Endonucleases, alkaline and acidic DNase activities were determined spectrophotometrically from homogenates of brain tissue. **Results:** Copper is believed to be a likely cause of oxidative damage to the DNA molecule, as manifested by increased alkaline and acidic DNase activity. The results of this study show that GSH is a potent chelator that binds copper and enables its elimination from the body. **Conclusions:** In this experiment, the beneficial role of GSH supplements, which has an antioxidant character, in the prevention and reduction of the adverse effects of chronic copper intoxication was demonstrated. In this way, GSH acts as a powerful protector and antioxidant.

Disclosure: No significant relationships. **Keywords:** DNase; Copper; Brain of rats; glutathione

EPV0338

Investigation of the glucocorticoid receptor co-chaperone FKBP5 in individuals with first-episode psychosis

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Introduction: Stress has been associated with the onset and progression of neuropsychiatric conditions. The neuroendocrine response to psychosocial stressors is mediated via the hypothalamus-pituitary-adrenal axis, resulting in systemic glucocorticoid secretion. FKBP5 is a co-chaperone of the cortisol-bound glucocorticoid receptor. FKBP5 Single Nucleotide Polymorphisms (SNPs) may indicate stress-response alterations, thus affecting vulnerability or resilience to neuropsychiatric phenotypes.

Objectives: To investigate the FKBP5 polymorphism rs1360780 and FKBP5 mRNA levels in a well-characterized, drug-naïve sample of First-Episode Psychosis (FEP) individuals and matched controls. **Methods:** For genotyping rs1360780, whole blood DNA was extracted from FEP individuals and matched controls. The presence of the C (protective) \rightarrow T (risk) alleles was assessed using TaqMan SNP genotyping assay. Peripheral Blood Mononuclear Cells (PBMCs) were isolated and whole RNA was extracted. FKBP5 mRNA levels were detected with RT-qPCR, using SYBRgreen. Results were normalized against the 18s rRNA reference gene. Statistical analysis was performed in GraphPad Prism 8.

Results: The distribution of $C \rightarrow T$ alleles of rs1360780 genotyped in FEP (N=44) and controls (N=39) indicate a statistically significant prevalence of the C/C alleles in FEP individuals (*p=0.0432). mRNA FKBP5 data revealed increased levels of FKBP5 in FEP individuals (N=25) compared to controls (N=18), (***p=0.0007). **Conclusions:** Our data show increased FKBP5 mRNA levels in FEP individuals compared to matched controls, as well as the presence of the rs1360780 protective (C) allele. Follow up studies include investigation of the translational profile of stress-mediators, in order to pave an individualized approach towards deciphering psychosis onset pathobiology.

Disclosure: No significant relationships. **Keywords:** glucocorticoids; FKBP5; stress; First episode psychosis

EPV0339

The psychiatric phenotype of 15q11.2-q13.3 duplications

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Introduction: 15q11.2-q13.3 region is prone to genomic rearrangements leading to both deletions and duplications. A wide spectrum of neuropsychiatric conditions, such as developmental delay/ intellectual disability (DD/ID), autism, attention-deficit hyperactivity disorder, schizophrenia, epilepsy was reported in association with genomic imbalances of this region.

Objectives: In this paper we report on 9 children carrying 15q11.2q13.3 duplications.

Methods: Seven boys and two girls, aged 15 months to 15 years, were included in the study. Genomic investigations were carried out by array-based comparative genomic hybridization (Agilent Technologies). In all patients the psychomotor development, dysmorphic features, neuroimaging and EEG anomalies were assessed. Psychologic and psychiatric evaluation was performed with specific tests.

Results: The size of the duplications ranged from 9.65 Mb to 0.38 Mb. All patients presented speech delay. Autistic behavior and muscular hypotonia were detected in 8 out of 9 patients, DD/ID in 6. Two children presented epileptic seizures, in addition 4 other children had EEG anomalies. Facial dysmorphic features were observed in 5 patients. Neuroimaging studies showed anomalies in 4 children. The smallest region of overlap in our patient group harbors CHRNA7 gene, a candidate for the behavioral abnormalities.

Conclusions: 15q duplications encompassing CHRNA7 gene were associated with different neuropsychiatric features in our patients. Our results further support the association of 15q duplications with neuropsychiatric phenotypes, with clinical heterogeneity and variable severity, which is yet to be explained. Acknowledgment: The research leading to these results has received funding from the EEA RO NO Grant 2014-2021, the project contract No 6/2019.

Disclosure: No significant relationships.

Keywords: intellectual disability; autism; 15q11.2-q13.3 duplications; phenotype