e-Poster walk: Genetics & molecular neurobiology and neuroscience in psychiatry

EW0169

Meta-analysis update of association between dopamine transporter SLC6A3 gene polymorphism, smoking cessation

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The SLC6A3 gene is involved in the dopamine pathway, which influences smoking behavior. This study was conducted to present updated results of a meta-analysis to evaluate the association between SLC6A3 polymorphism and smoking cessation. In total, eight studies were assessed, and 9-repeat alleles and no 9-repeat alleles were compared by smoking cessation outcomes. No significant association between SLC6A3 genotype and smoking cessation was observed for the main analysis (odds ratio = 1.128; 95% confidence interval = 0.981–1.298). In conclusion, the genetic variations in SLC6A3 are not associated with smoking cessation, which is not consistent with the results of the previous meta-analysis.

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EW0170

Qualitative meta-analysis to identify genomic variants that are correlated with the development of Schizophrenia

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Introduction Schizophrenia is a devastating and complex disease, which occurs in approximately 1% of the general population. Symptoms include hallucinations, delusions and patients' social withdrawal. Schizophrenia's etiology remains unclear, however, both patients' genetic profile and environmental factors play a significant role.

Objectives Our study's primary objective was to identify genetic variants related with schizophrenia's development in non-Caucasians populations and to explore whether these polymorphisms can be also found in schizophrenia patients of Caucasian origin.

Methods To achieve that, we screened Science Direct and PubMed medical literature databases to identify research articles correlating genes and variants with the development of schizophrenia. Next step was the categorization of studies according to samples' origin and the identification of genomic variants that are correlated with schizophrenia ($P < 0.001$) but have never been studied in Caucasian populations.

Results In total, 108 and 47 studies, in non-Caucasian and Caucasian populations respectively, were identified, in which 157 ($P < 0.05$) and 18 ($P < 0.001$) variants were associated with the development of the disease in non-Caucasian populations.

Conclusions From our qualitative meta-analysis 18 variants that were correlated with schizophrenia's etiology were identified ($P < 0.001$), which will be further investigated in a multi-cultural Caucasian cohort.

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EW0171

Potential values and risks of biomarker use in differential diagnosis of neurocognitive disorders

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Introduction Neurocognitive disorders are the only psychiatric disorders which underlying pathogeny can potentially be determined. This has important implications, for it makes possible the use of biomarkers in order to gain better diagnosis, and opens a door to more accurate treatments. Nonetheless, as biomarkers are not exclusive of a single disorder, the lengths of its utility are still unknown.

Objectives and aims To understand the values and limitations of biomarkers in differential diagnosis of dementias.

Methods We present three cases followed in the Neurology ward of our hospital, in which they were admitted for diagnosis and treatment of a subacute form of dementia. Medical history, core symptoms, screening tests for cognitive impairment, MRI, EEG and biomarkers in cerebrospinal fluid were used for diagnosis.

Results Two cases had consistent clinical features and complementary explorations, and they were respectively diagnosed as Creutzfeldt-Jakob Disease and Lewy Body Dementia; however, the last case showed contradictory results between clinic and complementary explorations, particularly 14-3-3 protein, which was positive and led to the initial diagnosis as Creutzfeldt-Jakob Disease, which was proven wrong once necropsy was practiced.

Conclusions Although complementary explorations, and biomarkers in particular, are of invaluable utility in the accurate diagnosis of multiple psychiatric diseases, they must always be considered within a context given by biography and clinical features, because, when failing to do so, they can lead to misdiagnosis and delay of correct treatment.

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EW0172

Spatio-temporal perception and boundaries of self: Evaluation of peripersonal space in schizotypy traits

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Introduction The peripersonal space is described as that area within the boundary between self and non-self. An accurate judgment of peripersonal space boundaries may depend on the capacity to create an organized and structured mental representation that integrates signals from different sensory modalities and

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brain regions. Empirical evidence suggests that these functions are altered in schizotypy, which is thought to reflect the subclinical expression of the symptoms of schizophrenia in the general population. A number of clinical studies reported that interpersonal interaction and social stimulation have an impact on the onset and progress of schizophrenia.
Objectives We conducted a study on personal space in a sample of student screened for schizotypal traits using a paradigm that was not affected by emotional and social interference.
Aims The aim was to evaluate the relationship between personal space and schizotypy traits.
Methods Thirty-four subject recruited for the study completed the Schizotypal Personality Questionnaire (SPQ). According to the SPQ results participants were splitted into two groups (High, Low). Each participant performed a PeriPersonal Space (PPS) task.
Results Our results show a more extended boundary of the peripersonal space in people with high schizotypy compared to people with low schizotypy even without emotional and social interference.
Conclusions People with high traits of schizotypy suffer from a difficulty in social integration because of being unable to adapt the social behavior. A better understanding of the mechanisms for abnormal interactive behavior could provide significant valid guidelines for innovating insertion programs that aims to improve social functioning.
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EW0173

Poor CYP2D6 and ultrarapid CYP2C19 metabolizer: Clinical challenge in psychiatric treatment

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Introduction Finding the right medication in psychiatry can be very demanding both for the doctor and for the patient. It becomes extremely grueling when the patient has a rare type of metabolizing enzymes, and many drugs may be ineffective or cause side effects.
Objectives To highlight the therapeutic difficulties in psychiatric treatment of the patient with complex genetic cytochrome P450 system alterations.
Aims To provide an example on a complicated treatment course of the patient that is poor CYP2D6 and ultrarapid CYP2C19 metabolizer.
Methods Literature review in scientific database–Pubmed–and case report presentation.
Results We report a case of a woman in her early twenties who was repeatedly referred for psychiatric treatment. A diagnosis of paranoid schizophrenia was established, but all treatment rounds were unsuccessful, the illness kept progressing, and major depressive disorder aggravated the clinical picture. The patient became suicidal and injured herself. During the sixth hospitalization in one year the CYP2D6, CYP2C19 and CYP2C9 genotyping was done. CYP2C19 ultrarapid (*1/*17) and CYP2D6 poor metabolizer (*4/*5) profile was discovered. Drugs, that should have been avoided due to the patient's genetic profile, had been prescribed throughout five hospitalizations in a row.
Conclusions As ultrarapid CYP2C19 metabolizers compose around 3–4% and poor CYP2D6–6–10% of Caucasians, this case presents a rare genetic variant that only 0.18–0.4% of Caucasian population may have. These cases can be extremely clinically challenging and affect healthcare outcomes and costs. Further studies that would include clinical effectivity, drug concentration and genetic testing results are needed.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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EW0174

Insight gained from genome-wide interaction and enrichment analysis on weight gain during citalopram treatment

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Introduction Weight gain is a side effect of pharmacological antidepressant treatments, causing a poorer compliance, increasing the risk of metabolic syndrome and periods of untreated disease.
Objectives The ability to precisely prescribe pharmacological treatments based on personal genetic makeups would increase the quality of the current antidepressant treatments.
Aims The molecular pathways enriched during citalopram induced weight gain are identified.
Methods 643 depressed citalopram treated individuals with available clinical and genome-wide genetic information were investigated in the present contribution in order to identify the molecular pathways that holds the key to weight gain. Statistics were conducted in R environment (Bioconductor and Reactome packages), ANOVA and MANCOVA served when appropriate. Plink was used for genetic analysis in a linux environment.
Results One hundred and eleven individuals had their weight increased after treatment with citalopram. The axon guidance (*P.* adjust = 0.005) and the developmental biology pathway (*P.* adjust = 0.01) were found to be enriched in genetic variations associated with weight gain.
Conclusions The development biology pathway includes molecular cascades involved in the regulation of beta-cell development, and the transcriptional regulation of white adipocyte differentiation. A number of variations were harboured by genes whose products are involved in the synthesis of collagen (*COL4A3*, *COL5A1* and *ITGA1*), activity of the thyroid-hormones (*NCOR1* and *NCOR2*), energy metabolism (*ADIPOQ, PPARGC1A*) and myogenic differentiation (*CDON*). A molecular pathway analysis conducted in a sample of depressed patients identifies new candidate genes whose future investigation may grant relevant insights in the molecular events that drive weight gain during antidepressant treatment.
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EW0175

Predicting antidepressant response from genes

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Introduction Pharmacogenetics may inform an accurate prescribing of antidepressants by identifying the genetic background