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

K.-W. Nilsson*, C. Åslund
Uppsala University, Centre for clinical research, Västerås, Sweden
* *Corresponding author.*

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

N. Ramoz[1,*], S. Guillaume[2,3], P. Courtet[2,3], P. Gorwood[1,4]
[1] *Inserm U894, Center of Psychiatry and Neuroscience, Paris, France*
[2] *CHU de Montpellier, hôpital Lapeyronie–Psychiatry, Montpellier, France*
[3] *Inserm, U1061, Montpellier, France*
[4] *Hôpital Sainte-Anne, CMME, Paris, France*
* *Corresponding author.*

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

E.E. Tsermpini[1,*], Y. Zhang[2], P. Niola[3], C. Chillotti[4], R. Ardau[4], G. Severino[3], A. Bocchetta[4], G.P. Patrinos[1], M.T.M. Lee[2], A. Squassina[3]
[1] *University of Patras, School of Health Science, Department of Pharmacy, Laboratory of Molecular Biology and Immunology, Patras, Greece*
[2] *RIKEN Center for Integrative Medical Sciences, Laboratory for International Alliance on Genomic Research, Yokohama, Japan*
[3] *University of Cagliari, Section of Neurosciences and Clinical Pharmacology, Department of Biomedical Sciences, School of Medicine, Cagliari, Italy*
[4] *University Hospital of Cagliari, Unit of Clinical Pharmacology, Cagliari, Italy*
* *Corresponding author.*

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 ($F = 4.166$, $P = 0.046$).