

**Table 3.** Allelic frequencies of BDNF (rs6265) in BD cases and healthy controls

	ALLELIC VARIATION	
	C	T
CASES	235(78.3%)	65(21.6%)
CONTROLS	211(70.3%)	89(29.6%)
CHI-SQUARE-5.032	DF(Degree of freedom)-1	p-value- 0.024

**Conclusions:** Our study found that Val66Val genotype and Val allele were higher in cases and could be a potential biomarker for bipolar disorder (BD), which is consistent with previous research conducted on the European population. However, further investigations are required to gain a more comprehensive understanding of its impact on BD, including its association with serum BDNF levels, treatment outcomes, and a more diverse study population.

**Disclosure of Interest:** None Declared

## EPP0585

### Pharmacogenomics in Psychiatry: An Asian Perspective

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**Introduction:** Pharmacogenomic testing in psychiatry is an emerging area with the potential clinical application of guiding medication choice and dosing. However, this has not been adopted widely due to a combination of barriers that include a varying evidence base, clinician and patient familiarity and acceptance, uncertainty about cost-effectiveness, and regulatory requirements.

**Objectives:** This review aims to examine recent updates in this field and provide a contextualised summary and recommendations for Asian populations. The recommendations serve to guide healthcare professionals in the utility of pharmacogenomic testing in psychiatric practice.

**Methods:** A review of recent literature about current evidence and guidelines surrounding pharmacogenomics in psychiatric practice was carried out with particular attention paid to literature evaluating Asian populations. Literature was reviewed for the different classes of psychotropics with supplementary information about Asian populations included where available. Existing evidence about combinatorial pharmacogenomic panels was also reviewed.

**Results:** In line with the available body of evidence, we recommend that pharmacogenomic testing should be employed as an augmenting tool to guide medication selection and dosing in certain clinical situations, and not as part of standard or routine clinical practice. Pharmacogenomic testing should also be mainly limited to the known drug-gene pairs such as the anti-depressants and CYP2C19 or CYP2D6. Clinicians should also be aware that many of the gene-drug associations have not been evaluated for clinical outcomes. Combinatorial pharmacogenomic panels are not presently recommended as there is limited and inconclusive available evidence on clinical outcomes.

**Conclusions:** Pharmacogenomic testing in psychiatry is not recommended as standard or routine clinical practice. Exceptions may include concerns about drug concentrations (due to

metaboliser status) or potential severe adverse drug reactions/ Pharmacogenomic testing should be mainly limited to the known drug-gene pairs such as the anti-depressants and CYP2C19 or CYP2D6.

**Disclosure of Interest:** None Declared

## EPP0586

### GWAS in interaction with childhood traumas implicates novel variants and genes previously associated with suicide-related factors in the background of suicidal ideation

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**Introduction:** Although suicide claims more lives than war and homicide, we still have no sufficient and effective methods either for its prediction or for its prevention. Our screening methods are laborious and subjective both on the side of the patient and on the side of the clinician. Understanding the genetic background of suicidal behaviour would help identify biomarkers for screening as well as pathways as potential targets for novel intervention and prevention approaches. However, in spite of a number of GWAS studies, results are few and rarely replicate, and generally accurate phenotyping and sufficient consideration of environmental stressors is also missing.

**Objectives:** In our present study we performed a genome-wide analysis study for suicidal ideation in interaction with early childhood traumas in a deep-phenotyped general population sample.

**Methods:** Our analysis used data from 1800 volunteers in the NewMood project. As outcome phenotype the suicidal ideation item of the Brief Symptom Inventory was used. A modified version of the Childhood Trauma Questionnaire was used to assess early adverse experiences. A genome-wide association analysis was performed with Plink 1.9, including a total of 3,474,641 variants after quality control steps, followed by genome-wide by environment interaction analyses. Our models included control variables for sex, age, and the top 10 genomic principal components. Functional annotation of SNPs was carried out using FUMA v1.5.6, gene-based tests were performed using MAGMA v1.08.

**Results:** 7 SNPs met suggestive significance in main effect analyses, of which 2 reached genome-wide significance including *rs79912020* ( $p=3.21E-10$ ,  $\beta=0.746$ ) and *rs10236520* ( $p=1.71E-08$ ,  $\beta=0.484$ ), with no significant findings in gene-based tests. Interaction analyses with childhood adversities yielded 31 SNPs that met genome-wide significance, including *rs7983955* ( $p=2.28E-11$ ,  $\beta=0.182$ ), *rs141039461* ( $p=3.90E-11$ ,  $\beta=0.0541$ ), *rs12692827* ( $p=3.69E-10$ ,  $\beta=0.0612$ ) as the top SNPs. In interaction with childhood adversities, 31 genes showed a significant association in gene-based tests, including *RBFOX1* ( $p=1.09E-10$ ), *GRM7* ( $p=1.20E-10$ ), *MTCH1* ( $p=5.59E-09$ ), and *CDH13* ( $p=6.60E-09$ ) as the most significant findings.