Methods: 40 patients with FEP and 30 healthy controls have been recruited to the study. Patients with affective psychosis, drug-related psychosis and patients with diagnosed encephalitis were excluded. The sera were tested with immune fluorescent assays for anti-NMDAR antibodies. A non-specific method was used to test anti-brain antibody activity on monkey-cerebellum and rat-hippocampus slices.
Results: Neither the samples from the 40 patients, nor the samples of healthy controls contained anti-NMDAR antibodies. 14 of the patients' and only 6 of the healthy controls' serum showed positive reaction of the neuroendothelium. These results suggest that there is a difference between the groups, although the results are not significant.
Conclusions: None of the 40 patients proved positive for anti-NMDAR antibodies in agreement with previous studies. However, a higher proportion of samples from the FEP group showed activity in the neuroendothelium of non-specific immune fluorescent assays compared to healthy controls. Based on literature and on our experience, it is possible, that unknown autoimmune antibodies play role in FEP.

Disclosure: No significant relationships.
Keywords: anti-NMDA receptor encephalitis; antibody; autoimmune encephalitis; First Episode Psychosis

EPP0766

Altered Complement System Activity in Schizophrenia: Overexpression of C4 and/or Abnormal Expression of Complement Control Proteins in the DLPFC, Parietal Cortex, Temporal Cortex, Associative Striatum, Hippocampus, Cerebellum and Whole Blood

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Introduction: In schizophrenia, abnormal synaptic pruning during adolescence may be due to an altered Complement system activity. While this hypothesis is supported by *C4* overexpression in various brain regions of individuals with schizophrenia, such alterations should be replicated and extended to other brain regions relevant to schizophrenia. Moreover, transcriptional studies of genes coding for proteins regulating the Complement system activity are lacking. Furthermore, it remains unknown whether cerebral and peripheral expression of *C4* and Complement control proteins (CCP) are related.
Objectives: To identify altered expression of *C4* and CCP (*CSMD1*, *CSMD2*, *CD46*) coding genes at the cerebral and peripheral levels in schizophrenic individuals.
Methods: We explored *C4* and CCP coding genes expression at the cerebral and peripheral levels. Using *shiny*GEO application we analyzed gene expression from eight Gene Expression Omnibus datasets obtained from 196 schizophrenic individuals and 182 control subjects. First, we compared gene expression between schizophrenic patients and controls in postmortem cerebral samples from 7 different brain regions. Then, we compared gene expression between schizophrenic patients and controls in 4 peripheral tissues.
Results: We observed *C4* overexpression in the DLPFC, parietal, temporal cortex and associative striatum of schizophrenic individuals. We report altered transcriptional patterns of CCP genes in the DLPFC, hippocampus and cerebellum of schizophrenic individuals. *CD46* expression was altered in opposite directions between brain and blood of schizophrenic individuals. No significant alteration of *C4* expression was observed in peripheral tissues.
Conclusions: Our results support the hypothesis of an altered Complement system activity in various brain regions of schizophrenic individuals which may disrupt the synaptic pruning process during adolescence.

Disclosure: No significant relationships.
Keywords: Complement system; schizophrénia; Brain; Gene expression

EPP0767

Dynamics of immune markers in different variants of post-psychotic depression after first-episode psychosis in young adult age.

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Introduction: Research in recent decades focuses on understanding the role of the immune system in First-Episode Psychosis (FEP) at a young age. Our studies indicate that different stages of schizophrenia differ in the spectrum of inflammation markers. These indicators reflect the activity of the pathological process, using them as markers of the clinical state of patients at different stages of the disease.
Objectives: To assess the relationship of immune markers with the clinical features of remission in patients after FEP.
Methods: Fifty patients aged 15-25 years with post-psychotic depression (PD) after FEP (F20, F25) and 30 healthy men were included in the study. The follow-up period was two years. PD typological variants with positive affectivity (PA) (n=30) and negative affectivity (NA) (n=20) were distinguished. Leukocyte elastase (LE), a1-proteinase inhibitor (a1-PI) activity, and S-100B autoantibodies in plasma samples were measured.
Results: The increase of LE and a1-PI activity in plasma of both types of PD patients compared to controls was detected ($p<0.01$). There was the highest LE activity and S-100B autoantibodies in PD with NA ($p<0.05$). The different dynamics of immune markers in both groups were correlated to the clinical features of remission. PD with PA was associated with a decrease in inflammatory markers ($p<0.05$) and a favorable prognosis. PD patients with NA had a further increase in LE activity and S-100B autoantibodies ($p<0.01$), and an unfavorable prognosis.