

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 group was characterized 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<sup>1,2\*</sup>, O. Owoye<sup>2</sup> and A. Malomo<sup>2</sup>

<sup>1</sup>Anatomy, Olabisi Onabanjo University, Sagamu and <sup>2</sup>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 Fibrillary 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 ( $0.19 \pm 0.00 \mu\text{M}/\mu\text{g}$  tissue) but not in the preventive ( $0.18 \pm 0.00 \mu\text{M}/\mu\text{g}$  tissue) groups relative to dizocilpine-control ( $0.18 \pm 0.00 \mu\text{M}/\mu\text{g}$  tissue). Hippocampal neuronal density was significantly increased by dizocilpine ( $21.25 \pm 1.11$  neurons/ $100 \mu\text{m}^2$ ) but modulated by NSO in the preventive ( $17.25 \pm 0.51$  neurons/ $100 \mu\text{m}^2$ ) and reversal groups ( $12.00 \pm 0.71$  neurons/ $100 \mu\text{m}^2$ ). Significant neuronal de-arborisation that occurred in the dizocilpine-control ( $989.90 \pm 253.9 \mu\text{m}^2/2.5\text{mm}^2$  area) was inhibited by NSO in the preventive ( $1678 \pm 370.90 \mu\text{m}^2/2.5\text{mm}^2$  area) and reversal ( $1639 \pm 314.80 \mu\text{m}^2/2.5\text{mm}^2$  area) treatments. Compared to dizocilpine-control ( $4219 \pm 127.3$  ODU), NSO increased mGluR-2 expression in the preventive ( $4945 \pm 17.00$  ODU) and reversal ( $4116 \pm 24.97$  ODU) groups. The GFAP expression in NSO-treated animals relative to dizocilpine-control ( $5510 \pm 38.45$  ODU) was significantly reduced in the preventive ( $4945 \pm 17.00$  ODU) and reversal ( $4116 \pm 24.97$  ODU) measures.

**Conclusions:** *Nigella sativa* oil mitigated schizophrenic symptoms induced by dizocilpine in mice via modulation of hippocampal glutamate, metabotropic glutamate receptor-II upregulation, astroglial 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