome. However, neurobiological mechanisms of this heterogeneity are understudied.

**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  $20.8 \pm 3.23$ , illness duration  $1.3 \pm 2.1$  years, all males) and twenty-six HI (mean age  $25.17 \pm 3.46$ , 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.

**Disclosure:** The study was supported by RFBR Grant 20-013-00748.

**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

hospitalized in an acute state of the depressive phase, and they did not receive therapy for more than 6 months. Blood was collected before the start of therapy. Serum was purified from major proteins by affinity chromatography and separated by 1D-electrophoresis. After trypsinolysis, the proteins were identified by HPLC/mass spectrometry. The ELISA kit was used to determine the amount of zNMDAR1.

**Results:** We identified a protein that does not occur in healthy people: a subunit of the glutamate NMDA receptor zeta-1 (zNMDAR1). As a result, we found a statistically significant ( $p = 0.037$ ) almost fivefold increase in the concentration of this protein in the serum of patients with bipolar disorder (0.64 [0.18; 0.78] ng/ml) compared with healthy individuals.

**Conclusions:** Thus, in bipolar disorder NMDAR is damaged, which can lead appearance of their subunits in the serum, and which indicated a violation of glutamatergic neurotransmission. Then this protein claims the role of markers of bipolar disorder. *Mass spectrometric analysis was carried out of the "Human Proteome" Core Facility of the Institute of Biomedical Chemistry Moscow. RSW project, state registration number AAAA-A19-119020690013-2.*

**Disclosure:** No significant relationships.

**Keywords:** bipolar disorder; proteomics; biomarker

## EPP0090

### The Relationships Between Strategies Of Stress Coping And Temperament-Character Traits In Subjects With Bipolar Disorder

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**Introduction:** Bipolar disorder (BD) is a severe mood disorder, which is characterized by a cycling between the mania and major depression. The relationship between coping strategies and temperament-character traits in BD is unclear at this time.

**Objectives:** The aim of our study was to assess the relationship between strategies of coping stress and temperament-character traits in individuals with BD.

**Methods:** 168 patients diagnosed with BD in full remission were included. All participants were diagnosed by an experienced consultant psychiatrist based on DSM-5 and were assessed with Young Mania Rating Scale (YMRS) for confirmation to remission. Socio-demographic datas of all participants was obtained and Temperament Evaluation of Memphis, Pisa, Paris and San Diego-Autoquestionnaire (TEMPS-A) and Coping with Stress Scale (CSS) were applied.

**Results:** 75 patients (44.6%) were female and the mean age of the sample was  $32.64 \pm 10.74$  years, the mean duration of illness was  $8.23 \pm 5.52$  years and was found that the mean score of YMRS  $5.35 \pm 4.19$ . It was presented Table 1 whether there was a statistically significant correlation between TEMPS-A and CSS subscales.

**Conclusions:** As coping strategies may be related to temperament-character traits and that could be important for psychological interventions in patients with BD.

	Depressive	Hypertimic	Cyclothymic	Irritable	Anxious
Avoidance	-,067	-,159	,098	-,150	-,083
	,485	,095	,305	,115	,387
Problem-focused coping strategies	-,268	-,153	,366	-,246	-,134
	,004	,109	,000	-,009	,161
Social support	-,191	-,495	-,060	-,646	-,416
	,044	,000	,535	,000	,000
Total	-,256	-,399	,149	-,370	-,324
	,007	,000	,118	,000	,001

**Disclosure:** No significant relationships.

**Keywords:** bipolar disorders; strategies of stress coping; temperament-character traits

## EPP0091

### Cariprazine's efficacy in treating affective symptoms – pooled data from schizophrenia and bipolar depression trials

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**Introduction:** Affective symptoms are a common feature of schizophrenia and define bipolar disorder. Alterations in dopamine neurotransmission and activity at D<sub>3</sub>-D<sub>2</sub> receptors is associated with depressive symptoms providing the rationale for targeting D<sub>3</sub>-D<sub>2</sub> receptors with partial agonists.

**Objectives:** The aim of the analysis herein is to examine and compare the efficacy of cariprazine in treating affective symptoms in both schizophrenia and bipolar depression.

**Methods:** Data from 3 schizophrenia [NCT00694707, NCT01104766, NCT01104779] and 3 bipolar I depression studies [NCT013896447, NCT02670538, NCT0267055] were pooled for the analyses. To investigate efficacy across individual affective symptoms, the Marder anxiety/depression and negative symptom items of the Positive and Negative Syndrome Scale (PANSS) and single items of the Montgomery-Asberg Depression Rating Scale (MADRS) were analysed. Improvement across affective symptoms was examined primarily evaluating least square mean differences (LSMDs) in comparison to placebo in mean change from baseline.

**Results:** The pooled ITT population was comprised of persons with schizophrenia (placebo=442, cariprazine=1024) and bipolar disorder (placebo=460, cariprazine=923). Cariprazine resulted in a significantly greater reduction when compared to placebo in three out of the four Marder anxiety/depression items; anxiety ( $p < 0.01$ ), tension ( $p < 0.001$ ) and depression ( $p < 0.05$ ). Similarly, cariprazine was significantly better than placebo in 9 out of the 10 MADRS individual items; apparent sadness ( $p < 0.001$ ), reported sadness ( $p < 0.001$ ), reduced sleep ( $p < 0.05$ ), reduced appetite ( $p < 0.001$ ), concentration difficulties