e-Poster viewing: Genetics & molecular neurobiology

EV0586

Adult with autism – oxidative stress, co-morbidity and predisposition

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Introduction The etiology of autism spectrum disorder (ASD) is unclear. Studies involving children with ASD suggest that oxidative stress could explain some of the pathology. Few reports have investigated the role of oxidative stress into adulthood. Furthermore, the knowledge on psychiatric and somatic co-morbidities, as well as socio-economic status in a trajectory across lifespan is sparse.
Objectives Investigating oxidative stress related markers in ASD, along with trajectories in socio-economic functioning and co-morbidities.
Aims To evaluate the importance of oxidative stress in the neurobiology of adults with ASD and assess the socio-economic level of functioning and co-morbidities.
Methods Plasma levels of antioxidant super-oxide-dismutase isoenzymes (SOD1 and SOD2) and pro-oxidant xanthineoxidase (XO) were measured in 56 patients ≥18 years of age, diagnosed in childhood with ASD (F84.0, F84.1, F84.5 or F84.8), along with gender and age matched controls. Participants were interviewed regarding their health, familial predisposition and social status.
Results Cases showed higher levels of SOD1 (268.2 ng/mL vs. 205.6 ng/mL). We found no differences regarding SOD2 and XO. Patients had a higher BMI (27 vs. 24), fewer drank alcohol (40% vs. 75%), more had a psychiatric co-morbidity (50% vs. 2%), more had family member with a psychiatric diagnosis (80% vs. 50%). None of the bio-psycho-social factors showed association with biomarker levels.
Conclusion Oxidative stress seems to play a role in ASD. Furthermore, patients with ASD often have psychiatric co-morbidities; more often have a family history of psychiatric diagnoses, and are less healthy physically.
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Evaluation of serum microRNA expression profile in panic disorder patients

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Introduction Even though it has begun to be investigated in recent years, studies of microRNA (miRNA) in anxiety disorders are limited. Our research is the first miRNA expression study in panic disorder, which excludes of drug use and additional psychiatric disorders.
Objective We aimed to determine the availability of miRNAs as biomarkers in the serum levels of panic disorder and to demonstrate the changing expression of miRNAs.
Methods In the research, 35 panic disorder patients and 35 healthy controls were administered a socio-demographic and clinical information form, SCID-I, PDSS. 2 tubes of peripheral venous blood were taken from each group for genetic evaluation. miRNA expression analysis was performed in those samples by the RT-PCR method.
Results Compared with the healthy control group, 8 miRNA expression levels were found different in panic disorder group. Five of them were up-regulated and 3 of them were down-regulated. There was no correlation between the level of miRNA expression and PDSS total score and PDSS sub-items. miR-1297 and miR-4465 expression levels were statistically significant between the two groups. Both miRNAs are also known to arrange the gene regions that affect $GABA_A$ receptor subtypes.
Conclusions miR-1297 and miR-4465 regulate the GABAA gene that is thought to play a role in the etiology of panic disorder (Wong et al., 2014, Wang 2016). In panic disorder group, miR-1297 and miR-4465 expression levels were found to be up-regulated from the healthy control group.
Keywords Panic disorder; miR-1297; miR-4465; GABA
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SHANK3 mutation in consanguineous schizophrenia family in northwest Algeria

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Introduction Several studies have asserted the existence of a strong and complex genetic component in the determination of psychotic disorders. GWAS studies conducted over the past decade lead to the identification of only a few low effect associations, calling questioning the hypothesis of "common disease – common variants" for a model involving a large number of rare variants.
Aims Here, we studied a multigenerational multiplex family with schizophrenia a high rate of consanguinity, located in the northwest of Algeria. This study aims to identify inherited rare variants of schizophrenia using new genetic technologies.
Methods This family has received complete clinical (DIGS, DSM-IV criteria), genealogical investigations, CNV analysis using CGH Microarray Kit 244 K (Santa Clara, CA) and WES (by GAIIx Illumina/HiSeq 2000) focused in CNV regions, that were performed in the department of genetics in the university hospital of Geneva.
Results We identify 11 affected members by psychotic disorders. The main CNVs analysis results found in a schizophrenic member a Del 22q13.33 affecting SHANK3 gene. WES regarding these regions identified a mutation at position 511178000 in SHANK3 gene in all the selected affected relatives.
Discussion Several studies have asserted the association of SHANK3 mutations with schizophrenia and autism disorders. This is the first observation of rs511,178,000 in schizophrenia phenotype.
Conclusion In total, this highly informative family have identified new rare genetic variant of schizophrenia. The search for this mutation in wider control population in would be useful to validate these data.