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

http://dx.doi.org/10.1016/j.eurpsy.2017.01.2041

EW0173

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

E. Dlugauskas[1,*], A. Lengvenyte[2], R. Strumila[3], A. Utkus[4]
[1] *Vilnius University, Clinic of Psychiatry, Center of Neurology, Departament of Psychiatry, Departament of Psychiatry, Vilnius, Lithuania*
[2] *Vilnius University, Faculty of Medicine, Clinic of Psychiatry, Vilnius, Lithuania*
[3] *Vilnius University, Faculty of Medicine, Faculty of Medicine, Vilnius, Lithuania*
[4] *Vilnius University, Department of Human and Medical Genetics, Center for Medical Genetics, Vilnius, Lithuania*
[*] *Corresponding author.*

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.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.2042

EW0174

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

H. Corfitsen, A. Drago[*]
Psykiatrisk Forskningsenhed Vest, Department of Clinical Medicine, Aarhus University, Herning, Denmark
[*] *Corresponding author.*

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

http://dx.doi.org/10.1016/j.eurpsy.2017.01.2043

EW0175

Predicting antidepressant response from genes

H. Corfitsen, A. Drago[*]
Psykiatrisk Forskningsenhed Vest, Department of Clinical Medicine, Aarhus University, Herning, Denmark
[*] *Corresponding author.*

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