

UHPLC-MS/MS. ANCOVA was performed adjusting for sampling, sociodemographic, health and lifestyle variables.

Results F2-isoprostanes did not differ between controls and patients, or by antidepressant use. Patients (current or remitted) using antidepressants had lower 8-OHdG (adjusted mean 38.3 pmol/L) compared to patients (current or remitted) without antidepressants (44.7 pmol/L) and controls (44.9 pmol/L, $P < 0.001$; Cohen's d 0.26). Findings for 8-OHdG were similar over all disorders and all antidepressant types (SSRIs, TCAs, SNRIs; $P < 0.001$).

Conclusion Contrary to previous findings this large-scale study did not find increased oxidative stress measured by F2-isoprostanes or 8-OHdG in MDD or anxiety disorders. 8-OHdG levels were lower in antidepressant users, which suggests antidepressants may have antioxidant properties.

Disclosure of interest The authors have not supplied their declaration of competing interest.

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EW418

Antioxidant uric acid is lower in current major depression and anxiety disorders

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Introduction It has been hypothesized that lowered antioxidant capacity, which leads to increased oxidative stress, may be involved in the pathophysiology of major depressive disorder (MDD) and anxiety disorders and might be altered by antidepressant treatment.

Objectives This study investigated the association of plasma uric acid, the greatest contributor to blood antioxidant capacity, with MDD, generalized anxiety disorder, social phobia, panic disorder, agoraphobia and antidepressants in a large cohort.

Methods Data was derived from the Netherlands Study of Depression and Anxiety including patients with current ($n = 1648$) or remitted ($n = 609$) MDD and/or anxiety disorder(s) (of which $n = 710$ antidepressant users) and 618 controls. Diagnoses were established with the Composite Interview Diagnostic Instrument. Symptom severity was ascertained in all participants with the Inventory of Depressive Symptoms and the Beck Anxiety Inventory. ANCOVA and regression analyses were adjusted for sociodemographic, health and lifestyle variables.

Results Plasma uric acid was lower in those with current MDD and/or anxiety disorder(s) (adjusted mean 270 $\mu\text{mol/L}$) compared to those with remitted disorders (280 $\mu\text{mol/L}$, $P < 0.001$) or to controls (281 $\mu\text{mol/L}$, $P < 0.001$; Cohen's d 0.14). Within patients antidepressants were not associated with uric acid levels. Increasing symptom severity was associated with lower uric acid levels for both depression ($\beta = -0.05$, $P = 0.001$) and anxiety symptoms ($\beta = -0.05$, $P = 0.004$).

Conclusion This large scale study finds that the antioxidant uric acid is lower in current, but not remitted, MDD or anxiety disorders and in persons with higher symptom severity, suggesting disturbances in redox homeostasis play a role in the pathophysiology of depression and anxiety disorders.

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EW419

Interleukin-receptor antagonist (IL1-RA) with respect to schizophrenia psychopathology

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Introduction The influence of the immune deregulation on the risk and psychopathology of schizophrenia is increasingly recognized in the literature.

Aim To assess the association between serum IL-1RA on schizophrenia psychopathology.

Methods We recruited 88 schizophrenia patients (38 males and 49 females, mean age 38.12 ± 12.67 years) and 88 healthy adult control subjects (68 males, 20 females, mean age 40.63 ± 7.99 years). Lifetime psychopathology was evaluated using Operational Criteria for Psychotic Illness (OPCRIT) checklist, while current psychopathology was assessed using Positive and Negative Syndrome Scale (PANSS). Serum samples were stored in aliquots at -80°C . Serum levels of IL-1RA were measured using Immunoassay (ELISA).

Results There were statistically significant differences between schizophrenia patients and healthy controls (median \pm interquartile range: 350.81 ± 227.04 and 888.74 ± 762.63 , respectively [pg/ml]) (U Mann-Whitney test, $Z = -7.99$, $P < 0.0001$). There were no differences in serum IL-1RA levels between male and female among patients with schizophrenia (U Mann-Whitney test, $Z = -0.22$, $P = 0.82$) nor among healthy control subjects (U Mann-Whitney test, $Z = -0.17$, $P = 0.86$). Among schizophrenia patients, there was a trend-level association between IL-1RA serum level with negative symptoms (Spearman correlation coefficient, $r = -0.23$, $P = 0.056$), positive symptoms (Spearman correlation coefficient $r = -0.22$, $P = 0.066$), and on a statistically significant level with general symptoms (Spearman correlation coefficient $r = -0.28$, $P = 0.018$).

Conclusion Serum IL-1RA level is higher in schizophrenia patients in comparison to healthy controls and it is associated with schizophrenia psychopathology.

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EW423

Immunomodulatory role of paliperidone in the poly(I:C) model of schizophrenia

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Introduction Alterations on the innate inflammatory response may underlie the pathophysiology of psychiatric diseases, but the mechanisms implicated remain elusive. Current antipsychotics

modulate pro/anti-inflammatory pathways, but the specific mechanisms involved remain elusive. One attractive possibility is the regulation of the intracellular signalling pathways of the innate immune receptors Toll-like 3 (TLR3), which triggers antiviral and inflammatory responses.

Aims To elucidate the regulatory role of paliperidone on maternal immune activation (MIA) induced alterations on TLR3 pathway and on the two emerging endogenous antiinflammatory/antioxidant mechanisms NRF2/antioxidant enzymes pathway and the cytokine milieu regulating M1/M2 polarization in microglia.

Methods Pregnant mice were treated with the synthetic Toll-like Receptor 3 (TLR3) agonist Poly(I:C) in gestational day 9 and chronically treated with paliperidone (0,05 mg/kg i.p.) in adult offspring. Animals were sacrificed one day after treatment and behavioral test. Inflammation oxidative stress-related mediators were analysed at mRNA and protein level in prefrontal cortex samples. In addition, behavioral test t-maze was conducted.

Results Paliperidone prevented TLR3 pathway activation and the subsequent MIA-induced neuroinflammatory response. Also, paliperidone induced an increment in the activity and protein expression of nuclear NRF2, as well as increased mRNA levels of the antioxidant enzymes HO1, SOD and catalase in the MIA model. Otherwise, paliperidone increases the antiinflammatory cytokines levels TGF β and IL-10 in favour of a M2 microglia profile and increased the levels of the M2 cellular markers Arg1 and FOLR2.

Conclusions The modulation of neuroinflammation and enhancement of endogenous antioxidant/anti-inflammatory pathways by current and new antipsychotics could represent an interesting therapeutic strategy for the future.

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EW424

Psychosis among HIV-infected patients –a serious and complex association

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Introduction Psychosis represents an uncommon but serious complication in the course of HIV infection, and always requires a careful differential diagnosis.

Objectives To provide an overview of psychosis in HIV-infected patients.

Methods Literature review based on PubMed/MEDLINE, using the keywords “HIV” and “psychosis”.

Results Psychosis in HIV-positive individuals can be divided into psychotic disorders predating HIV infection and new-onset psychotic disorders in HIV-seropositive patients. The pathophysiology of psychosis in this population is complex and a multifactorial etiology is likely in most instances. The authors will analyze them and describe the differences of psychopathological pattern in first-episode psychosis between HIV-positive and HIV-negative patients. Antipsychotic agents are the treatments of choice regardless of the underlying diagnosis. However, they should always be used at the lowest possible dose for the shortest possible duration. Increased sensitivity to extrapyramidal reactions, high risk for dyslipidemia and hyperglycemia, potential interactions between HAART and some antipsychotic agents are also important considerations. Importantly, psychosis may be a harbinger of dementia. Cross-sectional studies have also suggested that psychosis may

adversely impact the morbidity and mortality associated with HIV-infection.

Conclusions Psychosis disorders may arise before or at any time during the course of HIV infection. A solid understanding of the complex relationship between psychosis and HIV allows for better evaluation and more effective treatment for psychotic individuals at risk for or infected with HIV. Thus, both HIV care programs and psychiatric care clinics should be made familiar with this important subject.

Disclosure of interest The authors have not supplied their declaration of competing interest.

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EW425

Neuroleptic effect in aggressive mice after the transplantation of immune cells treated in vitro with chlorpromazine

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Introduction Existence of integration, mutual relations of nervous and immune systems, which cellular elements are characterized by expressed phenotype and functional similarity, means the possibility of immune cells participation in the regulation of higher nervous activity.

Objectives Previously, we demonstrated the possibility of targeted regulation of animal's behavior by the transplantation of immune cells with definite functional characteristics. Based on the our previous research results in the present study, we investigated the modulating effect of the immune cells, treated in vitro with chlorpromazine on the nervous and immune systems functional activity in aggressive mice.

Methods (CBA \times C57Bl/6) F1 aggressive mice, exposed to 10-days chronic social stress, were undergoing the transplantation of immune cells in vitro treated with chlorpromazine. Animal's behavioral parameters, cytokines synthesis in the brain and immune cells before and after transplantation were estimated.

Results It was shown that aggression is associated with the increased production of spleen T-helper 1 cell-derived cytokines IL-2 and IFN γ , as well as decreased TNF α production by the spleen mononuclear phagocyte cells. These alterations were more pronounced following mitogen stimulation. Spleen cells, obtaining from aggressive mice, were treated in vitro with chlorpromazine and then injected intravenously into syngeneic aggressive recipients. The cell's transplantation led to the reduction of the recipient's motor activity in the “open field” and Porsolt swimming tests and normalized cytokines synthesis in the brain and immune cells.

Conclusion Research results demonstrated the neuroleptic effect in aggressive mice, obtained by the transplantation of immune cells treated in vitro with chlorpromazine.

Disclosure of interest The authors have not supplied their declaration of competing interest.

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EW426

Impact of anti-inflammatory drugs on the risk of anxiety disorders after critical illness

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