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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doi: 10.1192/j.eurpsy.2024.694

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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doi: 10.1192/j.eurpsy.2024.695

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 laborous 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, β=0.746) and *rs10236520* (p=1.71E-08, β=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, β=0.182), *rs141039461* (p=3.90E-11, β=0.0541), *rs12692827* (p=3.69E-10, β=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.

Conclusions: Our results indicate several important novel SNPs associated with suicidal ideation when considered in interaction with the effect of childhood adversities. Furthermore, gene-based analyses replicate several genes playing a key role in central nervous system function such as *GRM7* (encoding metabotropic glutamate receptor 7) or previously implicated in association with suicide (*CDH13*) or suicide-related factors such as aggression (*RBFOX1*).
Funding: NAP2022-I-4/2022, K143391, 2019-2.1.7-ERA-NET-2020-00005, TKP2021-EGA-25

Disclosure of Interest: None Declared

EPP0587

Can compassion impact us on a cellular level? Preliminary findings on the effects of a compassion focused intervention on immunological markers and CTRA gene expression

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doi: 10.1192/j.eurpsy.2024.696

Introduction: Addressing mental and physical health problems and promoting wellbeing in educational settings is a global priority. Teachers present a high risk of stress and burnout, which negatively impacts their professional performance as well as their mental and physical health. Compassion-based interventions have been found effective in promoting psychosocial and physiological wellbeing.
Objectives: The current paper presents preliminary findings of the impact of a 6-module Compassionate Mind Training intervention for Teachers (CMT-T) on immunological markers and the Conserved Transcriptional Response to Adversity (CTRA; a gene expression signature that involves a group of 53 genes: pro-inflammatory genes, type I interferon response and genes related to antibody synthesis).
Methods: A pilot non-controlled study was conducted in a sample of public-school teachers in Portugal (n=36). Participants were assessed at 4 time-points: 1) Extended Baseline Control_M0, in order to establish a within-subjects psychological and biophysiological baseline (8 weeks before the start of the CMT-T); 2) Pre-intervention_M1 (8-weeks after M0); 3) Post-intervention_M2 (8-weeks after M1); and 4) Follow-up_M3 (3 months after the CMT-T end). In all assessment moments, participants completed a set of psychological self-report measures and were assessed in immunological and epigenetic biological markers through the collection of blood. After M1, teachers completed the 8-week group CMT-T intervention and given access to its resources and materials. They were instructed to practice daily and incorporate the teachings in their personal and professional lives. All assessments and the CMT-T intervention took place at the schools.
Results: Preliminary data on the impact of CMT-T on Immune Response Profiling revealed that teachers' Natural Killer (i.e., NK) cells were decreased after the CMT-T intervention. In regard to the CTRA gene expression, results showed that type one interferon response genes (e.g., IFI16, IFI27L2, IFITM2, IFITM3, IFITM4P) were decreased after the intervention. In addition, we observed that the gene c-Jun, a pro-inflammatory gene, had a decreased expression after the CMT-T intervention.
Conclusions: These preliminary findings seem to corroborate previous studies involving the type one interferon response, the pro-inflammatory genes and antibody synthesis genes in a signature involving 53 genes previously described as the CTRA gene signature. Furthermore, our results suggest that cultivating compassion using a compassion focused intervention may have a positive impact on markers of the immune system response, associated with how our bodies respond to stress, infection and cancer, as well as, on reducing the expression of genes related to our bodies' response to stress and inflammation.

Disclosure of Interest: None Declared

EPP0588

ASCL1 dysfunction contributes to the pathogenesis of schizophrenia by regulating genes associated with neuronal signature formation and neuroplasticity

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doi: 10.1192/j.eurpsy.2024.697

Introduction: ASCL1 (Achaete-scute homolog 1) is a neuron-specific transcription factor involved in CNS maturation in the mammalian brain. It has been shown to be associated with schizophrenia (SZ), Parkinson's disease, and the development of brain tumors. ASCL1 is expressed in the neuroblastoma cell line SH-SY5Y, which is a widely used model for the study of neurodevelopmental diseases, including SZ.
Objectives: The aim of this work was to study the effect of functional *ASCL1* knockout on the transcriptional landscape of SH-SY5Y cells in undifferentiated and neuron-like phenotypes.
Methods: For ASCL1 deletion, SH-SY5Y was sequentially transduced with two lentiviral vectors. One pLV-rtTA-Cas9-(nls)-pCMV-eGFP-PuroR-T2A-rTetR (derived from pCW-Cas9 and pEGFP-Puro) construct encoded Cas9. Stably transduced lines were selected for 3-5 days on puromycin (2 g/L). The inducibility of Cas9 expression was checked after adding the inducer oxytetracycline to the culture medium. The second construct (based on pLK05-tagRFP) encoded, a pair of guide RNAs targeting the start