e-Poster viewing: Genetics & molecular neurobiology

EV0586

morbidities.

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

M. Bækgaard Thorsen ^{1, 3}, N. Bilenberg ², P. Munk-Jørgensen ¹, Å. Fex Svenningsen ³, N. Heegaard ⁴, T. Maria Michel ¹

¹ Institute of Clinical Research, University of Southern Denmark, Research Unit of Psychiatry, Odense, Denmark

- ² Institute of Clinical Research, University of Southern Denmark,
 Research Unit of Child, and Adolescent Psychiatry, Odense, Denmark
 ³ Institute of Molecular Medicine, University of Southern Denmark,
 Department of Neurobiology Research, Odense, Denmark
- ⁴ Institute of Clinical Research, University of Southern Denmark, Research Unit of Clinical Biochemistry, Odense, Denmark * Corresponding author.

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-

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.

Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.916

EV0587

Evaluation of serum microRNA expression profile in panic disorder patients

F.P. Çökmüş^{1,*}, E. Özmen¹, T. Alkın², M.B. Batır³, F.S. Çam³

¹ Celal Bayar University School of Medicine, Psychiatry, Manisa, Turkey

- ² Dokuz Eylül University School of Medicine, Psychiatry, Izmir, Turkey ³ Celal Bayar University School of Medicine, Medical Genetic, Manisa, Turkey
- * Corresponding author.

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 GABAA 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 Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.917

EV0588

SHANK3 mutation in consanguineous schizophrenia family in northwest Algeria

A. Dahdouh 1,*, J. Prados 2, M. Guipponi 2, F. Bena 2, W. Adouan 2, S. Antonarakis 2

- ¹ University of Oran, department of psychiatry, Oran, Algeria
- ² University of Geneva, department of medical genetics, Geneva, Switzerland
- * Corresponding author.

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.

Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.918

EV0589

Genetic determinants of psychic resilience after a diagnosis of cancer

M. Christensen¹, A. Drago²,

- ¹ Via University College, Via University College, Holstebro, Denmark
- ² Psykiatrisk Forskningsenhed Vest, Department of Clinical Medicine, Aarhus University, Herning, Denmark
- * Corresponding author.

Introduction Co-morbidity between cancer and psychiatric disorders including adjustment disorder, depressive disorders or angst can seriously influence the prognosis and the quality of life of patients.

Aim The identification of the psychological and biological profile of patients at risk for such co-morbidity is not yet available. Classical candidate genes such as the BDNF, the 5-HTLPR and genes whose products are involved in inflammatory events have received some attention, but results are inconclusive.

Object and methods In the present review the association between cancer and psychiatric disorders is reviewed, a focus on the investigation of the Gene X environment and the epigenetic control over the activation of the HPA axis is proposed as a tool to refine the definition of the biologic profile at risk for co-morbidity between psychiatry and cancer.

Results and conclusion A number of genes and socio-demographic variables that may influence risk to suffer from a psychiatric disorder after a diagnosis of cancer is identified and discussed. The identification of such biologic and socio-demographic profile is instrumental in the identification of subjects at risk of a double diagnosis, both somatic and psychiatric. An early identification of such profile risk would pave the way to the implementation of early intervention strategies.

Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.919

EV0590

Is 22q11.2 deletion syndrome a genetic subtype of schizophrenia?

R. Hernandez Anton ¹,*, H. De La Red Gallego ¹, M. Gomez Garcia ¹, A. Alonso Sanchez ¹, E. Mayor Toranzo ¹, J.A. Blanco Garrote ¹, M. De Lorenzo Calzon ¹, M. Hernandez Garcia ¹, E. Dominguez ², F. Uribe Ladron De Cegama ¹, V. Molina Rodriguez ¹

- ¹ Hospital Clinico Universitario Valladolid, Psiquiatria, Valladolid, Spain
- ² Hospital La Mancha Centro, Psiquiatria, Alcazar De San Juan, Spain
- * Corresponding author.

Introduction 22q11.2 deletion syndrome is a primary immunodeficiency due to micro-deletion on the large arm of chromosome 22. Patients suffer from several anomalies, including metal illness, that such the case we present, mean a warning sign for further study.

Methods Twenty-one years-old male, with psychotic symptoms, typical of schizophrenia, behavioral disorders and mental confusion, plus epileptic episodes and psychomotor agitation. Two previous incomes with the diagnosis of psychotic disorder not otherwise specified. Treated with anti-psychotics at low doses with inter-episode stability.

Background Prematurity, low birth weight, neonatal asphyxia, generalized seizures, otitis and recurrent urinary tract infections, hypernasal voice, poor academic performance, difficulty relating. Physical examination: hypernasal voice, furred tongue, dysmorphic

faces, scoliosis, hipotanía, stereotypes, delusions, auditory hallucinationsd negative symptoms.

Results We considered the possibility of a neurodevelopmental disorder, with a multidisciplinary approach, resulting in the diagnosis of paranoid schizophrenia and velocardiofacial syndrome, which had gone unnoticed. Mean doses of clozapine, haloperidol and topiramate were used. He accepted psychiatry and other specialties follow-up, since it requires a complex and multidisciplinary approach.

Conclusions Definition of velocardiofacial Syndrome and lack of consensus on terminology:

- syndrome 22q11.2 DS as genetic subtype of schizophrenia? Opportunity to study the pathogenesis of schizophrenia;
- the importance of a comprehensive approach to early diagnosis, clinical improvement and preventing complications.

Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.920

EV0591

The genetic study of computer game addiction

D. Mavani ¹,*, V. Soldatkin², E. Mashkina ³, A. Dyachenko ¹, E. Karpova ¹, O. Bukhanovskaya ¹

¹ Scientific Center for Treatment and Rehabilitation "Phoenix", Psychiatry department, Rostov-on-Don, Russia

² Rostov State Medical University, Psychiatry department, Rostov-on-Don, Russia

 3 South Federal University, Genetics department, Rostov-on-Don, Russia

* Corresponding author.

Introduction Addiction to computer games (CA) is growing with a lightning speed in whole world. Very few studies are focused in the genetic basis of this disorder.

Objectives To study the COMT and MAOA polymorphism in addicts to computer games.

Methods Totally 42 persons were included in this study, 22 of them had CA and 20 were totally healthy. Out of 22 gamers, 10 persons had only CA. The rest of 12 patients suffered from another psychiatric disorder besides of CA (Schizotypal disorder, depression, bipolar disorder). Their mean age was 16 years (15; 17) and all of them were males.

Results The total frequency of alleles 3R and 5R of MAOA gene in patients with CA was 30.0%, which doesn't have any statistical difference with the healthy persons. The genotype frequency of Val158Met of COMT gene is high in CA rather than in healthy persons ($\chi^2 = 6.85$, P = 0.03). Also, the homozygotes Val are much more in CA patients (59.1%) than in healthy persons (25%). On the other hand, the Val/Met combination is lower in CA patients (18.2%) than in healthy persons (55.0%).

Conclusion The Val158Met polymorphism of gene COMT may lead to CA formation.

Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.921

EV0592

Family-based association study between the brain derived neurotrophic factor (bdnf) gene and the attention deficit hyperactivity disorder in a Mexican population

J.P. Sánchez de la Cruz^{1,*}, A. López López², C.A. Tovilla Zárate¹, R. Molina Sólis², A. Valencia Hernández², L. Gómez Valencia², M.M. Rivera Angles², M.L. López Narváez³, D.Y. Bermúdez Ocaña ¹