Introduction: There is evidence that the concentrations of the main inhibitory neurotransmitter (GABA) may be altered in schizophrenia. The purpose of this study is to find the changes in the GABA concentration in the area of anterior and posterior cingulate cortex of patients with early-stage schizophrenia using the spectral-edited magnetic resonance spectroscopy.
Objectives: To measure the cerebral concentrations of the gamma-aminobutyric acid in schizophrenia patients at early stage.
Methods: Thirty-one subject, 18 controls (11m+7f, 29.6±5.7 y.o.) and 13 schizophrenia patients (F20.0, 8m+5f, 27.5±3.1 y.o.). Philips Achieva dStream 3T MRI scanner, standard head coil. The 3D T1w head images and MEGA-PRESS GABA spectra in ACC and PCC areas were acquired with the following parameters: 50x25x25 mm, TR = 2 s, TE = 64 ms, 180-editing pulses applied at 1.9 ppm and 7.6 ppm, NSA = 288 (acq.time ~10 min). GABA spectra were processed in Gannet program. The intensities of the GABA, Glutamate+glutamine (Glx), creatine (Cr) and unsuppressed water signals were acquired. T-test was used in search for between-group differences.
Results: In ACC region, significant reduction of the GABA/Water was observed (by ~15%, p=0.02) as well as a trend to a decrease in GABA/Cr (by ~10%, p=0.07) in schizophrenia. In PCC, no significant GABA/Water or GABA/Cr differences were observed. Glx/Water and Glx/Cr in both areas were also unchanged.
Image:

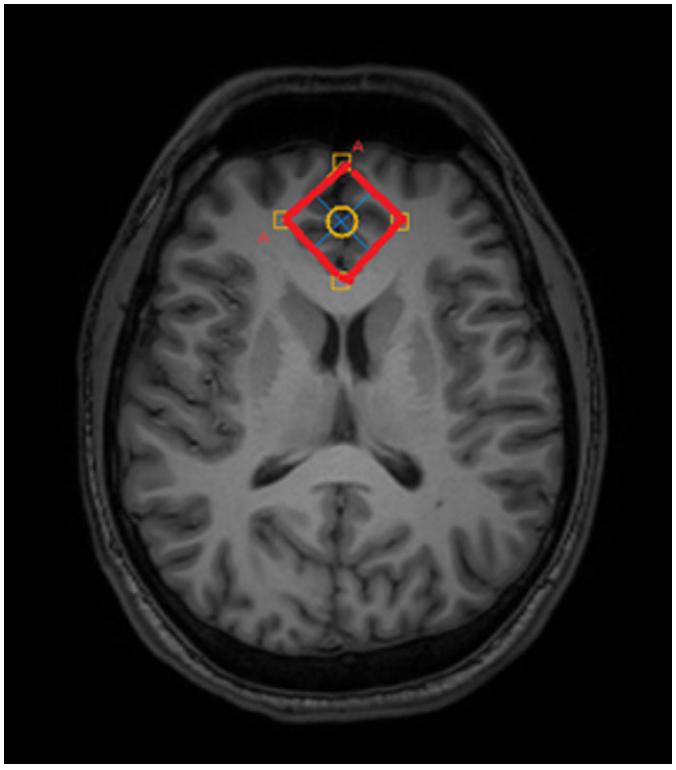
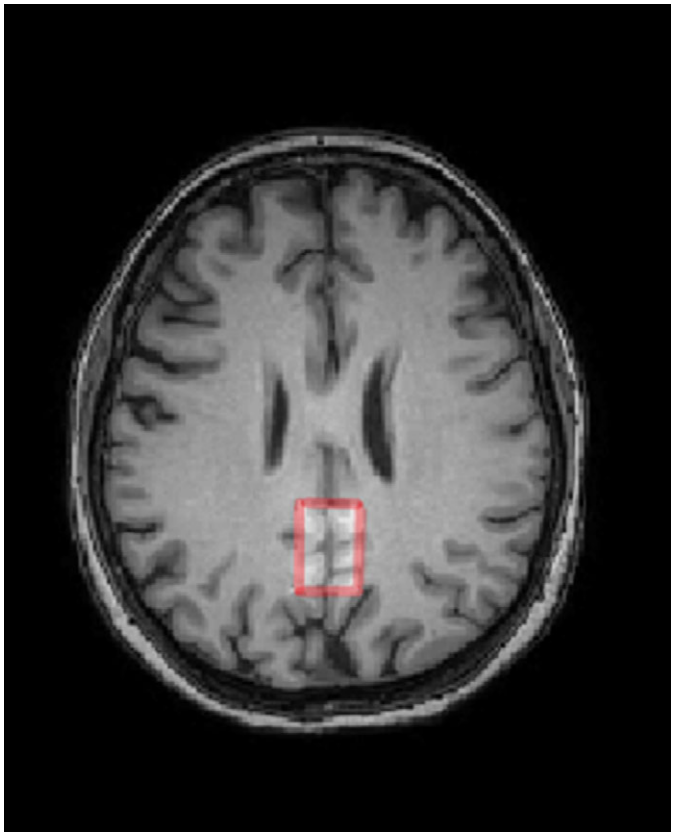


Image 2:



Conclusions: This study provides insight into neurotransmitter alterations at early-stage schizophrenia. The results demonstrate the region-specific changes in the balance of the main neurotransmitters. Since this balance is crucial for the normal cerebral functioning, the results may facilitate better understanding of the dynamics of the pathological process and provide additional information for understanding the biological mechanisms of the schizophrenia development.

Disclosure of Interest: None Declared

EPP0998

Altered Functional Connectivity of Salience Network in Mood Disorders

A. Todeva-Radneva*, R. Paunova, D. Stoyanov, T. Zdravkova and S. Kandilarova

Psychiatry and Medical Psychology and Research Institute, Medical University of Plovdiv, Plovdiv, Bulgaria
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.1273

Introduction: Despite the significant scientific progress in the study of the mechanisms underlying mental disorders, stable biomarkers facilitating their diagnosis and differential diagnosis are lacking. Therefore, we attempted to explore possible functional

connectivity (FC) differences across some of the more prevalent mental disorders, namely mood disorders.
Objectives: The current study aimed at investigating the alterations of the functional connectivity of major seeds of the Salience Network (SN) (the Anterior Cingulate Cortex (ACC), and Anterior Insula (AI) in patients with unipolar (Major Depressive Disorder-MDD) or bipolar (Bipolar Disorder-BD) depression as compared to healthy controls (HC).
Methods: For this study 103 adult subjects underwent resting-state Magnetic Resonance Imaging of whom 60 were patients with a depressive episode (MDD: n=35; BD: n=25), and 43 were healthy controls (HC). The individuals in both groups were matched for age and gender. Each participant has provided written informed consent (Ethics Committee: P186/22.01.2021). Seed-to-voxel analysis was performed via the CONN toolbox running on MATLAB. Random Field Theory parametric statistics was used with a cluster-level FWE correction $p<0.05$.
Results: Both the ACC and the right AI demonstrated a statistically significant increase in the FC to the somatosensory cortex and the motor cortex in patients as opposed to HC. In addition, there was hyperconnectivity between ACC and the right Superior Frontal Gyrus, Precuneus, and the Superior Parietal Lobule bilaterally in patients as well. A reduced FC between the ACC and the hippocampus and parahippocampus was observed in depression in comparison to HC. The analysis of the left AI seed yielded no statistically significant between-group differences.
Conclusions: Our results demonstrate aberrant connectivity between nodes of the SN, the Default Mode Network, and the Limbic Network which might provide clarification on the mechanisms of impaired balance between internally and externally oriented attention, affective and cognitive control in depression. In addition, the alterations of the FC between SN and the somatosensory and motor cortices may be suggested as a possible explanation of the disturbances in the psychomotor activity in mood disorders.

Disclosure of Interest: A. Todeva-Radneva Grant / Research support from: This work has been funded by the Bulgarian National Program "European Scientific Networks" - Project DIP Neuroscience, R. Paunova Grant / Research support from: This work has been funded by the Bulgarian National Program "European Scientific Networks" - Project DIP Neuroscience, D. Stoyanov Grant / Research support from: This work has been funded by the Bulgarian National Program "European Scientific Networks" - Project DIP Neuroscience, T. Zdravkova Grant / Research support from: This work has been funded by the Bulgarian National Program "European Scientific Networks" - Project DIP Neuroscience, S. Kandilarova Grant / Research support from: This work has been funded by the Bulgarian National Program "European Scientific Networks" - Project DIP Neuroscience

EPP0999

Novel mitochondrial mechanisms of cognitive regulation in subjects with cognitive impairments

B. Bigio[1]*, R. Lima-Filho[2], O. Barnhill[3], F. Sudo[2], C. Drummond[4], N. Assunção[4], B. Vanderborght[4], F. Tovar-Moll[4], P. Mattos[4], S. Ferreira[2], F. De Felice[5], M. Lourenco[2] and C. Nasca[1,6]

[1]Department of Psychiatry, New York University Grossman School of Medicine, New York, United States; [2]Institute of Medical Biochemistry Leopoldo de Meis, Rio de Janeiro, Brazil; [3]Rockefeller University, New York, United States; [4]D'Or Institute for Research and Education (IDOR), Rio de Janeiro, Brazil; [5]Queen's University, Kingston, Canada and [6]Department of Neuroscience and Physiology, New York University Grossman School of Medicine, New York, United States
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.1274

Introduction: Prior mechanistic studies in rodents showed decreased levels of the pivotal mitochondrial metabolite acetyl-L-carnitine (LAC) in relation to cognitive deficits and depressive-like behavior (Neuron 2017, 10.1016/j.neuron.2017.09.020, PNAS 2013, 10.1073/pnas.1216100110), providing the basis for the current translational study.
Objectives: The main objective of this work was to ascertain the role of this specific mitochondrial signaling pathway in subjects with cognitive impairments (CI), and potential sex differences in these mechanisms.
Methods: We used computational approaches, ultraperformance liquid chromatography–tandem mass spectrometry (UPLC-MS/MS) and available plasma samples from a well-characterized cohort of 71 subjects, including subjects with CI and age- and sex-matched cognitively healthy controls (HC).
Results: Our newest findings showed decreased levels of LAC in subjects with CI as compared to age- and sex-matched HC. We also found important sex differences in carnitine levels in relation to cognitive function as assessed by using the Mini Mental Status Exam (MMSE). Specifically, the degree of carnitine deficiency reflected the severity of cognitive dysfunction in a sex-specific manner. Using computational approaches, we found that the integration of these mitochondrial measures with canonical biomarkers improves diagnostic accuracy.
Conclusions: The current findings of sex differences in carnitine deficiency in subjects with CI suggest a possible sex-specific mitochondrial phenotype of vulnerability to cognitive dysfunction, and point to LAC-related mitochondrial metabolism as a new signaling pathway of cognitive regulation.

Disclosure of Interest: None Declared

EPP1000

Resting-state gamma oscillations in adult Autism spectrum disorder: A High-Density EEG study

B. Kakuszi[1]*, B. Szuromi[2], M. Tóth[1], I. Bitter[1] and P. Czobor[1]

[1]Psychiatry and Psychotherapy, Semmelweis University and [2]OMIII Nyírő Gyula Hospital, Budapest, Hungary
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.1275

Introduction: Autism is neurodevelopmental disorder with a heterogeneous presentation of symptoms, which include disturbances in sensory, motor and cognitive processes, among which social cognitive impairments and social interaction difficulties play prominent role. Despite the fact that these impairments can lead to lifelong disability and difficulties in everyday functioning, their neurobiological basis remains largely unknown. Neural oscillations in the gamma band have been shown to be an important candidate neurobiological marker of higher order cognitive processes and