

**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:** (CBAx C57Bl/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<sup>1\*</sup>, E. Shevela<sup>2</sup>, M. Knyazheva<sup>1</sup>, I. Savkin<sup>1</sup>, T. Amstislavskaya<sup>3</sup>, A. Ostanin<sup>2</sup> and E. Chernykh<sup>2</sup>

<sup>1</sup>Neuroimmunology Lab, State Research Institute of Fundamental and Clinical Immunology, Novosibirsk, Russian Federation; <sup>2</sup>Cellular Immunotherapy Laboratory, State Research Institute of Fundamental and Clinical Immunology, Novosibirsk, Russian Federation and

<sup>3</sup>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- $\alpha$ , IL-1 $\beta$ , IL-6, INF $\gamma$ ) 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<sup>1\*</sup>, M. Omelchenko<sup>2</sup>, I. Otman<sup>1</sup>, A. Yakimets<sup>2</sup>, Z. Sarmanova<sup>1</sup>, V. Kaleda<sup>2</sup> and T. Klyushnik<sup>1</sup>

<sup>1</sup>Laboratory Of Neuroimmunology, Mental Health Research Centre, Moscow, Russian Federation and <sup>2</sup>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- $\alpha$ , IL-6, IL-10, leukocyte elastase (LE), CRP,  $\alpha$ 1-proteinase inhibitor ( $\alpha$ 1-PI), anti-S100-beta antibodies) were determined in blood.