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