reflect the relevance of resilient coping in the activation of non-kin relationships in old age.

Keywords Personal social networks; Ego-centred networks; Resilient coping; Elderly

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

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O090

Being afraid of compassion: Fears of compassion as mediators between early emotional memories and psychopathological symptoms in adulthood

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Introduction There is evidence suggesting that for some individuals self-generating compassion and being open to compassion from others can be difficult or aversive. To date, however, no study has explored how these fears of compassion are associated with early emotional memories, such as shame or safeness memories, and to symptoms of depression and anxiety in adulthood. The current study set out to investigate the mediator effect of fears of compassion on the relationship between the traumatic and centrality features of shame memories, early memories of warmth and safeness, and symptoms of depression and anxiety.

Method In this cross-sectional study, participants were 302 individuals (171 women; age M = 36.28; SD = 11.45) recruited from the general community population, who completed self-report measures of fears of compassion (for self, for others and from others), shame memories, safeness memories, depression and anxiety.

Results Path analysis showed that fears of compassion for self and of receiving compassion from others mediated the effects of shame traumatic memory, centrality of shame memory and early memories of warmth and safeness on depressive and anxiety symptoms. Fear of compassion for self was the best predictor of depression and anxiety.

Conclusions Fears of compassion may render an individual more vulnerable to defeat and threat responses when faced with stressful life events, which can manifest as symptoms of depression or anxiety. Clinical implications might be derived from these findings as these fears, as well as the negative emotional memories fuelling them, may need to be addressed in therapy to assist patients in self-generating and receiving compassion.

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

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Oral communications: Rehabilitation and psychoeducation and schizophrenia and other psychotic disorders

O091

Genetic counselling in psychiatric disorder with high suicide risk

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Introduction A better understanding of the genomics of mental illnesses allowed genetic counselling to be provided to individuals with severe mental illness and their families.

Aim The present study was aimed at assessing the efficacy of genetic counselling for severe mental illnesses with high suicide risk.

Method Assessment was performed before and after genetic counselling session. Measures used were evaluation of traumatic events in childhood, multidimensional scale for perception of social support (SMSSP), positive and negative affect schedule (PANAS-X), Brief Psychiatric Rating Scale (BPRS), Paykel questionnaire and Genetic Counselling Outcome Scale (GCOS). Paykel's questionnaire consists of five questions about suicidal thoughts and attempts, including: life-weariness, death wishes, suicidal ideation, suicidal plans and suicide attempts. Intervention and assessment lasted approximately one and a half hour. Data from 48 patients was analysed.

Results Mean age of participants was M = 38.4, SD = 9.7, and the group was batter represented by females (57%). The participants had various diagnoses, 22% had schizophrenia, 36% bipolar disorder and 42% recurrent depressive disorder. Forty percent of participants reported suicidal ideation and 22,5% had a past history of suicide attempt. Genetic counselling had a direct positive influence upon GCOS specific items and reduced the Paykel scores among participants presenting with suicidal ideation.

Conclusion Genetic counselling offers information about the disorder, the role of genetics and the impact of environmental factors. Preliminary data suggest that providing genetic counselling decreases the suicidal ideation frequency.

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

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O092

Analysing CYP2D6*4 Allele frequency in patients with schizophrenia

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Introduction Schizophrenia is treated with antipsychotics and other psychotropic medications, many of which are substrates for the highly polymorphic CYP2D6 enzyme. The most frequent variant allele is CYP2D6*4- leading cause of poor metabolism (PM) phenotype. PM causes the reduction of therapeutic response, increase the risk of adverse drug reactions and increase the plasma concentration of both drug and its metabolites above the levels of toxicity.

The Aim Analysing CYP2D6*4 allele frequency among schizophrenic patients for further individualisation and rationalisation of therapy.

Patients and methods Research was conducted on 38 schizophrenic patients and 110 healthy individuals. CYP2D6*4 allele was detected with allele specific PCR.

Results Both wild type allele carriers are 55% of the schizophrenic patients, 45% are wild type/*4heterozygous, and *4/*4 homozygous are not identified. There is a statistically significant difference in the genotype distribution ($P < 0.05$) between schizophrenic patients and healthy individuals. Significantly higher *4 allele frequency (37%) comparing to healthy individuals ($P < 0.0001$) indicates the necessary caution in administration of CYP2D6 substrates. A lower frequency of PMs in schizophrenic patients than in healthy individuals could be explained with CYP2D6 neuroactive substrate metabolism. Forty-five percent of the schizophrenic patients are intermediate metabolisers carrying the higher risk of adverse

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