aET, which is likely to be a consequence of previous endotoxin aggression. There were correlations between ET concentration and antibodies to neuroantigens S-100B and MBP. We also revealed the association between the activity of the inflammatory marker with the severity of clinical symptoms in patients.
Conclusions: Results suggest the relationship between systemic inflammation markers and indicators of systemic endotoxemia and their involvement in the pathogenesis of endogenous psychosis.

Disclosure: No significant relationships.
Keywords: endogenous psychosis; systemic inflammation; systemic endotoxemia; antiendotoxin antibodies

EPV1108

Immunoregulatory and neuroprotective activity of ovocystatin

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Introduction: Ovocystatin has beneficial properties for cognitive function in young rats and might prevents aging-related cognitive impairment in older animals, as well as reduces memory decline in APP/PS1 mice model.
Objectives: Our study aimed at assessing the impact of ovocystatin on microglia activation and neurogenesis.
Methods: Immunoactivation: Mouse wild type microglia were stimulated with ovocystatin at dose of 100 micrograms/ml. The effect of ovocystatin on nitric oxide production and interleukin 1 beta secretion were determined. Neurogenesis: Primary rat hippocampal neurons of H19-7 cell line was used. The impact of ovocystatin on proliferation, nitric oxide production, and expression of markers of neurogenesis: microtubule-associated protein 2 (MAP2, isoforms A/B and C/D) and Synapsin 1, were determined.
Results: It was shown that ovocystatin does not stimulate microglial cells to produce inflammatory mediators. Whereas, no toxic effect of ovocystatin (1-100 ug/ml) on H19-7 cells viability, and dose-dependent down-regulation of proliferation were demonstrated. It was also shown that in primary hippocampal neurons of H19-7 cells incubated with ovocystatin (100 micrograms/ml), the expression level of MAP2 C/D (75kDa) - characteristic form of immature neurons is unchanged. However, the increased expression of MAP2 A/B protein (280 kDa) – characteristic for mature neurons was observed after 6 and 24h incubation with ovocystatin. Relatively to MAP2 A/B, increased expression of synapsin 1 was observed.
Conclusions: The ovocystatin might be a potential activator of molecular mechanisms in primary hippocampal neurons, participating in regulation of neurogenesis. Nevertheless, further studies are needed.

Disclosure: No significant relationships.
Keywords: ovocystatin; Immunoactivation; Neurogenesis

EPV1109

Immunology and psychosis: 22q11.2 syndrome as a model of study

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Introduction: The 22q11.2 deletion syndrome (22q11DS) defines a set of developmental abnormalities due to a loss of genetic material. Phenotypic expression is highly varied. The immunological alterations that present as a severe combined immunodeficiency and the neurodevelopmental alterations stand out, especially the psychotic symptoms. It is the best described genetic alteration for the development of psychosis, presenting up to 30% of patients with compatible symptoms, with various hypotheses that justify it.
Objectives: General objective:
1. Justify that SD2q11 can be used as a model for human research on the origin of psychosis.
Specific objectives: 1. Describe the pro-inflammatory state present in SD2q11.
2. Describe differences at the immunological level in SD2q11 among those patients with presence of psychotic symptoms of those who do not present them.
3. Describe possible biomarkers.
Methods: A systematic review of the literature in the last 5 years using electronic resources (PubMed and WOS) until June 2021 following the PRISMA recommendations.
Results: Three original articles were reviewed. There is a very marked pro-inflammatory state in 22q11DS patients with psychotic symptoms. They present an increase in subpopulations of CD4. The IL-17 is important in the formation in primitive stages of the hippocampus, its ease of breaking the blood-brain barrier (BBB). The neutrophil / lymphocyte ratio is presented as a possible biomarker to predict patients at high risk of developing psychosis.
Conclusions: There is sufficient evidence that patients with 22q11DS can be used as a research model regarding possible hypotheses about the genesis of psychosis.

Disclosure: No significant relationships.
Keywords: Digeorge syndrome; psychosis; schizophrenia; inflammation

EPV1110

Leukocyte elastase, a1-proteinase inhibitor, and autoantibodies to neuroantigens in diagnostics of endogenous depressive disorders

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Introduction: It has been suggested that the activation of systemic inflammatory response in depression is associated with inflammatory changes in the brain (neuroinflammation) and may reflect the severity of the clinical symptoms in patients.
Objectives: To study the relationship between clinical and immune parameters in patients with endogenous depressive disorders for the possible use of these indicators for diagnostics of these conditions.
Methods: Patients with bipolar affective disorder (group 1) and recurrent depressive disorder (group 2) (F31, F32, F33) were examined before the therapy. Mentally healthy age- and gender-matched persons were investigated as controls. The severity of depressive symptoms was assessed by HDRS. The activity of inflammatory indicators (leukocyte elastase (LE) and 1-proteinase inhibitor (α1-PI)), as well as the level of autoantibodies (AB) to S-100B and MBP, were measured in plasma.
Results: Group 1 was characterized by an increase of LE and α1-PI activity in comparison with the control group ($p<0.001$; $p=0.002$) and group 2 ($p<0.05$). No significant difference in AB to neuroantigens was found. Group 2 was distinguished by the increase in activity of the inflammatory indicators ($p<0.01$; $p<0.05$) as well as the autoimmune reactions to neuroantigens compared with control one ($p=0.03$). The correlations between complex assessment of the immune system and the severity of depressive symptoms in both groups were revealed ($\chi2=6.1$; $p=0.013$; $\chi2=4.8$; $p=0.05$).
Conclusions: Revealed correlations suggest that inflammatory markers are involved in the pathogenesis of endogenous depressive disorders and can be used as an additional differential diagnostics criterion for the assessment of the clinical state of patients.

Disclosure: No significant relationships.
Keywords: endogenous depressive disorders; leukocyte elastase; a1-proteinase inhibitor; autoimmune reactions

EPV1111

Psychiatric symptoms in autoimmune encephalitis. A case report.

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Introduction: Early stages of autoimmune encefalitis (AE) often present cognitive and neuropsychiatric symptoms such as personality change, irritability, axiety, depression, behavioral disorders, hallucinations, disorientation, sleep-wake cycle reversals, …). Thus often these cases are first treated as psychiatric disorders.
Objectives: A literature review throughout a case report presentation.
Methods: We present the case of a 25-year old female with a medical history of iron-deficiency anemia who arrives at the emergency service. She presents the following one week of evolution clinical picture: complex auditive hallucinations, behavioral disturbances, sleep disorder and short term memory impairment. Neurological examination, LP and craneal CT are all normal. CSF analysis has no abnormalities. Thus she entered the psychiatric ward. There she was treated with neuroleptics with no improvement of symptoms presenting a severe psychomotor agitation and language impairment. After neurology interconsultation AE is suspected.
Results: She was performed an EEG (left temporal epileptiform activity), CSF (inflammatory pattern), MRI (bilateral temporal lobe hyperintensity). Suspecting limbic encephalitis the presence of anti-NMDAR antibodies was tested , which turned out to be positive. First she was treated with corticotherapy with mild results. Then she was treated with intravenous immunoglobulin improving significantly.
Conclusions: Anti-NMDAR encephalitis is usually a multistage illness. Early in the course of disease psychiatric manifestations are not rare. Therefore the proper diagnosis and approach of AE may requiere a highly organized assessment, starting with detailed history and physical examination and an appropriate testing to exclude other possible relevant pathologies.

Disclosure: No significant relationships.
Keywords: autoimmune encephalitis; neuroleptics; auditive hallucinations; immunoglobulin

EPV1113

Imunne system and schyzophrenia

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Introduction: Schizophrenia affects approximately 1% of the world population, having a devastating impact not only in patients but in all society. As a result, it has been subject of extensive investigation and the presence of certain genes was associated with an increased risk of developing schizophrenia. However, the presence of these genes is not sufficient, therefore, other factors are necessarily involved.Observation of the association between schizophrenia and inflammatory states of the Central Nervous System led to the hypothesis that a dysfunction of the immune system may play a central role in this process.
Objectives: In this work we intend to make a brief review of the existing literature related to the immunological theory of schizophrenia.
Methods: A bibliographic research was conducted in Medline library using the following terms: “schizophrenia and immune system”; “schizophrenia and inflammation” and “schizophrenia and neuroinflammation”.
Results: The survey results reveal increasing evidence of the key role of the immune system in schizophrenia. Several studies show benefits of treatment with anti-inflammatory drugs in patients at an early stage of the disease. In the same way, it was verified that pro and anti-inflammatory cytokines influence glutamatergic transmission and tryptophan metabolism. Furthermore, the decrease in microglial activity appears to have a beneficial effect on schizophrenia.
Conclusions: Future will say if neuroimmunology mechanisms are primary or a secondary consequence in Schizophrenia. Recent discoveries in this area are encouraging and open the possibility of new therapeutic targets and new therapeutic approaches to this disease.