clei RNA-sequencing from the dorsolateral prefrontal cortex of patients with Major Depressive Disorder (MDD) and healthy controls (HC) using Sparse Decomposition of Arrays (SDA), a tensor decomposition method which handles higher dimensional data and allows to identify non-overlapping components. We aimed to uncover co-expression patterns across multiple cell types and to identify differentially co-expressed gene set associated with risk for psychiatric disorders.

Methods: RNA-sequencing data from 33 male individuals (17 patients with MDD who died by suicide) yielded 6477 genes across 22 cell types, which were given as input to SDA and clustered into 23 co-expression components. Components over-representing MDD top risk loci genes were associated with MDD diagnosis. We additionally evaluated the enrichment for schizophrenia (SCZ), bipolar disorder, suicide attempt, neurologic and immune disorders. We associated components with cell types via an analysis of cell type loading and via overrepresentation according to multiple reference atlases. Finally, we explored the gene ontology enrichment of diagnosis-associated components.

Results: Of 15 components not associated with confounders, four overrepresented MDD top risk loci (p[corrected]<.05). Only one of them also differed between MDD and HC (T = 2.9; p[FDR]=.024). The same component was also enriched for SCZ top risk loci genes (p[corrected]<.05). Cell type loading ascribed this component to parvalbumin-positive inhibitory neurons and astrocytes; the reference atlases highlighted neuronal specificity. Gene ontologies for this component included neuron projection morphogenesis, postsynaptic and somatodendritic compartment.

Discussion: We identified a cell-type-specific gene set differentially co-expressed in suicidal patients with MDD relative to HC. This gene set is also associated with shared risk for MDD and SCZ, both featuring increased suicide rate relative to the general population. Overrepresentation analyses point to an essential role of this gene set in local prefrontal neuronal circuits in which both genetic risk and co-expression differences between patients and controls converge.

Disclosure: Nothing to disclose.

doi: 10.1016/j.euroneuro.2022.07.513

F90.

SYSTEMATIC EXPLORATION OF PSYCHIATRIC GENETIC RESEARCH PUBLICATIONS IN LATIN AMERICA

Diana Garro, María Jesús Picado, Erika Espinoza, Daniela Ugalde, Gabriela Chavarria-Soley, Gabriel Macaya, Henriette Raventós

Universidad de Costa Rica

Background: Psychiatric and neurological disorders represent approximately 22% of the total diseases that affect populations in Latin America and the Caribbean. Even though psychiatric disorders have a great impact in terms of mortality, morbidity, and disability across all stages of life, research in mental health has not been a focus for funding agencies, in comparison to other health related research areas. In spite of this situation, several research efforts in different Latin American countries have focused on the genetics of psychiatric disorders. Additionally, some large international research initiatives have included populations of Latin American origin.

The aim of this study is to review the research published with Latin American populations between 2010-2019. We focused mainly on sources of funding, and how this is related to the scope and complexity of the study.

Methods: We performed a systematic literature search from 2010-2019 in the RedalyC and PubMed databases in Spanish, Portuguese, and English. The search terms were constructed with the following format: “Genetic” AND “Psychiatric disorder”, with the phrase “psychiatric disorder” being sequentially replaced by each of ten of the most common psychiatric disorders.

Results: In general, more articles were published within the 2015-2016 period. Additionally, between the 2010-2019 period, depression (20%) and Alzheimer (19%) were the most common disorders found. Most of the Latin American countries’ publications were internationally funded, with the exception of Brazil, for which 86% of all publications were locally funded. We found differences between locus specific studies and genome wide studies, regarding sources of funding (local or international) (x^2=9.46; p=0.009). 82% of all whole genome studies were funded by international sources. Over 70% of the studies used SNPs, mostly for association studies, and studies with endophenotypes or traits associated with the disorder. Finally, case/control studies represented the most frequent study design (42%), however an important proportion of the studies were family-based (33%).

Discussion: Our results showed that local funds are generally not large enough for genome-wide studies in Latin America, with the exception of Brazil. As a result, larger studies are often done in collaboration with international partners. We also found that in larger studies the participants are Latin American ancestry subjects living in the USA, not in Latin America. Additionally, our results showed the important role of family-based studies, which have traditionally been a strength in Latin American genetic research because of large family sizes.

In conclusion, limited local funding for research in Latin America has probably contributed to limited sample size, scope, and complexity of psychiatric genetic research studies. Increasing diversity in psychiatric genetics needs to be a future goal in order to improve generalizability and applicability in clinical settings.

Disclosure: Nothing to disclose.

F91.

A BLUEPRINT FOR BIOBANKING IN EVERYDAY CLINICAL PRACTICE IN PSYCHIATRY: THE MUNICH MENTAL HEALTH BIOBANK

Abstract not included.

F92.

CLARIFYING THE CAUSES OF CONSISTENT AND INCONSISTENT FINDINGS IN GENETICS

Abstract not included.

doi: 10.1016/j.euroneuro.2022.07.514

F93.

MAPPING THE COMMON GENETIC ARCHITECTURE OF EARLY TEMPERAMENT: FIRST-LOOK RESULTS FROM GENOME-WIDE ASSOCIATION ANALYSES OF INFANT COHORTS

Anja Hollowell [1], Anna Gui [1], Beate St Pourcain [2], Christel Middeldorp [3], Elma Fitzsimons [4], Mark H. Johnson [5], Frank Dudbridge [6], Angelica Ronald [1]

[1] *Birkbeck, University of London;* [2] *Max Planck Institute for Psycholinguistics;* [3] *University of Queensland;* [4] *University College London;* [5] *University of Cambridge;* [6] *University of Leicester*

Background: Temperament represents individual differences in behaviour and emotional reactivity that can be observed at an early age and that persist over time. Temperament at an early age has been shown to be associated with neurodevelopmental conditions in later life. For example, Shephard and colleagues (2019) reported an association between temperamental traits in infancy and ADHD and ASD at age 7. Thus, elucidating the genetic basis of infant temperament may be important for understanding the early developmental mechanisms of these conditions. The current evidence for the genetic influence on infant temperament has been derived from twin studies, which indicate that these traits are heritable. The Genetics of Early Milestones and Skills (GEMS) project is collaborating with multiple large birth cohorts in order to investigate the genetic architecture underlying infant milestones and traits. We will outline GEMS and present some first-look results from the UK Millennium Cohort Study, one of the participating cohorts.
Methods: Temperament was measured in the UK Millennium Cohort Study (total genotyped N=9259) (Connelly and Platt 2014). Parents completed questionnaire items about fussiness at nine months which were used to create a pro-rated composite score (N=6691, NF=3368, NM=3323; M=0.81 years, SD=0.04 years). At 36 months, temperament scales were derived from items from the parent-report Strengths and Difficulties Questionnaire (N= 6621, NF=3351, NM=3270; M=3.12 years, SD= 0.18 years). Participants were genotyped using the Infinium Global Screening Array, and genotype data were imputed to the Haplotype Reference Consortium release 1.1. Post-imputation quality control has been conducted using plink1.9 (Chang et al., 2015). Genome-Wide Association Studies (GWAS) of temperament traits at these two ages were conducted, including age, sex, and ten ancestry principal components as covariates. LD score regression was used to investigate genetic correlations between early temperament and later neurodevelopmental and psychiatric disorders.
Results: Phenotypically, there was variability in all temperament phenotypes for the planned GWAS analyses. Fussiness at nine months was approaching a normal distribution (skewness = 0.16; kurtosis = -0.42). Emotionality, Shyness and Activity at 36 months were all positively skewed (skewness = 0.73-2.00; kurtosis = -0.48-5.98). Sociability at 36 months was slightly negatively skewed (skewness = -0.49; kurtosis = 0.37). All scales showed significant mean sex differences.

A total of 169,593 SNPs and 7171 individuals have passed quality control. The GWAS scripts have been prepared. The genetic analyses are underway, and we will present the results at the conference
Discussion: This is the first discovery GWAS of infant temperament. Further future planned analyses in GEMS include multi-ancestry GWAS, meta-GWAS analyses split by sex, and exploration of genetic pleiotropy through LD score regression and polygenic score analyses. This research will have implications for the understanding of possible biological mechanisms that influence temperament and how temperament associates genetically with neurodevelopmental and psychiatric outcomes.

Disclosure: Nothing to disclose.

doi: 10.1016/j.euroneuro.2022.07.515

F94.

INVESTIGATING THE DEVELOPMENTAL HETEROGENEITY OF EMOTIONAL SYMPTOMS THROUGHOUT DEVELOPMENT, ASSOCIATED GENETIC AND EARLY LIFE RISK FACTORS, AND EARLY ADULT OUTCOMES IN THE TWIN'S EARLY DEVELOPMENT STUDY

Elisavet Palaiologou [1], Ewan Carr [2], Elham Assary [1], Geneviève Morneau-Vaillancourt [1], Celestine Lockhart [1], Alicia J. Peel [1], Ellen J. Thompson [1], Robert Plomin [1], Thalia C. Eley [1]

[1] *Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology and Neuroscience, King's College London;* [2] *Institute of Psychology, Psychiatry and Neuroscience, King's College London*

Background: The onset and course of emotional symptoms vary across development and individuals. However, within single populations, homogenous subgroups of individuals (i.e., trajectory classes) following similar patterns of symptom severity and stability have been identified. These trajectories have been differentially associated with both ge-