psychosis, regarding genetic vulnerability and/or other environmental factors, possibly also mediated by psychological mechanisms. Long-term modifications to the transcriptome are likely mediated by epigenetic mechanisms. There is also growing evidence supporting an association between childhood trauma and adulthood dysregulation of the immune system, which could help clarify the relationship between trauma and mental disorders, namely psychosis.
Objectives: Review evidence regarding the relationship of childhood trauma, immune system and psychosis.
Methods: Literature review using Medline database.
Results: The prevalence and severity of childhood trauma is characterized by both biological alterations and increased risk of experiencing symptoms of psychosis. Childhood trauma, namely through its effects on IL6 levels, may be a risk factor for schizophrenia in general. Some studies point to a direct relationship between childhood trauma, immunity and psychosis when examined along a continuum from non-clinical controls to psychotic disorders such as schizophrenia.
Conclusions: For better understanding this association, these findings must be replicated in larger cohorts. If the impact of childhood trauma on immune function in adulthood does indeed contribute to psychopathology, an improved understanding of this relationship may lead to new and possibly more specific treatment options. Other clinical implications of these findings include increased emphasis in establishing more comprehensive screening of early trauma in patients with psychotic symptoms, as well as the importance of screen and follow children who report traumatic events for emergence of psychotic symptoms.

Keywords: childhood trauma; ımmune system; psychosis

EPP1017

Systemic endotoxinemia as a probable factor in reducing the treatment effectiveness of endogenous psychosis

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Introduction: Inflammation is an important factor in the pathogenesis of endogenous psychosis. An inducer of inflammatory reactions can be endotoxin aggression of intestinal origin.
Objectives: To determine the level of inflammation markers and indicators of systemic endotoxinemia in blood of patients with endogenous psychosis in relation to assessment of the treatment effectiveness.
Methods: 25 patients with endogenous psychosis (F20, F25) were examined before and after treatment. The control group consisted of 25 healthy people. The activity of inflammatory markers - leukocyte elastase, α1-antitrypsin, antibodies to S-100B, and indicators of systemic endotoxinemia – endotoxin concentration and antiendotoxin immunity activity were measured in blood serum. The treatment effectiveness was assessed by the dynamics of inflammatory markers.
Results: Based on the results of determining the studied parameters before treatment, all patients were divided into two groups. In the 1st group (6 patients, 24%), an increase of inflammatory markers activity and high concentration of endotoxin in the blood serum were revealed ($p<0,001$, $p<0,05$, respectively). In the 2nd group (19 patients, 76%), only activation of inflammatory reactions ($p<0,001$) was detected. After therapy in the 1st group of patients, there was no positive dynamics of all studied markers, which indicated an active course of the pathological process. In the 2nd group, the normalization of inflammatory markers was shown ($p<0,05$), which corresponded to the formation of remission.
Conclusions: The results indicate that endotoxic aggression contributes to reduction of the effectiveness of endogenous psychosis therapy and can be considered as an additional therapeutic target.

Keywords: endogenous psychosis; inflammatory markers; treatment effectiveness; systemic endotoxemia

EPP1018

Immune heterogeneity of non-psychotic mental disorders

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Introduction: Current studies indicate the involvement of inflammation in the pathogenesis of chronic non-infectious diseases, and therefore it is of interest to study the role of inflammation markers in non-psychotic mental disorders (NPMD).
Objectives: To identify a number of inflammatory markers in serum of patients with NPMD.
Methods: 73 patients with NPMD were examined (F43.2; F06.6). The comparison group consisted of 76 patients with endogenous psychosis (EGP) (F20.0; F25.0). The control group included 80 healthy people. The serum activity of leukocyte elastase (LE), α1-proteinase inhibitor (α1-PI) and the level of autoantibodies (aAb) to neuroantigens were determined.
Results: Three groups of patients with different variants of inflammatory response to the pathological process were identified. In group 1 (23.3%), all indices corresponded to the control values, which indicated the absence of the pathological process in brain. In group 2, there was a significant increase in activity both LE and α1-PI compared to control ($p<0.05$). This type of immune reaction characterized a balanced inflammatory response. It was found in 52% of patients with NPMD and in all patients with EGP. The aAb level also exceeded the control values ($p<0.05$). Group 3 (24.7%) showed an increase in α1-PI activity ($p<0.05$), but not in LE activity compared to control. Insufficient LE activity reflects a decrease in the functional activity of neutrophils.
Conclusions: The immune heterogeneity of NPMD according to the level of inflammatory markers was identified. 52% of patients with NPMD have a pronounced activation of inflammatory reactions accompanied by increased levels of aAb to neuroantigens.

Keywords: non-psychotic mental disorders; inflammatory markers