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Introduction: Oxidative stress is the main characteristic of several diseases including Bipolar Disorder (BD). The involvement of oxysterol derivatives has recently been reported. In this study, the involvement of oxidative stress in the alteration of cholesterol in PTB patients will be evaluated.

Objectives: To assess the association of oxidative stress and oxysterol profiles in subjects with BD and compare them to healthy physical and mental controls.

Methods: This is a case-control study involving subjects with BD. Selected based on DSM-5 criteria, an assessment of positive and negative symptoms was performed using the Positive and Negative Syndrome Scale (PANSS). Controls included in this study were matched to patients by age and gender. For all patients and control. Eight parameters of oxidative status were assessed: plasma ferric reducing capacity (FRAP), carbonyl proteins (PC), protein products of advanced oxidation (AOPP), reduced glutathione (GSH), total thiols, malondialdehyde (MDA), glutathione peroxidase activity (GSH-Px) and catalase activity (CAT) analyzed by colorimetric methods. In addition, six cholesterol derivatives: oxysterols are measured by ULPC MS/MS.

Results: This study included 33 patients with BD and 40 controls. Plasma GSH levels were significantly reduced in patients compared to controls ($p < 0.001$). Moreover, MDA, AOPP, PC and GSH-Px activity were significantly increased in patients compared to controls ($p=0.005$; $p=0.003$; $p<0.001$ and $p=0.05$, respectively). Significantly higher levels were observed for cholestane- 3β , 5α , 6β -triol, 27-hydroxycholesterol (27-OHC), and cholestanol in patients with PTB. The concentration of 24(S)-hydroxycholesterol (24-OHC) was significantly lower in patients compared to controls. 25-OHC was positively and significantly correlated with CAT and GSH-Px activities ($p=0.035$ and $p=0.010$). 27-OHC was negatively and significantly correlated with MDA ($p=0.014$). Binary logistic regression revealed an association between the parameters: 27-OHC, 24-OHC, PC and MDA and the occurrence of PTB (OR = 1.007, 95% CI= 1.002-1.013), (OR = 0.956; 95% CI = 0.927 - 0.986), (OR = 39.925; 95% CI = 1.101 - 44.483) and (OR = 4.238; 95% CI = 1.091 - 16.466), respectively.

Conclusions: Our data support the relationship between disruption of redox homeostasis and oxidation of lipids and cholesterol in BD.

Disclosure of Interest: None Declared

EPP0532

Coping strategies in bipolar patients: A comparative study with siblings and healthy controls

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Introduction: Data regarding coping strategies used by bipolar patients to deal with psychosocial stress and their consequences in adaptational outcomes are scant. Moreover, family studies have reported the presence of several similarities between bipolar patients and their relatives regarding genetics, biology, personality traits, temperaments and stressful lived life experiences. Bipolar patients and their siblings had significantly higher global score of life events and more events in the field of work, socio-family events and health than control subjects. This might suggest that patients with bipolar disorder would be distinguished from their family members by the coping strategies they use to deal with stress.

Objectives: In this study, we aimed to compare perceived stress and coping strategies of remitted bipolar I patients with those of their siblings and controls.

Methods: A descriptive and comparative study of case-control type was conducted. Were included 46 bipolar I patients, 46 siblings and 50 controls. The three groups were matched for age and sex. Assessments of perceived stress and coping strategies were performed using the 10-item Perceived Stress Scale (PSS) and the 28-item Brief COPE respectively.

Results: Mean age of bipolar I patients was 39 ± 13 years. Thirty-one patients (67%) reported family history of one or more psychiatric disorders. Mean duration of bipolar disorder was 11.83 ± 9.92 years.

There was no significant difference between the three groups on PSS scores. Bipolar patients and siblings were more likely to use emotion-focused coping strategies than controls ($p=0.001$). Controls used problem-focused coping strategies more than bipolar patients ($p = 0.02$). Compared to controls, bipolar patients were less likely to use active coping and planning, but they showed higher scores in the dimensions of humor, religion and behavioral disengagement with intergroup p value: **0.02; 0.019; 0.002** respectively.

Conclusions: Our findings suggest that bipolar I patients were more likely to use maladaptive coping strategies to deal with stress than their siblings. Based on this observation, it seems advisable to study coping strategies used by bipolar patients, in order to reinforce adaptive strategies and to reduce maladaptive ones.

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EPP0533

Gut permeability and low-grade inflammation in bipolar disorder

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Introduction: Systemic inflammation has been increasingly related to bipolar disorder -BD- (Tanaka et al. *Neurosci Res* 2017;115

59-63). Intestinal bacterial translocation has been postulated as one of the causes of this inflammation (Nguyen et al. *J Psychiatr Res* 2018;99 50-61). A possible pathway is through the lipopolysaccharide, which is presented to CD14 through lipopolysaccharide binding protein (LBP) leading to a release of systemic inflammatory markers like C-reactive protein (CPR) (Funda et al. *Infect Immun* 2001;69 3772-81).

Objectives: 1) Describe gut permeability in patients with BD through the determination of intestinal inflammatory markers (LBP, sCD14) in plasma; 2) Analyze variables associated with intestinal inflammation.

Methods: Cross-sectional study of 38 patients with BD [mean age=45.50 (SD=10.93; range 23-68); males=15 (39.5%)], recruited from mental health outpatient clinics in Oviedo (Spain).

Assessment: Pro-inflammation biomarkers [CRP (mg/dL), Erythrocyte Sedimentation Rate (ESR) (mm/h), Neutrophil/Lymphocyte, Monocyte/Lymphocyte, Platelet/Lymphocyte and Systemic Immune Inflammation Indexes]. Indirect markers of intestinal bacterial translocation [LBP, soluble CD14 (sCD14)]. Dichotomous variables were created for LBP, considering LBP ≥ 15 $\mu\text{g/dL}$ as increased gut permeability; and for CPR, considering $\text{CRP} \geq 0.3$ as systemic inflammation. Metabolic syndrome [ATPIII criteria: glucose, HDL, triglycerides (mg/dl), arterial pressure (mmHg), abdominal circumference (cm)], body mass index (BMI) (kg/m²), smoking, cannabis or alcohol use. Statistical analyses: t-Student test, multiple linear regression analyses.

Results: Average LBP was 14.60 $\mu\text{g/dL}$ (SD=6.4) and 15 patients (39.5%) had increased gut permeability. Moreover, average CPR was 0.40 mg/dL (SD=0.58) and 16 patients (47.1%) showed systemic inflammation. There were no patients with increased levels of sCD14.

Associations were found between LBP and CPR ($r=0.357$; $p=0.032$), cannabis use in the last month ($t=-2.293$; $p=0.029$), BMI ($r=0.433$; $p=0.008$) and abdominal obesity ($t=3.006$; $p=0.005$); but no with age or sex.

Subsequently, a multiple linear regression model for LBP was calculated with variables previously mentioned, and age (based on expert criteria). The overall regression was statistically significant ($R^2=0.49$, $F=9.273$, $p<0.001$). It was found that CPR, abdominal obesity, and cannabis use in the last month significantly predicted LBP levels (table 1).

Table 1. Multiple linear regression analyses to LBP

	B	SE	β	t	p
CPR	4.842	1.529	0.439	3.167	0.004
Abdominal obesity	4.810	1.849	0.362	2.601	0.014
Cannabis use	-5.048	2.273	-0.296	-2.221	0.034

Conclusions: More than one third of patients with BD had increased gut permeability. Almost 50% had systemic inflammation. Intestinal permeability was directly related to abdominal obesity and systemic inflammation, but inversely related to cannabis use.

Disclosure of Interest: None Declared

EPP0534

Genetic underpinnings of YMRS and MADRS scores variations in a bipolar sample

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Introduction: Bipolar disorder (BD) is a chronic hereditary disorder. Trial and error principles and long period of untreated disorder mandate further research. Relatively recent advances in statistical computing and techniques introduced Polygenic risk scores (PRS) as predictors of the genetic susceptibility to diseases. Although they provide an estimate of the risk of developing specific pathologies, they are a genome-wide measure. PRS do not provide specific information on the biological meaning of the variants. The use of subsets of risk variants (limited to one or few related biological pathways) to calculate pathway-PRS (pPRS) may provide an estimate of the functioning of specific molecular cascades.

Objectives: In the present study we calculated pPRS and tested them as potential predictive factors which, together with other clinical/environmental features, may estimate the treatment outcome of BD individuals in a clinical realistic treatment environment.

Methods: 1538 BD (41.39 \pm 12.66 years, 59.17% females) individuals from STEP-BD were included in the analysis. A latent class analysis identified three groups of patients according to the YMRS and MADRS scores variations during ~ 1 year (308.47 \pm 293.83 days YMRS, 357.78 \pm 367.76 days MADRS). A GWAS analysis with clinical covariates provided the input for pPRS calculation. SNPs with best nominal significance and biologic relevance were prioritized through GTEx. A molecular pathway analysis (MPA) based on the interaction network of drugs used for treatment provided the genetic data needed for pPRS calculation. A Neural network was built using pPRS as features together with other variables (including Sex, Age, Scores at baseline) to predict the 3 groups previously identified. Performance was evaluated through 5-fold cross-validation, Python, R and Bash served for environments. Gene Ontology, ReactomePA and Bioconductor were key packages together with Cytoscape, Plink, impute and gtool.

Results: Ten biological networks were retrieved from MPA: 1) GO:0016705 + GO:0016641, 2)GO:0019585, 3)GO:003018, 4) GO:0099589 + GO:1904014, 5)GO:0015464 + GO:1905144, 6) GO:0004935 + GO:0004364 + GO:00031690, 7)GO:1903351 + GO:1903350, 8)GO:0016917 + GO:0007214, 9)GO:0008066 + GO:0007215, 10)GO:0048016. Risk variants within the genes contained in each group were used to compute pPRS. The ten pPRS were used to compute a neural network to predict treatment outcomes.

Conclusions: BD treatment is influenced by socio-demographic, clinical and genetic factors. To tackle this complexity, we tried to implement an approach where the multivariate analysis encompasses clinical analysis and the biologic background of treatment