EPP0453

The role of miRNA in diagnosing and clarifying the pathomechanisms in major depressive disorder

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Introduction: There are currently no diagnostic or treatment-guiding biomarkers for major depressive disorder (MDD). Micro-ribonucleic acids (miRNA) may facilitate understanding the reorganisation of gene expression networks in MDD. Identifying miRNA and target mRNA pathways that contribute to MDD may open new therapeutic avenues, such as inhibiting endogenous miRNA or administering exogenous miRNA.
Objectives: This study investigates how miRNAs can clarify the molecular mechanisms of MDD by comparing the miRNA levels in the blood serum of patients with MDD and healthy individuals. The study also investigates the discriminative ability of miRNAs to distinguish between depressed patients and healthy controls.
Methods: Sixty depressed patients were matched with 60 healthy controls based on age, gender, ethnicity, and years of education. The severity of depression was measured using the Hamilton Depression Rating Scale, and venous blood was collected for miRNA profiling. Using the QIAGEN Ingenuity Pathway Analysis, networks were constructed to identify the biological pathways associated with MDD influenced by the differentially expressed miRNAs. Analyses of the receiver operating characteristic (ROC) were performed to examine the capacity of miRNAs to distinguish between depressed and healthy individuals.
Results: Six miRNAs (miR-542-3p, miR-181b-3p, miR190a-5p, miR-33a-3p, miR-3690, and miR-6895-3p) were significantly down-regulated in untreated depressed patients compared to healthy controls. miR-542-3p has experimentally validated mRNA targets predicted to be associated with MDD. ROC analyses determined that a panel containing miR-542-3p, miR181b-3p, and miR-3690 distinguished between depressed and healthy individuals with an area under the curve value of 0.67.
Conclusions: Specific miRNAs, including miR-542-3p, miR181b-3p, and miR-3690, may be biomarkers with targets implicated in the pathophysiology of depression. They could also be used to distinguish accurately between depressed and healthy individuals.

Disclosure of Interest: None Declared

EPP0454

Association of rs11644461 GRIN2A with clinical phenotype of schizophrenia

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Introduction: The glutamatergic system plays an important role in the neurobiology of schizophrenia. A lot of number of variants in the GRIN genes have been found in patients with various neuropsychiatric disorders (Myers et al. F1000Res 2019; 8(F1000 Faculty Rev) 1940). GluN2A, encoded by the GRIN2A gene, is the most abundant of the GluN2 NMDA receptor subunits in the mammalian CNS. Clinical symptoms of schizophrenia vary among individuals. The GRIN2A gene has previously been shown to be associated with early onset schizophrenia (Poltavskaya et al. Life (Basel) 2021; 11(10) 997).
Objectives: The aim of the study was to identify associations of the GRIN2A gene rs11644461 polymorphism with features of the course of schizophrenia.
Methods: This study was carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki 1975). 805 patients with schizophrenia (ICD-10: F20) were included. Clinical examination and diagnostic evaluation were performed using the Positive and Negative Syndrome Scale (PANSS). From the general group of patients, 2 subgroups were distinguished according to the PANSS survey: 391 patients with leading negative symptoms and 414 patients with leading positive symptoms. Also 2 subgroups were distinguished from the general group of patients: 398 patients with a continuous course of schizophrenia and 257 patients with episodic schizophrenia. Genotyping was performed by real-time PCR.
Results: An association of the C rs11644461 GRIN2A allele with the continuous course of schizophrenia was revealed (p<0,047). The rs11644461 polymorphism was not associated with the leading symptoms of the disease (positive or negative). At the same time, the values of the total score on the PANSS scale differed statistically significantly in carriers of different genotypes for this polymorphism. The sum of PANSS scores (Me [Q25 – Q75]) in carriers of the TC rs11644461 genotype was statistically significantly higher (106 [92–113]) than in carriers of the CC genotype (101 [87–108]) (p=0.006).
Conclusions: According to the results obtained, carriers of the TC rs11644461 GRIN2A genotype have a higher severity of schizophrenia symptoms according to the PANSS scale than carriers of the CC genotype. Also in the present study, it was shown that the C allele rs11644461 GRIN2A is associated with the continuous course of schizophrenia, which indicates the contribution of this locus to the formation of the course of the disease.

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EPP0455

Noncoding de novo mutations contribute to autism via long-range chromatin interactions

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