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.

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

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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

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specifically responding to a certain drug. Despite decades of efforts though, pharmacogenetics appears to be still in its infancy.
Aim A clearer understanding of the pharmacodynamics and pharmacokinetics events in combination with the genetic and epigenetic controls of cells and molecular cascade must inform the future of personalised medicine.
Objectives To systematically review the current cutting edge knowledge about pharmacognetics in the search for the next groundbreaking biological key events that may provide the keys to future treatments.
Methods The major online databases are systematically searched with common keywords by two independent researchers and conflicting findings are solved during regular meetings dedicated to the topic in object. Manual searching of single bibliographies is also put in place.
Results Genes belonging to the serotoningeric, dopaminergic, glutamatergic and GABAergic systems are classic candidates for pharmacogenetics whose role was not confirmed by GWAS analyses, which, on the other hand, identified genes related to molecular pathways not associated with direct target of drugs used for the treatment of depression.
Conclusion Both hypothesis driven candidate genetic investigations and GWAS analyses have been conducted so far, leading to the identification of a handful of potential good candidates, but the replication rate of the positive association findings lags behind expectations. The current knowledge about the pharmacodyncamic and pharmacokinetic genetic determinants of antidepressant response is critically analysed and new candidates are presented discussed.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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EW0176

A molecular pathway analysis informs the genetic risk for arrhythmia during antipsychotic treatment

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Background Arrhythmia is a potentially fatal side effect of antipsychotics. A biologic predictive tool to prevent it is missing.
Aim Identification of a genetic profile at risk for antipsychotic induced arrhythmia.
Objective Identifying a molecular pathway enriched for antipsychotic induced QT-modifications.
Methods Seven hundred and sixty-five SKZ individuals, $M = 556$, age = 40.93 ± 11.03 were included. QT-variation was a phase-specific created variable. A nested mixed regression served in R for clinical and molecular pathway analyses. Plink served for genetic analyses. Quality checking was standard, inflation factor was controlled by lambda values.
Results Quetiapine and Perphenazine were associated with QT variation ($P = 0.002$; Estimate = 5.79 and $P = 5.67e\text{-}06$; Estimate = 8.96 respectively). No other significant association was detected. No inflation was detected. Axon guidance and Collagen biosynthesis (Table 1) were associated with QT variation at a conservative (adjusted) P value < 0.01.
Conclusions Two molecular pathways were identified as possibly involved in QT modifications during antispsychotic treatment in SKZ patients. Previous evidence supports a role of the same pathways in cardiac disorders [1,2]. Interaction of specific SNPs with the drugs will be focus of further research.

Table 1 Molecular pathways enriched in association with QT modifications.

ID	Description	Gene Ratio	BgRatio	*P*-value	*P*.adjust	Qvalue
422475	Axon guidance	19/135	292/6750	4.6e–06	0.0022	0.0021
1650814	Collagen biosynthesis and modifying enzymes	8/135	59/6750	1.9e–05	0.0047	0.0044

Disclosure of interest The authors have not supplied their declaration of competing interest.
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EW0177

A molecular pathway analysis stresses the role of inflammation towards cognition in Schizophrenia

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Background Cognitive processes are impaired in Schizophrenia (SKZ). The nature of such impairment escapes definition.
Aim Identification of a genetic profile at risk of cognitive impairment.
Object Identifying a molecular pathways enriched for mutations associated with cognitive impairment.
Methods Seven hundred and sixty-five individuals from the CATIE, $M = 556$, mean age = 40.93 ± 11.03 were included. Verbal memory was outcome. R and Plink served for the analyses. Inflation factor was controlled by lambda values. Input for the pathway analysis were SNPs associated with outcome ($P < 0.05$) genomewide.
Results Gender (male, $P = 2.34e{-}05$; $t = -4.26$) and years of education ($P = 1.57e{-}03$; $t = 6.502$) were associated with verbal memory. Inflammation and oxidation were associated with outcome (Table 1, adj_$P < 0.01$).
Conclusions Being male and poorly educated were associated with poorer verbal memory. Inflammation and the arachidonic acid pathway were enriched in mutations associated with poorer verbal memory. This finding is in line with previous reports [1,2,3].

Table 1 Pathways enriched in association with verbal memory.

Description	GeneRatio	BgRatio	*P*value	*P*.adjust
Synthesis of Leukotrienes	5/105	17/6750	4.42E–06	0.0009
Arachidonic acid metabolism	7/105	45/6750	5.03E–06	0.0009
Glutathione synthesis and recycling	4/105	11/6750	1.68E–05	0.0021

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