protocol was approved by the University's Ethics Committee. High resolution structural MRI was acquired, and preprocessing was performed using SPM 12 toolbox. The structural covariance method was applied consisting of calculation of the correlation across subjects between the different pairs of regions by using the gray matter average volume. We used the threshold statistic to binarize the covariance matrix and transform it into an adjacency matrix. This allows us to compare psychiatric disorders at a network level by calculating measures such as authorities, hubs and outdegree.

Results: 61 statistically significant regions were found for the whole sample. The matrices of the four groups were compared according to their 'authorities' ,'hubs' and 'outdegree' as first, second and third ranking variables, respectively. In the group comparison between HC and BD patients the top five significant regions were Planum temporale (PT), Putamen, Precuneus (PreCu), Calcarine cortex (Calc_cor) and Postcentral gyrus medial segment (PostCGms). The MDD group demonstrated the following regions with most significant difference including Precentral gyrus (PreCG), Entorhinal area (EntA), Amygdala (Amy), Anterior cingulate gyrus (ACC), Anterior insula (AI). While SCH grop was charachterized by ACC, PreCG – medial segment, PostCGms, anterior orbital gyrus, and frontal pole.

Conclusions: The results of our study demonstrated that schizophrenia and mood disorders have specific disturbances in brain network structural organization, affecting hubs of default mode network, salience network, motor, sensory and visual cortex, as well as limbic system. These alterations might elucidate the pathophysiological mechanisms of common symptoms of the disorders under investigation including perceptual, affective and cognitive disturbances.

Disclosure of Interest: None Declared

EPP0344

Modulatory effects of Nigella sativa l. oil on the hippocampus of dizocilpine-induced schizophrenia in BALB/c MICE

R. O. Folarin[1,2*], O. Owoeye[2] and A. Malomo[2]

[1]Anatomy, Olabisi Onabanjo University, Sagamu and [2]Anatomy, University of Ibadan, Ibadan, Nigeria
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.661

Introduction: Schizophrenia is a neuropsychiatric disorder characterised by positive, negative and cognitive behavioral symptoms. Despite years of research, the need for suitable therapy remains elusive. *Nigella sativa* oil (NSO) is a medicinal plant notable for its dietary, neuroprotective and anti-inflammatory properties. However, there is paucity of information on its neuroprotective potentials in schizophrenia.

Objectives: This study was designed to investigate the modulatory effects of NSO on the hippocampus of dizocilpine-induced schizophrenia in mice.

Methods: Sixty 14-weeks old male BALB/c mice (23-25g) were divided into five groups (n=12); control (normal saline, 1 mL/kg), NSO (1 mL/kg), dizocilpine-control (0.5 mg/kg) all for 7 days, while NSO (1 mL/kg for 7 days) + dizocilpine (0.5 mg/kg, for another 7 days) for preventive measure, and dizocilpine (0.5 mg/kg for 7 days) + NSO (1 mL/kg for another 7 days) for reversal. Dizocilpine and NSO were administered intraperitoneally and orally, respectively. Open field box was used for stereotypic popping. Animals were euthanised after behavioral studies, and harvested brains were weighed. Hippocampal glutamate was determined spectrophotometrically. Neuronal arrangement, sizes and densities were determined in perfused brain tissues using haematoxylin and eosin stain. Dendritic arborisations were assessed using Golgi stain. Metabotropic glutamate receptor-II (mGluR-2) and Glia Fibrilliary Acidic Protein (GFAP) were evaluated immunohistochemically. Data were analysed using descriptive statistics and ANOVA at $\alpha_{0.05}$.

Results: Stereotypic popping was observed in dizocilpine-control but not in the preventive and reversal NSO-treated animals. The NSO increased glutamate levels in the reversal (0.19±0.00 μM/μg tissue) but not in the preventive (0.18±0.00 μM/μg tissue) groups relative to dizocilpine-control (0.18±0.00 μM/μg tissue). Hippocampal neuronal density was significantly increased by dizocilpine (21.25±1.11neurons/100μm^2) but modulated by NSO in the preventive (17.25±0.51 neurons/100μm^2) and reversal groups (12.00±0.71 neurons/100μm^2). Significant neuronal de-arborisation that occurred in the dizocilpine-control (989.90±253.9 μm^2/2.5mm^{2}area) was inhibited by NSO in the preventive (1678±370.90 μm^2/2.5mm^{2}area) and reversal (1639±314.80 μm^2/2.5mm^{2}area) treatments. Compared to dizocilpine-control (4219±127.3 ODU), NSO increased mGluR-2 expression in the preventive (4945±17.00 ODU) and reversal (4116±24.97 ODU) groups. The GFAP expression in NSO-treated animals relative to dizocilpine-control (5510±38.45 ODU) was significantly reduced in the preventive (4945±17.00 ODU) and reversal (4116±24.97 ODU) measures.

Conclusions: *Nigella sativa* oil mitigated schizophrenic symptoms induced by dizocilpine in mice via modulation of hippocampal glutamate, metabotropic glutamate receptor–II upregulation, astrogliosis inhibition and neuroprotective mechanisms.

Disclosure of Interest: None Declared

EPP0345

Modulation of excitatory and inhibitory systems in autism spectrum disorder: the role of cannabinoids

S. Marini*, L. D'Agostino, C. Ciamarra and A. Gentile

Mental Health, National Health Service, Termoli, Italy
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.662

Introduction: Autism Spectrum Disorder (ASD) includes a group of developmental disabilities characterized by patterns of delay and deviance in the development of social, communicative, cognitive skills and the presence of repetitive and stereotyped behaviors as well as restricted interests (APA, 2013 DSM 5th ed.). Although the etiopathogenesis of autism has not yet been elucidated, past literature has highlighted an imbalance between glutamatergic and gamma-aminobutyric acid (GABA)-ergic neurotransmission (Harada et al. J Autism Dev Disord 2011;41:447-54.). A cortical deficiency of GABA in young people with ASD has been reported (Rojas et al. Neuroimage 2013;86:28-34.). Endocannabinoids act in numerous synapses of the central nervous system, maintaining an adequate synaptic homeostasis, preventing excess stimulation at the level of excitatory or inhibitory synapses. They therefore appear to be fundamental for the short- and long-term control of synaptic

plasticity (Castillo et al. Neuron 2012;76,70-81). The endocannabinoid system appears to play an important role in some clinical presentations of autism, such as socialization. Indeed, Autism Spectrum Disorder seems to be characterized by a hypofunctionality of the endocannabinoid system (Aran et al. Mol Autism 2019;10, 2).
Objectives: The present work aims to describe the current state of the art regarding the possible role of cannabinoids in the modulation of the excitatory and inhibitory systems in individuals with ASD.
Methods: We carried out a search on PubMed concerning the randomized clinical trials on the modulating effect of excitatory and inhibitory cannabinoid systems in autism. Three eligible articles were found according to the purpose of the present study.
Results: The results of the three articles considered highlighted a cannabinoid (CBD)-related increase in glutamate in subcortical regions (basal ganglia) and a decrease in cortical regions (dorsomedial prefrontal cortex), both in subjects with and without ASD. CBD increased GABA transmission in the subcortical regions of neurotypical subjects, while it decreased it in the same areas of the ASD group. Furthermore, CBD modulated low-frequency activity, used as a measure of brain activity and functional connectivity in the brains of adults with ASD.
Conclusions: Data from the three functional MRI studies demonstrated that CBD influences cortical and subcortical connectivity on an adult sample. This effect was notable only in the ASD group but not in the controls. However, further studies are needed to confirm the results obtained so far.

Disclosure of Interest: None Declared

EPP0346

Anti-amyloid-β Monoclonal Antibodies as Promising Disease-Modifying Therapies in Alzheimer's Disease: A Focus on Aducanumab, Lecanemab, Crenezumab, Gantenerumab and Solanezumab

V. S. D. Melo[1]*, C. A. Rodrigues[1] and I. A. Silva[2]

[1]Psychiatry, Centro Hospitalar do Médio Tejo, Tomar and [2]Psychiatry, Unidade Local de Saúde do Norte Alentejano, Portalegre, Portugal
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.663

Introduction: Alzheimer's disease (AD) is the most prevalent form of age-related dementia in the world. The body of evidence suggesting that its main pathological features consist of amyloid-β (Aβ) plaque deposits and neurofibrillary tangles formed by hyperphosphorylated tau protein is robust. The drugs currently on the market have no effect on disease progression and provide only partial symptomatic relief, which creates a large unmet medical need. Anti-Aβ monoclonal antibodies (mAbs) have been shown to reduce amyloid plaques. Therefore, passive immunization is a major hope for treatment of AD.
Objectives: This review aims to summarize the up to date knowledge and experience with Anti-Aβ mAbs with positive clinical or biomarker effects in long-duration trials.
Methods: A narrative review was conducted based on a search in *Google Scholar* and *Pubmed*, using the following terms or combinations "anti-aβ protofibril antibody"; "early alzheimer's disease"; "immunotherapy for Alzheimer's disease". Peer-reviewed literature published between 2016 and April 2022 was screened on full-text for this purpose.
Results: Aducanumab surpassed a successful Phase 1B trial demonstrating a dose and time dependency for Aß reduction with a beneficial impact on some clinical measures after 1 year of treatment. Two large Phase 3 clinical trials were initiated and already discontinued based on futility analysis done and not based on safety concerns. Further analyses including participants exposed for longer periods of time at higher doses indicated that aducanumab reduced brain amyloid and decreased the rate of decline.
Lecanemab (BAN2401) completed a Phase 2 trial (2018) with evidence of amyloid reduction and slowing of cognitive decline and has now entered Phase 3. Aducanumab and BAN2401 showed significant efficacy on both clinical and biomarker outcomes.
Crenezumab Phase 2 trial results suggested efficacy in mild AD; a Phase 3 program was recently halted due to futility. This mAb is currently being assessed in a prevention trial involving a Colombian kindred with autosomal dominant AD.
Gantenerumab showed significant biomarker effects, with no clinical efficacy reported to date and is being assessed in Phase 3 trials after a trial in prodromal disease stopped for futility suggested that higher doses might be efficacious. Gantenerumab and solanezumab showed no drug-placebo differences in clinical outcomes of specific studies included in this review.
Conclusions: Therapies with anti-Aβ mAbs have been developed successively and conducted in clinical trials signaling a promising new era for AD drug development and providing compelling evidence for the prominent role of neurotoxic soluble amyloid oligomers in the pathogenesis of AD and as therapeutic targets. Lessons learned from these studies may also be a bridge to more efficacious, safe drugs in AD.

Disclosure of Interest: None Declared

Others 02

EPP0348

Cariprazine's efficacy in treating depressive symptoms – pooled data from schizophrenia, bipolar depression and major depression trials

R. S. McIntyre[1], R. Csehi[2]* and G. Németh[2]

[1]Psychiatry and Pharmacology, University of Toronto, Toronto, Canada and [2]Gedeon Richter Plc., Budapest, Hungary
*Corresponding author.
doi: 10.1192/j.eurpsy.2023.664

Introduction: Depressive symptoms are a common feature of schizophrenia (SCH) and define bipolar disorder and major depressive disorder (MDD). Their emergence is related to altered neurotransmission at the serotonin receptors and potentially at dopamine D3 receptors.
Objectives: The aim of this analysis was to examine the efficacy of cariprazine (CAR) in treating depressive symptoms in SCH, bipolar depression (BD) and MDD.
Methods: Clinical trials with randomised, double-blind, placebo (PLB)-controlled designs were included in these analyses. Data from 3 SCH [NCT00694707, NCT01104766, NCT01104779; 1.5-9 mg/d] and 3 BD [NCT01396447, NCT02670538, NCT02670551; 1.5-3