Introduction: Cell technologies actively used in the treatment of many diseases. These technologies are based on manipulating the patient's cells outside the body, as a result of which cells acquire a higher therapeutic potential.
Objectives: No doubt the essential role of immune cells and their biologically active products in the pathogenesis of depression, which allows to view the modulated immune cells as model objects for developing new approaches to immunotherapy for depression.
Methods: (CBAxC57Bl/6) F1 depressive-like male mice, developed under the long-term social stress, were undergoing the transplantation of syngeneic immune cells with in vitro caffeine-modulated functional activity. Recipient's behavior, immune and nervous systems functional activity were studied.
Results: It was found that immune cells isolated from depressive-like mice and treated in vitro with caffeine change their properties and after intravenous administration to syngeneic depressive-like recipients have a significant positive psycho- and neuroimmunomodulatory effects, affecting the main depression pathogenetic mechanisms: behavioral editing (reduction of anhedonia, stimulation of exploratory behavior and activity in the forced swimming test); hippocampal neurogenesis stimulation against the background of increased BDNF; modulation of cytokine production by brain cells, indicating a decrease in neuroinflammation; modulation of the immune system functional activity (stimulation of the immune response, splenocytes proliferation, reducing systemic inflammation, decrease spleen tryptophan catabolism).
Conclusions: The results serve as an experimental substantiation of a fundamentally new approach to immunotherapy of depression based on the introduction of immune cells with functional activity modulated outside the body and open up the possibility of developing new methods of immunotherapy of depressive states in humans.

Disclosure: No significant relationships.
Keywords: modulated immune cells; Cell technologies; Depression; Immunotherapy

EPV0490

Human type 2 macrophages biologically active soluble products in the editing of stress-induced depressive-like behavior

E. Markova[1]*, E. Shevela[2], M. Knyazheva[1], I. Savkin[1], T. Amstislavskaya[3], A. Ostanin[2] and E. Chernykh[2]

[1]Neuroimmunology Lab, State Research Institute of Fundamental and Clinical Immunology, Novosibirsk, Russian Federation; [2]Cellular Immunotherapy Laboratory, State Research Institute of Fundamental and Clinical Immunology, Novosibirsk, Russian Federation and [3]Translational Biopsychiatry Lab, Research Institute of Neurosciences and Medicine, Novosibirsk, Russian Federation

*Corresponding author.
doi: 10.1192/j.eurpsy.2021.2023

Introduction: In the scientific world widely discussed phenomenon of "cytokine-induced depression". Macrophages have high plasticity and are able to control the inflammatory response; in particular, anti-inflammatory type-2 macrophages have a pronounced potential due to complex soluble factors production.
Objectives: We have developed an original method for the type-2 macrophages generation; the resulting macrophages are characterized by the high level of a whole range of neurotrophic, neuroprotective, proangiogenic and anti-inflammatory factors production. The aim of the study was to investigate effects of human type-2 macrophages soluble products on behavioral phenotype and brain cytokines synthesis in depressive-like animals.
Methods: Type-2 macrophages were generated by culturing an adherent fraction of mononuclear cells with 50 ng/ml recombinant human GM-CSF in serum deprivation conditions for 7 days. (CBA x C57Bl/6)F1 depressive-like male mice, developed under the long-term social stress, were undergoing the human type-2 macrophages conditioned medium intranasal administration (60 ml twice daily for one animal) for 6 days. Mice behavioral phenotyping was carried out using an automatic registration system (Noldus Information Technology). Cytokines were determined by ELISA.
Results: Depressive-like mice behavioral phenotyping after type-2 macrophages conditioned medium administration revealed anhedonia decrease, motor activity stimulation in the open field and forced swimming tests, anxiety reduction in elevated plus maze. Behavioral changes were recorded against the pro-inflammatory cytokines (TNF-α, IL-1β, IL- 6, INFγ) decrease in striatum and hippocampus, as well as anti-inflammatory IL- 10 increase in hippocampus and hypothalamus.
Conclusions: Results demonstrated the effectiveness of human type-2 macrophages biologically active soluble products in relation to the stress-induced depressive-like behavior editing

Disclosure: No significant relationships.
Keywords: anti-inflammatory macrophages; depressive-like behavior; cytokines

EPV0491

The role of inflammation in pathogenesis of juvenile schizophrenia

S. Zozulya[1]*, M. Omelchenko[2], I. Otman[1], A. Yakimets[2], Z. Sarmanova[1], V. Kaleda[2] and T. Klyushnik[1]

[1]Laboratory Of Neuroimmunology, Mental Health Research Centre, Moscow, Russian Federation and [2]Department Of Youth Psychiatry, Mental Health Research Centre, Moscow, Russian Federation

*Corresponding author.
doi: 10.1192/j.eurpsy.2021.2024

Introduction: Inflammation is now known to be a key factor in the development of schizophrenia. In this regard, the study of the pathogenic role of inflammation in the early stages of schizophrenic process is of particular importance, making it possible to assess its activity and to predict the development of the disease.
Objectives: To compare the dynamics of inflammatory markers in blood of first-episode psychosis (FEP) patients and people at risk signs for schizophrenia in the course of the treatment. Juvenile depression (JD) with attenuated symptoms of schizophrenic spectrum (ASSS) was investigated as a risk group.
Methods: The patients aged 17-25 years (20 people, of which 10 FEP patients (F20) and 10 JD with ASSS ones (F32.1-2, F32.38, F32.8)) were examined at admission to the hospital and at discharge. The controls consisted of 10 healthy volunteers. Symptom severity was collected using PANSS, SOPS, SANS, HDRS. The inflammation markers (TNF-α, IL-6, IL-10, leukocyte elastase (LE), CRP, α1-proteinase inhibitor (α1-PI), anti-S100-beta antibodies) were determined in blood.

Results: An increase of inflammatory markers in both groups compared to controls was found (p<0,05). The highest values of IL-6, LE, CRP, α1-PI and anti-S100-beta antibodies in FEP patients were revealed (p=0,03). After the treatment, the positive trend of inflammatory markers in FEP patients (p<0,05), but not in JD with ASSS patients was detected (except LE activity, p<0,05).
Conclusions: The results confirm the pathogenic role of inflammation in the development of endogenous mental disorders. The inflammatory markers studied reflect the activity of the pathological process in the early stages of schizophrenia.

Disclosure: No significant relationships.
Keywords: attenuated symptoms of schizophrenic spectrum; first-episode psychosis; inflammatory markers; juvenile depression

EPV0492

Effects of long-term therapy with quetiapine and olanzapine on parameters of immunity and cytokine levels in patients with schizophrenia

O. Lobacheva[1]*, V. Nikitina[1], E. Kornetova[2], S. Vladimirova[3] and A. Semke[2]

[1]Laboratory Of Clinical Psychoneuroimmunology And Neurobiology, Tomsk National Research Medical Center, Tomsk, Russian Federation; [2]Department Of Endogenous Disorders, Mental Health Research Institute, Tomsk National Research Medical Center, Russian Academy of Sciences, Tomsk, Russian Federation and [3]Department Of Coordination Of Research, Mental Health Research Institute, Tomsk National Research Medical Center, Russian Academy of Sciences, Tomsk, Russian Federation

*Corresponding author.
doi: 10.1192/j.eurpsy.2021.2025

Introduction: The study of effects of long-term antipsychotic therapy in patients with schizophrenia is relevant.
Objectives: To study effects of long-term antipsychotic therapy on parameters of immunity and cytokine levels in patients with schizophrenia.
Methods: We examined 20 schizophrenic patients, who received quetiapine (group 1) and 17 - olanzapine (group 2) for more than 6 months before admission in the hospital as the main anti-recurrence therapy. Persons aged 20-63 years with length of the follow-up of the disease ≥1 year were included. The investigations included: phenotyping of immunocompetent cells into CD differentiation clusters by flow cytometry; mitogen-induced, spontaneous production of cytokines (IL2, IFN-γ, IL-4, TNF-α) were identified with use of kits for enzyme-linked immunosorbent assay (ELISA).
Results: It was shown that patients of group 1 in comparison with group 2 were characterized by lower values of CD3- lymphocytes (p=0.049), higher values of the spontaneous production of IFN-γ (p=0.01), mitogen-induced production of IL-2 (p=0.043) and IL-4 (p=0.059). In all examined low level of mitogen-induced of IFN-γ (p=0.0001) and TNFα (p=0.002; p=0.0001), high level of spontaneous production of TNFα (p=0.001) were revealed in relation to control.
Conclusions: It was found that the acute period of schizophrenia after prolonged treatment with atypical antipsychotics is accompanied by immunological imbalance and dysregulation of the cytokine system. More severe immune disorders when hospitalized during the exacerbation period were revealed in patients who had been receiving antipsychotic therapy with the atypical antipsychotic quetiapine for a long time. This can be associated with the features of the mechanism of action of atypical antipsychotics.

Disclosure: No significant relationships.
Keywords: Psychoneuroimmunology; schizophrénia; Antipsychotics

EPV0493

Autoantibody profiles are associated with specific clinical features in psychotic disorders

K.O. Schubert[1,2]*, A. Jernbom Falk[3], C. Galletly[1,2], D. Just[3], C. Toben[1], B. Baune[4,5], S. Clark[1], D. Liu[1], P. Nilsson[3] and A. Manberg[3]

[1]Discipline Of Psychiatry, The University of Adelaide, Adelaide, Australia; [2]Community Mental Health, Northern Adelaide Mental Health Services, Salisbury, Australia; [3]Science For Life Laboratory, Royal Institute of Technology, Stockholm, Sweden; [4]The University Of Melbourne, The Florey Institute of Neuroscience and Mental Health, Parkville, Australia and [5]Department Of Psychiatry And Psychotherapy, University of Münster, Münster, Germany

*Corresponding author.
doi: 10.1192/j.eurpsy.2021.2026

Introduction: Immune system abnormalities exist across a range of psychiatric disorders. Autoimmunity, characterized by the production of antibodies against the body's own antigens, is a feature of immune system dysfunction and could play a role in mental disorder pathophysiology. Better understanding of the associations of auto-immunoglobulin G (IgG) repertoires with clinical features of mental illness could yield novel models of psychosis pathophysiology and markers for biological patient stratification.
Objectives: To undertake global screening for auto-IgG expression in a large cohort of people with psychotic disorders; to determine whether associations exist between autoantibody expression and clinical features.
Methods: Cross-sectional quantification of auto-IgGs in blood plasma of 461 people with established psychotic disorder diagnoses. For global screening, pooled samples of phenotypically representative patient groups were exposed to planar protein microarrays containing 42,000 human antigens. For targeted profiling, expression levels of 380 autoantibodies were quantified by suspension bead array (SBA) in each patient's plasma.
Results: We identified highly individual autoantibody profiles with no evidence for co-expression patterns. We found 6 autoantibodies robustly associated with specific psychopathology: anti-AP3B2, detected in 5% of the cohort of whom 100% had persecutory delusions; anti-TDO2 (5% of the cohort, 100% hallucinations); anti-CRYGN (4%, 86% initial insomnia); anti-APMAP (3%, 86% poor appetite); anti-OLFM1 (2.5%, 100% above median cognitive function); and anti-WHAMMP3 (2%, 90% anhedonia and dysphoria). Examination of the auto-IgG binding site on the TDO2 protein revealed a putative pathophysiological mechanism involving the kynurenine pathway.
Conclusions: We identified 6 frequently occurring autoantibodies that were associated with specific clinical features in people with psychotic disorders.