cortex, hippocampus, hypothalamus, dorsal striatum, nucleus Accumbens, ventral tegmental area and amygdala) using quantitative PCR and ELISA methods in the different groups of mice (ad libitum, ad libitum with wheel, food restriction and food restriction with wheel). Statistical analysis will compare the measures for different samples by one-way or two-way ANOVAs depending the group of animals or brain regions and blood.
Results: To date, no difference of the level of transcription for *Bdnf* was observed between the different groups of mice (ad libitum, ad libitum with wheel, food restriction and food restriction with wheel) in the prefrontal cortex, hippocampus and hypothalamus. We expect significant differences of Bdnf expression in the other brain regions of interest for the food restricted animals with or without the wheel compared to ad libitum animals. We expect also differences in the level of expression of Bdnf in fasted animals compared to the refeed animals.
Conclusions: The BDNF could represent a potential biomarker of AN for the diagnostic and the prognosis in the evolution to the remission when weight recover and thus will allow a better understanding of the aetiology of AN. This study is supported by Fédération pour la Recherche sur le Cerveau.

Disclosure of Interest: None Declared

EPP0458

Behavioral signs of CHARGE syndrome and CHD7 mutational spectrum

N. Bouayed Abdelmoula* and B. Abdelmoula

Genomics of Signalopathies at the service of Medicine, Medical University of Sfax, Sfax, Tunisia
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.767

Introduction: CHARGE syndrome is a genetic entity caused by mutations in the chromodomain helicase DNA-binding protein 7 gene (CHD7) at 8q12.1. There are pleiotropic signs among individuals with this disorder. Diagnosis is clinical using medical criteria. CHD7 gene mutations are usually found in 90% of affected patients.
Objectives: The aim of this study was to report behavioral signs of CHARGE syndrome and their phenotype-genotype correlations.
Methods: Four Tunisian males from Sfax (Tunisia) with clinical features suggestive of CHARGE syndrome were examined at our genetic counselling at the medical University of Sfax. Assessment of facial dysmorphic and behavioral features, karyotyping using RHG banding and molecular screening of CHD7 mutations were performed. Molecular analysis was made using direct Sanger sequencing of the entire CHD7 gene.
Results: Molecular genetic analysis revealed two deletions of the CHD7 gene at exon 3 for the first patient and at exon 8 for the second. The two genetic alterations were associated to retarded growth development and genital hypoplasia. Sensory impairments included for the first visual defects and for the second auditory and olfactory defects. Besides constant delayed psychomotor development, the two patients shared receptive and expressive communication disorders, anxiety, attention deficit, cognitive impairment and intellectual disability. There were no aggressive traits nor major autistic features. Learning disabilities were also present for the two patients.
Conclusions: The CHD7 gene controls the developmental pathways as a transcriptional regulator in the nucleoplasm through chromatin organization. Mutational alterations lead according to the affected domains, and the structure of the nonfunctional CHD7 protein, to the perturbation of the regulation of the developmental pathways' genes expression. CHD7 is demonstrated as an important component of neurogenesis through two neuronal determination factors: Sox4 and Sox11. While nonsense, frameshift and missense mutations are most common, deletions and duplications are less frequent. Moreover, while exon 3 is commonly altered, mutations of exon 8, which is related to the CHD7 protein chromodomain, are very rare. Phenotype-genotype correlations according to the type of genomic alteration of CHD7 gene are rarely published, particularly concerning behavioral and psychological features of CHARGE association. Here, physical disorders of our two patients seem to be different but behavioral features seem to be common. Multidisciplinary care is thus required for CHARGE syndrome and molecular analysis must be indicated because the type of the genomic alterations may be a key step for a more accurate management of physical and behavioral disorders.

Disclosure of Interest: None Declared

EPP0460

"… wise, amazed, temp'rate, and furious, Loyal and neutral, in a moment": first heritability analysis of affective temperaments reports remarkably high SNP-based heritability

X. Gonda[1,2]*, D. Torok[3], N. Eszlari[3], D. Gyorik[3], A. Millinghoffer[4], G. Bagdy[3] and G. Juhasz[2,3,5]

[1]Department of Psychiatry and Psychotherapy, Semmelweis University; [2]NAP3.0 Neuropsychopharmacology Research Group, Hungarian Brain Research Program, Semmelweis University; [3]Department of Pharmacodynamics, Semmelweis University; [4]Department of Measurement and Information Systems, Budapest University of Technology and Economics and [5]SE-NAP 2 Genetic Brain Imaging Migraine Research Group, Hungarian Brain Research Program, Semmelweis University, Budapest, Hungary
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.768

Introduction: Depression shows a moderate heritability of 37-42%, which can be up to 75% in severely depressed samples 75%. At the same time SNP-based heritability of depression in GWAS-s is around 8-9%. Heterogeneity of the depressive phenotype may contribute not only to the lack of understanding its genetic background but may also hinder the identification of novel targets. Thus clinically relevant intermediate endophenotypes are needed for. The affective temperaments in the Akiskal model may be considered high-risk states or subclinical manifestations of mood disorders. Considering their strong genetic and biological background, high heritability in family studies, and their temporal stability, they may prove to be relevant endophenotypes for depression.
Objectives: The aim of the current study was to investigate the genetic determinants and heritability of affective temperaments based on a GWAS approach.
Methods: 775 subjects aged between 18-60 years recruited in Budapest, Hungary provided genetic samples and completed