

protein folding were also significantly associated but accounted for less than 1% of the variance. With APOE excluded, all pathways remained significant except proteasome-ubiquitin activity and protein folding.

Conclusions Genetic risk for LOAD can be split into contributions from different biological pathways. These offer a means to explore disease mechanisms and to stratify patients.

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

<http://dx.doi.org/10.1016/j.eurpsy.2017.01.2052>

EW0184

Peripheral levels of the micro-RNA miR-1202 are correlated with changes in brain activity and connectivity during an antidepressant treatment

J.P. Lopez^{1,2}, F. Pereira³, S. Richard-Devantoy⁴, Y. Ding⁴, L.M. Fiori⁴, P. Niola⁵, G. Turecki⁴, F. Jollant^{4,6,*}

¹ Max Plank Institute, Stress Neurobiology and Neurogenetics, Munich, Germany

² Human genetics department, McGill University, Montreal, Canada

³ Radiology department, Nîmes Academic Hospital, Nîmes, France

⁴ Psychiatry department, McGill University, Montreal, Canada

⁵ Biomedical Science department, Università degli studi Di Cagliari, Cagliari, Italy

⁶ Psychiatry department, Nîmes Academic Hospital, Nîmes, France

* Corresponding author.

Introduction Micro-RNAs are short non-coding sequences playing a major role in regulating gene expression. Peripheral levels of the micro-RNA miR-1202 have been shown to predict antidepressant response and to change during treatment. However, it is not clear to what extent these peripheral measures reflect central neural changes in vivo.

Objectives We aimed at investigating a potential link between peripheral micro-RNA and neuroimaging measures.

Methods At baseline and after 8 weeks of desvenlafaxine (50–100 mg die), twenty depressed patients were scanned with 3 T magnetic resonance imaging, first at rest then during the Go/NoGo task, a classical test of response inhibition. Blood samples were taken for RNA extraction.

Results During resting state, baseline miR-1202 levels were predictive of decreased connectivity between the posterior cingulate and the prefrontal, occipital and parietal cortices. Changes in miR-1202 levels were correlated with changes in activity in right precuneus within the default-mode network, and with decreased connectivity between the posterior cingulate and the temporal and prefrontal cortices, and the precuneus. During the Go/NoGo task, baseline levels and changes in these levels were correlated with activity changes in different regions, including bilateral prefrontal, insular, cingulate, and temporal cortices. Finally, secondary analyses suggest an association between miR-1202 levels and glutamate levels measured by spectroscopy in dorsomedial prefrontal cortex.

Conclusions This is the first study showing that baseline and changes in peripheral levels of one micro-RNA were associated with changes in brain activity and connectivity during an antidepressant treatment. MiR-1202 may act through the modulation of the glutamatergic system.

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

<http://dx.doi.org/10.1016/j.eurpsy.2017.01.2053>

EW0185

Concomitant 3q13.31 microdeletion and ring chromosome 22 in a patient with severe developmental delay,

ventriculomegaly, and Dandy-walker malformation

A. Kashevarova^{1,2,*}, E. Belyaeva³, N. Skryabin^{1,2}, S. Vasilyev^{1,2}, M. Lopatkina¹, L. Nazarenko³, A. Nikonov⁴, I. Lebedev^{1,2}

¹ Research Institute of Medical Genetics- Tomsk National Research Medical Center- Russian Academy of Sciences, Laboratory of Cytogenetics, Tomsk, Russia

² Tomsk State University, Laboratory of Human Ontogenetics, Tomsk, Russia

³ Research Institute of Medical Genetics, Tomsk National Research Medical Center, Russian Academy of Sciences, Laboratory of Hereditary Pathology, Tomsk, Russia

⁴ Diagnostic Center of Altai Region, Medical and Genetic Clinic, Barnaul, Russia

* Corresponding author.

Introduction Over 20% of patients with developmental delay (DD) has copy number variations (CNV) of unknown significance. Some CNV may be associated with disease in a patient and also present in their apparently healthy parents. According to the two-hit model another CNV may contribute to phenotypic variation of such genomic disorders.

Objectives DD diagnostics improvement.

Aims Understanding the pathogenic significance of concomitant 3q13.31 and 22q13.32–q13.33 microdeletions.

Methods Ring chromosome 22 was first detected by conventional cytogenetics. Microdeletions at 3q13.31 and 22q13.32–q13.33 were revealed by agilent technologies 60 K microarray and confirmed by qPCR. Ring chromosome was confirmed by FISH.

Results We present a four-year-old girl with del22q13.32–q13.33 resulted in a ring chromosome 22 and a single TUSC7 gene microdeletion at 3q13.31. The del22q13.32–q13.33 originated de novo, whereas del3q13.31 was inherited from healthy mother. The 22q13.32–q13.33 locus is associated with Phelan-McDermid syndrome (PHMDS, OMIM 606232). The patient demonstrated features both typical for the syndrome (psychomotor and speech development delay, autistic signs, aggression, sleep alteration, seizures) and atypical – attention deficit-hyperactivity disorder (ADHD), ventriculomegaly, and reduced size of cerebella hemispheres (Dandy-Walker variant). ADHD and ventriculomegaly were previously described in patients with del3q13.31 (OMIM 615433) but Dandy-Walker variant was observed in our patient for the first time. Possibly, atypical for PHMDS features, may result from trans-epistasis of microdeletions.

Conclusions Multiple CNVs in one patient complicate genotype-phenotype correlations due to possible overlapping phenotypes and/or modifying effect of variants. This study was supported by Russian Science Foundation, grant no. 16-15-10231.

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

<http://dx.doi.org/10.1016/j.eurpsy.2017.01.2054>

EW0186

CYP450 enzymes genetic polymorphism influence on treatment of affective disorders

A. Lengvenytė^{1,*}, R. Strumila², E. Długauskas³, A. Utkus⁴

¹ Clinic of Psychiatry, Vilnius University, Faculty of Medicine, Vilnius, Lithuania

² Vilnius University, Faculty of Medicine, Vilnius, Lithuania

³ Clinic of Psychiatry, Department of Psychiatry, Center of Neurology, Vilnius University, Vilnius, Lithuania

⁴ Department of Human and Medical Genetics, Vilnius University, Center for Medical Genetics, Vilnius, Lithuania

* Corresponding author.

Introduction Individualized treatment decisions in psychiatry may be important, since substantial part of first choice drugs are

ineffective or cause side effects. Polymorphic variants of genes that code CYP450 enzymes cause differences in their activity and therefore in efficacy and safety of drugs that are metabolized by them.

Aim of the study Determine whether pharmacogenetic testing of CYP2D6, CYP2C19 and CYP2C9 polymorphism would have had influence on selected patients' treatment courses.

Methods Five patients that were diagnosed for treatment-resistant mood disorders in Vilnius university hospital Santariskiu clinics centre of neurology, department of psychiatry were invited to give blood samples for genetic testing retrospectively. Patients' CYP2C19, CYP2D6 and CYP2C9 enzymes genetic polymorphism results were compared with previous empirical pharmacological treatment courses of these patients.

Results In four out of five cases significant polymorphism of CYP2C19 enzyme allele was detected. In all of these cases 1*/2* variant, that conditions intermediate metabolizer phenotype, was identified. Alterations in CYP2D6 and CYP2C19 regions were not found. In three cases the presence of varied genetic variant could have been clinically relevant. In two of these cases Sertraline and valproates, that are both metabolized by CYP2C19 enzyme, were taken by patients and side effects were observed. Unsuccessful treatment was repeated without effect, both in clinical and outpatient environment. Continuous rehospitalization took place until appropriate empirical treatments were established.

Conclusions Pharmacogenetic testing could have had influence on treatment choices for three out of five selected patients leading to less side effects and rehospitalizations.

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

<http://dx.doi.org/10.1016/j.eurpsy.2017.01.2055>

EW0187

Mthfr Allele distribution in Romanian schizophrenia patients

B. Nemes*, D. Cozman

Iuliu Hatieganu University of Medicine and Pharmacy Cluj-Napoca, Department of Medical Psychology, Cluj-Napoca, Romania

* Corresponding author.

Introduction Currently available data on the aetiology of schizophrenia suggests a major involvement of epigenetic mechanisms. One such mechanism could be the alteration of activation and silencing of genes, which involves DNA methylation and de-methylation. The main limiting enzyme involved in the methyl-donor cycle is methylene-tetra-hydro-folate-reductase (MTHFR), and the most frequently observed mutation in the MTHFR gene, altering its activity, is the C677T mutation.

Aim In the present study, we investigated the frequency of MTHFR C677T mutation and total plasma homocysteine (tHcy) concentrations in a sample of Romanian schizophrenia patients as compared to healthy controls.

Methods Seventy schizophrenia patients (35% females) with a mean age of 38.8 ± 20.5 years and 50 healthy controls (50% females) with a mean age of 36.3 ± 11.6 years were included. MTHFR genotype was determined through polymerase chain reaction and tHcy levels were determined through reversed phase high-pressure liquid chromatography.

Results Schizophrenia patients, registered higher frequency of the T allele, with the CC genotype observed in 39.4% of them, as compared to a frequency of 60.6% in the control group ($P=0.002$ –Fisher's exact test). tHcy concentrations did not differ between the two groups (10.7 ± 4.2 vs. 11.2 ± 4.1 , $P>0.005$ –Mann–Whitney U test).

Conclusions Romanian schizophrenia patients have a significantly higher frequency of the MTHFR C677T mutation, but without significant effect on tHcy concentrations.

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

<http://dx.doi.org/10.1016/j.eurpsy.2017.01.2056>

EW0188

Influence of 5-HTR2C polymorphisms on metabolic syndromes in Thai schizophrenia patients

A. Puangpetch^{1,*}, C. Na Nakorn¹, W. Unaharassamee², C. Sukasem¹

¹ Pathology service, Faculty of Medicine, Ramathibodi hospital, Mahidol University, Bangkok, Thailand

² Psychiatry, Somdet Chaopraya Institute of Psychiatry, Bangkok, Thailand

* Corresponding author.

Introduction Metabolic syndrome is a significant problem in the schizophrenia patients. Previous research demonstrated that single nucleotide polymorphisms in the serotonin 2C receptor (5HTR2C) genes are associated with metabolic syndrome related to schizophrenia patients taking atypical anti-psychotic drugs. This study aimed to investigate whether the effect of 3 SNPs in 5HTR2C gene on the presence of the metabolic syndrome in Thai schizophrenia patients.

Method We conducted a cross-sectional study and 154 patients were recruited. The schizophrenia patients were identified from a diagnostic and statistical manual of mental disorders, 4th edition, (DSM-IV) and criterion and determined the metabolic syndrome according to the 2005 international diabetes federation (IDF) Asia criteria. Patients were genotyped for the 5HTR2C rs51,8147, rs126,881,02, rs128,367,71 polymorphisms.

Results The preliminary analysis from 154 patients showed the metabolic syndrome prevalence was 38.73%, with 46.50% in male and 53.48% in female patients. The results showed that the patients who have heterozygous and homozygous variant on 5HTR2C gene (rs518,147 and rs126,881,02) showed a significant difference in the presence of metabolic syndrome when compare with patients who carry homozygous wild type ($P=0.007$), especially in male patients ($P=0.002$). The association between 5HTR2C polymorphisms and metabolic syndrome was found in male patients but not found in female patients.

Conclusion These findings suggest that 5HTR2C genotypes are associated with the metabolic syndrome in patients taking atypical anti-psychotics. However, the metabolic syndrome results from the multigenetic effects. The further studies should focus on the other genes, which were involved in metabolic syndrome.

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

<http://dx.doi.org/10.1016/j.eurpsy.2017.01.2057>

EW0189

Prevalence of the CYP2D6*10 (C100T) polymorphism in psycho-neurological patients in North-Western and Siberian regions of the Russia

N.A. Shnayder¹, R.F. Nasyrova², L.V. Lipatova³, V.V. Teplyashina², K.A. Sosina², N.A. Sivakova^{3,*}, E.N. Bochanova⁴, D.V. Dmitrenko¹, I.P. Artyuhov⁵, N.G. Neznanov⁶

¹ Voyno-Yasenetsky, the Department of Medical Genetics and Clinical Neurophysiology, The Krasnoyarsk State medical University named after Prof. V.F., Krasnoyarsk, Russia

² The Department of Personalized Psychiatry and Neurology, St. Petersburg Psychoneurological Research Institute named after V.M. Bekhterev, Saint-Petersburg, Russia