kynurenic acid (KYNA) between schizophrenia patients (SCZ) and healthy controls (HC). Secondary objective was to explore correlations between these concentrations and clinical characteristics.
Methods: In our two-centre prospective case-control study we measured plasma concentrations of TRP, KYN and KYNA in 36 healthy controls (HC) and 38 schizophrenia (SCZ) patients during acute exacerbation and remission and explored the correlations with clinical parameters using PANSS scale. The patients were matched with HC by age, sex and body mass index and exclusion criteria included obesity class 2 or higher, any concomitant organic mental or neurological disorder, acute or chronic inflammatory disease, and use of immunomodulatory drugs or psychoactive substances.
Results: TRP concentrations were significantly higher in HC than in SCZ patients in acute phase (p<0,001) and remission (p<0,001), while SCZ patients in acute phase had significantly higher TRP levels than in remission (p<0,01). Levels of KYNA and KYN were significantly lower in SCZ patients than in HC both in acute phase and remission, all with high statistical significance (p<0,001). There was no statistically significant difference between acute phase and remission neither for KYN (p>0,05), nor for KYNA (p>0,05). There was no correlation of plasma levels of TRP, KYN and KYNA with total PANSS score, PANSS positive scale score, PANSS negative scale score and PANSS general psychopathology scores, both in acute phase and remission (p>0,05). Also, there was no correlation between plasma levels of TRP, KYN and KYNA in SCZ patients in remission with improvements measured with PANSS scale (p>0,05).
Conclusions: Although there are concerns about the value of measurement of metabolites of kynurenine pathway in the peripheral blood, our data suggest that significantly decreased levels of KYN and KYNA could suggest that disrupted TRP degradation in SCZ patients may be reflected in the peripheral blood as well. Further studies of peripheral levels of kynurenine pathway metabolites on larger samples should also explore effects of antipsychotic therapy, but also their correlation with other clinical parameters such as neurocognition.

Disclosure of Interest: None Declared

EPP0342

Pro-inflammatory markers predict response to sequential pharmacotherapy in major depressive disorder: a CAN-BIND-1 report

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Introduction: Despite replicated cross-sectional evidence of aberrant levels of peripheral inflammatory markers in individuals with major depressive disorder (MDD), there is limited literature on associations between inflammatory tone and response to sequential pharmacotherapies.
Objectives: To assess associations between plasma levels of pro-inflammatory markers and treatment response to escitalopram and adjunctive aripiprazole in adults with MDD.
Methods: In a 16-week open-label clinical trial, 211 participants with MDD were treated with escitalopram 10– 20 mg daily for 8 weeks. Responders continued on escitalopram while non-responders received adjunctive aripiprazole 2–10 mg daily for 8 weeks. Plasma levels of pro-inflammatory markers – C-reactive protein, Interleukin (IL)-1β, IL-6, IL-17, Interferon gamma (IFN)-Γ, Tumour Necrosis Factor (TNF)-α, and Chemokine C–C motif ligand-2 (CCL-2) - measured at baseline, and after 2, 8 and 16 weeks were included in logistic regression analyses to assess associations between inflammatory markers and treatment response.
Results: Pre-treatment levels of IFN-Γ and CCL-2 were significantly higher in escitalopram non-responders compared to responders. Pre-treatment IFN-Γ and CCL-2 levels were significantly associated with a lower of odds of response to escitalopram at 8 weeks. Increases in CCL-2 levels from weeks 8 to 16 in escitalopram non-responders were significantly associated with higher odds of non-response to adjunctive aripiprazole at week 16.
Conclusions: Pre-treatment levels of IFN-Γ and CCL-2 were predictive of response to escitalopram. Increasing levels of these pro-inflammatory markers may predict non-response to adjunctive aripiprazole. These findings require validation in independent clinical populations.

Disclosure of Interest: None Declared

EPP0343

Disrupted structural brain networks across psychiatric disorders determined using multivariate graph analyses

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Introduction: Identifying the specific brain pattern characterizing psychiatric disorders could lead us to precise diagnostic process, better treatment plan and outcome prediction. Structural covariance is a graph-analysis method with which disruptions in large scale brain network organization can be observed. More studies, employing this method in psychiatry, are still needed.
Objectives: The current study aims to investigate how the main psychiatric disorders – schizophrenia, major depressive disorder, bipolar disorder, affect brain circuitry by means of multivariate graph theory, more specifically – structural covariance. We hypothesized that specific abnormalities in the brain circuits would be found in separate diagnostic entities.
Methods: 164 subjects were included with schizophrenia-SCH (n=17), bipolar disorder-BD(n=25), major depressive disorder–MDD(n=68) and a healthy control group-HC(n=54). Each participant provided a written informed consent and the study

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

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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

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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