response to CYP2D6 substrates comparing to wild type homozygous. As none of the analyzed patients was PM, exceeded plasma concentrations of medications above toxic levels are not expected when administrating the right dosage.
Conclusion Altered CYP2D6 metabolism may contribute to the vulnerability, clinical severity and treatment outcome of schizophrenia.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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O093

Differential susceptibility properties of the *5HTTLPR* gene in relation to depressive symptoms and delinquency

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Introduction The candidate gene-environment interaction (cG × E) research field in psychiatry has traditionally been dominated by the diathesis–stress framework, where certain genotypes are assumed to confer increased risk for adverse outcomes in a stressful environment. In later years, theories of differential susceptibility or biological sensitivity have been presented, suggesting that cGs that interact with environmental events do not exclusively confer a risk for behavioural or psychiatric disorders but rather seem to alter the sensitivity to both positive and negative environmental influences.
Aims The present study investigates the susceptibility properties of the *5HTTLPR* gene in relation to depressive symptoms and delinquency in two separate adolescent community samples: $n = 1457$, collected in 2006; and $n = 191$, collected in 2001.
Results Two-, three- and four-way interactions between the *5HTTLPR*, positive family environment, negative family environment, and sex were found in relation to both depressive symptoms and delinquency. However, the susceptibility properties of the *5HTTLPR* gene were distinctly less pronounced in relation to depressive symptoms.
Conclusions If the assumption that the *5HTTLPR* gene induces differential susceptibility to both positive and negative environmental influences is correct, the previous failures to measure and control for positive environmental factors might be a possible explanation for former inconsistent findings within the research field.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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O094

Epigenetics in the remission of anorexia nervosa: A follow-up study of whole-genome methylation profiles

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Introduction Anorexia nervosa (AN) is a severe psychiatric disorder. The epigenetic regulations are strongly suggested in AN. We and other groups have performed a whole-genome methylation study (methylome) in AN. We found that the differentially methylated CpG sites are located around genes involved in biological processes in link with embryonic morphogenesis, brain development and its plasticity, in particular adhesion and axon guidance. Here, we study an independent group of 40 AN patients. Furthermore, we have done a follow-up during more than one year, to compare the methylation profiles in subjects that evolve to the remission.
Objectives Our work is to replicate the methylome study in an independent AN cohort and to characterize profiles of methylation at two times for the same subjects to compare the AN patients that convert to remitters.
Aims Our goal is to identify diagnostic and prognostic epigenetic signatures for AN.
Methods Of the 40 AN patients, 18 evolved to remission. Furthermore, the blood samples of the subjects from the 2 times will be investigated, like this, each subject is its own control. Methylation of DNA is measured by using the Infinium HumanMethylation450 BeadChip technology.
Results Comparisons of AN to controls showed similar profiles of methylation involving the same biological processes as previously identified. We are comparing now the difference of methylation between the 18 remitters and the 18 actual AN, taking into account of the two times of samples.
Conclusions We expect to characterize specific methylation signature of the prognostic of the AN remission.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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O095

Exploring lithium impact on glomerular function in bipolar patients through pharmacogenomics

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Introduction Bipolar disorder (BD) is characterized by unusual shifts in mood and energy and affects 1 to 3% of the general population. Lithium (Li) can prevent patients from depression and mania, as well as reduce the risk of suicide. Unfortunately, a high rate of patients do not respond positively to Li treatment. In line with various studies, Li treatment is also associated with potentially severe adverse reactions, including renal dysfunctions. Specifically, it has been reported that Li may induce reduction of glomerular filtration rate (GFR) in long-term treated BD patients.
Aims The aim of our study was to evaluate the contribution of genetic variants in Li-induced reduction of the estimated GFR (eGFR) in bipolar patients, under long term Li therapy.
Objectives We screened the literature to identify genes previously shown to be associated with kidney function or Li mechanism of action and genotyped tag SNPs covering these genes.
Methods The sample comprised 70 Sardinian bipolar patients genotyped for 46 SNPs, located in 33 genes, with Invader assay and Sanger sequencing.
Results Our results showed that a SNP (rs378448) located in Acid Sensing Ion Channel Neurona-1 (*ACCN1*) gene, significantly interacted with years of Li treatment in reducing eGFR ($F = 4.166$, $P = 0.046$).

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Conclusions Our preliminary findings suggest that *ACCN1* (*ASIC2*) gene could be involved in modulating the susceptibility of BD patients to develop renal dysfunctions induced by chronic Li treatment.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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O096

Association between two single-nucleotide polymorphism of *TAAR1* gene and suicide attempts

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Introduction *TAAR1* is a G protein-coupled receptor expressed broadly throughout the brain. Recently, *TAAR1* has been demonstrated to be an important modulator of the dopaminergic, serotonergic and glutamatergic activity.
Aims Assessment of the relation between two single-nucleotide polymorphisms of *TAAR1* gene, suicide attempts and alcohol abuse.
Methods A total of 150 Polish patients were included, 59 subjects after suicide attempt vs. 91 controls. The chosen SNPs (rs759733834 and rs9402439) were studied using RFLP-PCR methods. The Hardy-Weinberg equilibrium was tested in control group.
Statistical tests Chi^2 or Yeates Chi^2 Test were used.
Results The mean age of study subjects and controls was: 38 ± 12.3 and 42 ± 12.8 respectively; 49% study males vs. 54% male controls. We did not observe the association between the carriage of the genotypes GG, GA and AA of rs759733834 polymorphisms in either of the groups. The distribution of genotypes in respect to rs9402439 polymorphism (CC, CG, GG) was also insignificant. Among patients with alcohol dependence, the frequency G allele of rs9402439 polymorphism was lower compared to non-addicted ones (27 vs. 47%) $P < 0.01$.
Conclusions *TAAR1* polymorphisms rs759733834 and rs9402439 are not related to suicide attempts. The carriage of allele G of rs9402439 polymorphism is related to lower risk of alcohol addiction OR 0.40 95%Cl 0.20–0.81. To our knowledge, this is the first study on the *TAAR1* receptor and the risk of suicide and it might offer a new insight into genetic etiology of *TAAR1* receptor.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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O097

Verbal learning and memory in at-risk mental state and first episode psychosis patients and their correlates to brain structural alterations

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Introduction Patients with a first episode psychosis (FEP) have repeatedly been shown to have gray matter (GM) volume alterations. Some of these neuroanatomical abnormalities are already evident in the at-risk mental state (ARMS) for psychosis. Not only GM alterations but also neurocognitive impairments predate the onset of frank psychosis with verbal learning and memory (VLM) being among the most impaired domains. Yet, their interconnection with alterations in GM volumes remains ambiguous.
Objective To evaluate associations of different subcortical GM volumes in the medial temporal lobe with VLM performance in ARMS and FEP patients.
Methods Data were collected within the prospective Früherkennung von Psychosen (FePsy) study, which aims to improve the early detection of psychosis. VLM was assessed using the California Verbal Learning Test (CVLT) and its latent variables Attention Span (AS), Learning Efficiency (LE), Delayed Memory (DM) and Inaccurate Memory (IM). Structural images were acquired using a 3 Tesla magnetic resonance imaging scanner.
Results Data from 59 ARMS and 47 FEP patients were analysed. Structural equation models revealed significant associations between the amygdala and AS, LE and IM; thalamus and LE and IM; and the caudate, hippocampus and putamen with IM. However, none of these significant results withstood correction for multiple testing.
Conclusions Although VLM is among the most impaired cognitive domains in emerging psychosis, we could not find an association between low performance in this domain and reductions in subcortical GM volumes. Our results suggest that deficits in this domain may not stem from alterations in subcortical structures.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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O098

The effects of deep-brain magnetic stimulation (DMS) on white matter deficits: New mechanism in major depressive disorder (MDD) treatment

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Deep-brain magnetic stimulation (DMS) is an effective therapy for various neuropsychiatric disorders including major depression disorder. The molecular and cellular mechanisms underlying the impacts of DMS on the brain remain unclear. Studies have reported abnormalities in the white matter of depressive brains, suggesting the involvement of myelin and oligodendrocyte pathologies in the development of major depressive disorder. In this study, we use a cuprizone induced demyelination animal model to generate depressive like behaviours and white matter and oligodendrocyte damages. Meanwhile, we treated the animal with DMS 20 minutes daily during the cuprizone challenge or recovery period. Behavioural tests, including nesting, new objective recognition, working memory and depression-like behaviours were tested periodically. Histological staining and western blotting were used to examine the underlying mechanism of DMS. We found that DMS reverse cuprizone induced behavioural deficits in acute demyelination but not during the recovery period. DMS alleviated demyelination and inflammation induced by cuprizone. During the recovery period, DMS had no impacts on overall neural progenitor cell proliferation, but enhanced the maturation of oligodendrocyte. This data suggest that DMS may be a promising treatment option for improving white matter function in psychiatric disorders and neurological diseases in future.