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Introduction: According to recent research violations of the oxidative-antioxidant balance may play an important role in the pathogenesis of schizophrenia, by changing generate, conduct and reproduce a nerve impulse. Antibodies with oxidoreductase activity may be involved in protection against oxidative stress in schizophrenia.

Objectives: The study was to compare the superoxide dismutase (SOD) activity of IgG in patients with acute schizophrenia and during remission.

Methods: The study included 20 patients with acute schizophrenia (mean PANSS total score $94,3 \pm 14$), 18 people with schizophrenia during remission (mean PANSS total score $54,7 \pm 9$), and 10 healthy individuals. All participants signed informed consent for the research. Isolation of IgG from blood serum was performed using affinity chromatography on Protein-G-Sepharose columns. The homogeneity of the substances is confirmed by the SDS PAGE method. SOD activity of IgG was carried out spectrophotometrically. Statistical processing was conducted with Statistica v.10.

Results: IgG of schizophrenia patients and healthy individuals have a SOD activity and studied activity is proved to be antibodies intrinsic property. The activity of antibodies in acute schizophrenia was 1.7 times higher than in healthy individuals ($p < 0.05$). In patients with schizophrenia during remission SOD activity of IgG was 2.4 times higher than in healthy individuals ($p < 0.05$).

Conclusions: We can assume that in the conditions of oppression antioxidant activity in schizophrenia patients, antibodies partially take over the function of protecting the body from patients with generalized oxidative stress. *This work is supported by the Russian Scientific Foundation, grant # 18-15-00053P.*

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Keywords: oxidative stress; schizophrenia; IgG

EPP0497

Molecular diagnosis of cytogenetic abnormalities in patients with schizophrenia.

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Introduction: Schizophrenia is a severe and chronic disorder causing significant disability and functional decline. Schizophrenia is a polygenic disease, with about 100 monogenic sites and 11 sites of chromosomal deletions / duplications involved in its pathogenesis identified. It is a pleiotropic disease, with causative genetic changes leading to multiple symptoms, including bipolar disorder, autism spectrum disorders, ADHD, mental retardation and

epilepsy. The chromosomal microarray (CMA) technology detects submicroscopic chromosomal changes, which are involved in neurodevelopmental disorders, and are subject to prenatal diagnosis. Pathological findings in CMA are detected in 10–20% of patients with neurodevelopmental disorders and can contribute significantly to medical follow-up, prognosis assessment, influence treatment choice, and allow prenatal diagnosis. Preliminary studies in schizophrenia identified pathological CMA findings in 10–30% of patients.

Objectives: CMA testing of schizophrenia patients to detect genetic changes causing the disease.

Methods: Recruitment of schizophrenia patients from the Haifa and Western Galilee districts of Clalit, genetic counseling in Carmel Hospital, CMA testing of the consenting patients.

Results: Schizophrenia patients with and without neurodevelopmental disorders underwent CMA analysis, with the findings of significant chromosomal submicroscopic disorders (such as 22q11 microdeletion, among others) in 30% of the patients, providing the explanation for the patients' symptoms and enabling specific medical follow-up and adjusted pharmacological treatment.

Conclusions: CMA can be used in diagnosing schizophrenia, assessing prognosis, adjusting pharmacological treatment and follow-up and providing genetic counseling including prenatal diagnosis, as in cases neurodevelopmental disorders. The findings support the application of CMA as part of a routine procedures in schizophrenia.

Disclosure: No significant relationships.

Keywords: Schizophrenia; Chromosomal abnormalities; CMA; diagnosis

Comorbidity/Dual Pathologies / Consultation Liaison Psychiatry and Psychosomatics 02

EPP0498

Polymorphism rs1108580 in DBH gene is associated with the risk of depression in alcohol-dependent patients

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Introduction: Alcohol dependence and depression are often combined, patients with comorbid pathology have a more severe course of the disease, a high risk of suicide and therapeutic resistance. Enzyme dopamine-beta-hydroxylase (DBH) is a key player in a link between dopamine and norepinephrine neuromediators and may be involved in alcohol dependence and depression comorbidity and genetic markers in DBH gene may be associated with the risk of comorbid state.

Objectives: To test an association of DBH gene polymorphisms rs161111580 and rs1108580 with depression risk in alcohol-dependent patients.

Methods: Our sample consisted of 104 inpatients diagnosed by ICD-10 criteria: 40 with alcohol dependence (AD group) (age 45.6 (SD 10.853), 5% females), 64 with depression and alcoholism