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

http://dx.doi.org/10.1016/j.eurpsy.2016.01.397

EW280

Polymorphism neuropeptide receptor gene S (NPSR1) and sleep disturbances

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Objective To study the association gene of candidate NPSR1 rs324981 with sleep disorders in the open population of men 45–64 years of Novosibirsk.
Materials and methods The study of the association candidate gene polymorphisms with sleep disorders was carried out during the examination of a random representative sample of men 45–69 years ($n = 1770$). The response rate was 61%. The median age is 56.5 year. Every 12 a man was selected for genotyping ($n = 147$). To assess the level of sleep was used a questionnaire which was filled with self-test. Statistical analysis was performed using SPSS-11.5.
Results The level of sleep disorders in the male population of 45–64 years was 79.9%. The frequency of homozygous C/C genotype of neuropeptide S (gene *NPSR1* rs324981) was 19.4%, T/T genotype occurs in 27.8%, C/T genotype −52.8%. Men dominated the T allele of −54.2%, and the C allele −45.8% growth trend Fnd dissatisfaction with the quality of their sleep among men. Men T-allele carriers, most evaluated their sleep as "satisfactory" in 69% of cases, ($\chi^2 = 15{,}713$ df = 8, $P < 0.05$).
Conclusion Association found men carrier T-allele of neuropeptide S (gene *NPSR1* rs324981), a sleep disorder.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.398

EW282

Borderline personality disorder and childhood maltreatment: A genome-wide methylation analysis

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Early life adversity plays a critical role in the emergence of borderline personality disorder (BPD) and this could occur through epigenetic programming. In this perspective, we aimed to determine whether childhood maltreatment could durably modify epigenetic processes by the means of a whole-genome methylation scan of BPD subjects. Using the Illumina Infinium® Human Methylation 450 Bead Chip, global methylation status of DNA extracted from peripheral blood leucocytes was correlated to the severity of childhood maltreatment in 96 BPD subjects suffering from a high level of child adversity and 93 subjects suffering from major depressive disorder (MDD) and reporting a low rate of child maltreatment. Several CpGs within or near the following genes (IL17RA, miR124-3, KCNQ2, EFNB1, OCA2, MFAP2, RPH3AL, WDR60, CST9L, EP400, A2ML1, NT5DC2, FAM163A and SPSB2) were found to be differently methylated, either in BPD compared with MDD or in relation to the severity of childhood maltreatment. A highly relevant biological result was observed for cg04927004 close to miR124-3 that was significantly associated with BPD and severity of childhood maltreatment. miR124-3 codes for a microRNA (miRNA) targeting several genes previously found to be associated with BPD such as NR3C1. Our results highlight the potentially important role played by miRNAs in the etiology of neuropsychiatric disorders such as BPD and the usefulness of using methylome-wide association studies to uncover such candidate genes. Moreover, they offer new understanding of the impact of maltreatments on biological processes leading to diseases and may ultimately result in the identification of relevant biomarkers.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.400

EW283

Effect of Disrupted-in-Schizophrenia 1 gene on treatment response in patients with a first episode of psychosis

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Introduction There is substantial evidence suggesting that individual variability in antipsychotic treatment response could be genetically determined. Disrupted-in-Schizophrenia 1 (DISC1) gene has been previously associated to the illness and to treatment response in a sample of patients suffering from psychosis. However, there is a lack of studies on the effect of DISC1 on treatment response in samples of first episode psychosis.
Objectives The aim of this study was to explore the relation between variations in DISC1 gene and treatment response to antipsychotics in a sample of drug-naïve patients with a first episode of psychosis.
Methods Two hundred and twenty Caucasian drug-naive patients experiencing a first episode of non-affective psychosis were genotyped for rs821616 (Ser704Cys), rs6675281 (Leu607Phe) and rs1000731. Early (6 weeks) response to antipsychotic treatment was assessed with the Brief Psychiatric Rating Scale, the Scale for the Assessment of Positive Symptoms, and the Scale for the Assessment of Negative Symptoms. Other clinical and socio-demographic variables were recorded to eliminate potential confounding effects.
Results We found a significant association between rs1000731 and treatment response. Thus, those patients homozygous for the G allele of rs1000731 were more frequently non-responders, measured with SANS, after 6 weeks of treatment, than those carrying the A allele ($X^2 = 4.019$; $P = 0.032$). Moreover, when analysing the clinical improvement longitudinally, we observed that those patients carrying the A allele for the rs1000731 presented a greater improvement in positive symptoms dimension ($F = 8.905$; $P = 0.003$).
Conclusions Our results suggest a minor contribution to antipsychotic drug response of genetic alterations in the DISC1 gene.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.401