

EPP0584

Social determinants of involuntary psychiatric hospital admissions in Ontario, CanadaK. P. Fung^{1*} and S. Kim²¹University of Toronto, Toronto and ²McMaster University, Hamilton, Canada

*Corresponding author.

doi: 10.1192/j.eurpsy.2024.692

Introduction: In Ontario, Canada, patients may be admitted to the hospital involuntarily if they are deemed to be suffering from symptoms of a mental disorder that may result in imminent serious bodily harm to themselves or others, or that may cause serious physical impairment to themselves (e.g., inability to keep themselves safe and warm in the winter). This measure can be life-saving. However, in addition to ethical and human rights considerations, resorting to coercive admissions may be an indication that those who are suffering from mental illness are not able to access or receive timely and appropriate intervention. While recent studies have suggested that the rate of involuntary hospital admission may be increasing, data on social determinants of involuntary hospital admissions are limited.

Objectives: We examined social factors associated with involuntary admissions using a Canadian provincial database.

Methods: Binary logistic regression models were conducted to examine the associations between social factors (low income, indigeneity, rurality, housing type) and involuntary admissions, controlling for age, sex, and psychiatric diagnoses. Data from March 2019 to March 2021 was extracted from the Ontario Mental Health Reporting System admission dataset, comprising of a sample of 9,848 patients admitted to eight psychiatric hospitals in Ontario. Odds ratios and 95% confidence intervals are reported.

Results: In 2021, the proportion of involuntary patients increased significantly by 6.8 percentage points to 55.7% compared to the previous year (48.9%). Indigenous status (First Nations, Metis, Inuit) [1.75 (1.38-2.21) **], living in rural areas [2.78 (2.48-3.12)], living in assisted residence [1.41 (1.21-1.64) **], homelessness [1.63 (1.38-1.91) **], male sex [1.21 (1.10-1.33) **] and younger age [0.99 (0.98-0.99) **] were associated with involuntary admissions, while income was not a significant factor. Compared to a diagnosis of a psychotic disorder, substance use disorders [0.11 (0.10-0.13) **] and mood and anxiety disorders [0.32 (0.29-0.36) **] showed decreased odds of involuntary admission, while neurocognitive disorders increased the odds of involuntary admission [3.86 (2.91-5.11) **].

Conclusions: Consistent with other findings, involuntary psychiatric hospital admissions in ON, Canada, have increased recently, which may in part be related to the pandemic. Rurality, indigenous status, and unstable housing have been found to be associated with involuntary admissions. The study findings support the need for better preventive and intervention strategies to serve vulnerable psychiatric patients, including addressing the social determinants of health such as housing, and increasing access to culturally competent and safe community-based mental health supports and services.

Disclosure of Interest: None Declared

Genetics and Molecular Neurobiology

EPP0584

Comparison of Val66met Polymorphism of BDNF gene in patients of bipolar disorder and healthy controls.M. Srikantamurthy^{1*}, S. Moirangthem², B. Viswanath², M. Purushottam³ and S. Jain²¹Department of psychiatry, Orygen youth health, Melbourne, Australia;²Department of psychiatry and ³Department of molecular genetics, National Institute of Mental Health and Neurosciences, Bangalore, India

*Corresponding author.

doi: 10.1192/j.eurpsy.2024.693

Introduction: The study aims to explore the utility of BDNF Val66Met polymorphism as a potential biomarker in Indian bipolar disorder patients and its correlation with clinical characteristics.

Objectives: Genotyping Val66Met in BDNF gene Exploring its association with bipolar disorder (BD).

Methods: 150 consenting BD patients and matched controls were recruited using a case-control study design. BD severity was assessed using Young's mania rating scale and the Clinical Global Impression - Severity (CGI-S) scale. BDNF Val66Met polymorphism was identified through real-time PCR after DNA extraction. Data was tested for normal distribution. Genotype frequencies between two groups were compared and the Hardy-Weinberg equilibrium assumptions were tested using Chi-Square tests. Clinical-genotypic associations were explored using the Kruskal-Wallis test and confirmed using hierarchical regression.

Results: Our sample had more males (60%) than females (40%) with mean age of 35.05 years. Most patients had established bipolar disorder and were severely ill (CGI: 38.75, YMRS). Val66Met SNP genotype frequency differed significantly between cases and controls. Val66Val genotype and Val allele were higher in cases. Results consistent with Hardy-Weinberg equilibrium.

Table 1. Genotype frequencies of BDNF (rs6265) in cases and controls

	GENOTYPE		
	CC	CT	TT
CASES	94(62.6%)	47(31.3%)	9(6%)
CONTROL	71(47.3%)	69(46%)	10(6.6%)
CHI-SQUARE- 7.431	DF(Degree of freedom) - 2	p-value- 0.024	

Table 2. Dominant genotype frequencies in cases and controls

	DOMINANT GENOTYPE	
	CC	CT+TT
CASES	94(62.6%)	56(37.3%)
CONTROLS	71(47.3%)	79(52.6)
CHI-SQUARE-7.125	DF(Degree of freedom)-1	p-value-0.007

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

S. E. Goh
Institute of Mental Health, Singapore, Singapore
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

X. Gonda^{1,2*}, S. Krause^{3,4}, B. Erdelyi-Hamza^{2,3}, S. Sutori⁴, Z. Gal⁴, N. Eszlari^{1,4}, G. Bagdy^{1,4}, G. Juhasz^{1,4} and D. Torok⁴
¹NAP3.0-SE Neuropsychopharmacology Research Group- Hungarian Brain Research Program; ²Department of Psychiatry and Psychotherapy; ³Doctoral School of Mental Health Sciences and ⁴Institute of Pharmacodynamics, Semmelweis University, Budapest, Hungary
*Corresponding author.
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* ($p=3.21E-10$, $\beta=0.746$) 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.