Biomedicine, Tomsk, Russian Federation and [3]Mental Health Research Institute, Tomsk National Research Medical Center of the Russian Academy of Sciences, Department Of Endogenous Disorders, Tomsk, Russian Federation
*Corresponding author.
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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±14), 18 people with schizophrenia during remission (mean PANSS total score 54,7±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.*

Disclosure: No significant relationships.
Keywords: oxidative stress; schizophrenia; IgG

EPP0497

Molecular diagnosis of cytogenetic abnormalities in patients with schizophrenia.

M. Bar Shai[1]*, A. Peleg[2] and T. Zalozhin[2]

[1]Carmel Medical Center, Clalit Healthcare Service, Psychiatry, Genetics, Haifa, Israel and [2]Genetics, Carmel Medical Center, Clalit Healthcare Service, Haifa, Israel, Genetics, Carmel Medical Center, Clalit Healthcare Service, Haifa, Israel, Haifa, Israel
*Corresponding author.
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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; Cromosomal 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

A. Nikolishin*, N. Chuprova and A. Kibitov

Serbsky National Medical Research Center on Psychiatry and Addictions, Molecular Genetics Laboratory, Moscow, Russian Federation
*Corresponding author.
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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 neuromediations 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

comorbidity (AD+D group) (age 41.2 (SD 9.903), 22% females) and 112 healthy controls (age 35.5 (SD 8.286), 15% females). rs1108580 and rs1611115 were detected by RT-PCR.
Results: For rs161111580, frequencies of minor T allele (p=0.031) and TT genotype (p=0.017) was higher, CC genotype (p=0.042) was lower in AD group vs. controls. rs161111580 T allele and TT genotype increases the risk of AD (OR=3.715, 95%CI [1.728-7.986], P=0.001 and OR=4.009, 95%CI [1.502-10.699], P=0.006). For rs161111580, frequency of TT genotype (p=0.009) was higher in AD+D group vs. controls. For rs1108580, frequency of major A allele (p=0.059, trend) was higher in AD+D, then in AD group. Major A allele rs1108580 increases the risk of depression in alcohol-dependent patients (OR=2.74, 95%CI [1.283-5.855], P=0.001).
Conclusions: It was shown that the DBH rs1108580 increases the risk of depression in patients with alcohol dependence.

Disclosure: No significant relationships.
Keywords: Alcohol dependence; Dopamine; Genetics; Depression

EPP0501

Symptoms of diabetes distress, depression, and anxiety in people with type 2 diabetes: identifying central and bridge symptoms using network analysis

A. Mcinerney[1]*, N. Lindekilde[2], A. Nouwen[3], N. Schmitz[4] and S. Deschenes[1]

[1]University College Dublin, Psychology, Dublin, Ireland; [2]University of Southern Denmark, Psychology, Odense, Denmark; [3]Middlesex University, Psychology, London, United Kingdom and [4]University of Tübingen, Medicine, Tubingen, Germany
*Corresponding author.
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Introduction: People with diabetes are vulnerable to diabetes-related distress and are more likely to experience depressive and anxiety symptoms than the general population. Diabetes distress, depressive, and anxiety symptoms also tend to commonly co-occur.
Objectives: This study aimed to apply network analysis to explore the associations between diabetes distress, depressive, and anxiety symptoms in a cohort of adults with type 2 diabetes.
Methods: Data were from the baseline (2011) assessment of the Evaluation of Diabetes Insulin Treatment (EDIT) study ($N = 1,796$; 49% female; mean age = 60, $SD = 8$) from Quebec, Canada. A first network using the 17 items of the diabetes distress scale (DDS-17) was estimated. A second network was estimated using the 17 items of the DDS-17, the 9 depressive items of the PHQ-9, and the 7 anxiety items of the GAD-7. Symptom centrality, network stability, and bridge symptoms were examined.
Results: Regimen-related and physician-related distress symptoms were amongst the most central (highly connected) in the diabetes distress network. *Worrying too much* (anxiety), *Not feeling motivated to keep up diabetes self-management* (diabetes distress), and *Feeling like a failure* (depression) were the most central symptoms in the combined network. *Feeling like a failure* (depression) was highly connected to diabetes distress symptoms, representing a potential bridge between diabetes distress and depression.
Conclusions: Identifying central and bridge symptoms may provide new insights into diabetes distress, depressive, and anxiety symptom maintenance and comorbidity in people with type 2 diabetes.

Disclosure: No significant relationships.
Keywords: Network Analysis; comorbidity; diabetes; diabetes-distress

EPP0502

Evaluation of the role of lisdexamfetamine on attention-deficit/hyperactivity disorder common psychiatric comorbidities: mechanistic insights on binge eating disorder and depression

J.R. Gutiérrez Casares[1]*, J. Quintero[2], P. Rodríguez[3], C. Montoto[3], T. Pozo-Rubio[3], C. Segú-Vergés[4] and M. Coma[5]

[1]Hospital Perpetuo Socorro, Unidad Ambulatoria De Psiquiatría Y Salud Mental De La Infancia, Niñez Y Adolescencia, Badajoz, Spain; [2]Hospital Universitario Infanta Leonor. Universidad Complutense., Servicio De Psiquiatría, Madrid, Spain; [3]Takeda Farmacéutica España, Medical Department, Madrid, Spain; [4]Anaxomics Biotech, Molecular Health Department, Barcelona, Spain and [5]Anaxomics SL, Molecular Physiology, Barcelona, Spain
*Corresponding author.
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Introduction: Attention-deficit/hyperactivity disorder (ADHD) is a psychiatric condition in which children suffer from inattentiveness, hyperactivity, and or impulsivity. ADHD patients frequently present comorbid psychiatric disorders: in adults, the most common are depression, substance-related disorders, anxiety, and eating disorders. Children and adolescents present conduct disorders, learning disorders, anxiety and depression. Since ADHD and its psychiatric comorbidities share similarities, a partial overlap of their pathophysiological mechanisms has been suggested. ADHD, can be treated with lisdexamfetamine (LDX), a prodrug indicated by the FDA as treatment for binge eating disorder (BED) and ADHD.
Objectives: To evaluate, through a systems biology-based *in silico* method, the efficacy of LDX as first-line ADHD treatment to improve ADHD psychiatric comorbidities. Furthermore, we explored the molecular mechanisms behind LDX's action.
Methods: We used the systems biology- and artificial intelligence-based Therapeutic Performance Mapping System (TPMS) technology to characterise and model ADHD comorbidities. Artificial neural networks (ANNs) algorithms were used to identify specific relationships between protein sets. Finally, we modelled the mechanisms of LDX for the most relevant comorbidities by using sampling methods and comorbidity-specific virtual patients in each case.
Results: This study predicts a strong relationship between LDX's targets and proteins involved in BED and depression (Fig 1). Our results could be explained not only by LDX role in neurotransmitter regulation, but also by modulation of neuroplasticity (BDNF/NTRK2, GSK3), neuroinflammation (interleukins, inflammasome), oxidative stress (NOS2, SOD), and the hypothalamic-pituitary-adrenal (HPA) axis (CRH, CRHR1).