

## EPP0984

### Depressive disorders in patients with sleep apnea syndrome

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**Introduction:** A growing body of literature has documented that obstructive sleep apnea syndrome (OSAS) is associated with an increased risk of depressive disorders.

**Objectives:** This study aimed to determine whether depression is associated with OSAS.

**Methods:** This was a descriptive prospective comparative study conducted over two years among patients with OSAS. Excessive daytime sleepiness was assessed by the Epworth Sleepiness Score (SES). The presence and intensity of depressive symptoms were screened using the Patient Health Questionnaire PHQ9. Data were analysed using SPSS software.

**Results:** A total of 139 patients participated in the survey with an average age of  $48.98 \pm 9.80$  years. According to the SES, the study population was divided into two groups: a group including 70 subjects with normal SES ( $< 11$ ); and a group including 69 subjects with pathological SES ( $\geq 11$ ). The PHQ9 depression score was higher in sleepy subjects with  $SES \geq 11$  compared to non-sleepless subjects; the difference being very highly significant ( $PHQ9=11.97 \pm 4.99$  and  $6.54 \pm 5.27$  respectively;  $p=0.0000$ ). The frequency of mild to moderate depressive disorders was found to be greater in non-sleepy subjects (94.3% and 78.3% respectively;  $p=0.007$ ). For moderately severe to severe depression, their frequency was more marked in sleepy subjects (21.7% and 5.7% respectively;  $p=0.007$ ).

**Conclusions:** Depressive disorders constitute a major comorbidity in OSAS. Therefore, it is necessary to improve the quality of these patients' health by the early detection of the symptoms of overlapping OSAS and depression.

**Disclosure of Interest:** None Declared

## EPP0985

### Association between Diffusion Tensor Imaging, inflammation and immunological alterations in unipolar and bipolar depression: a review

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**Introduction:** Major Depressive Disorder (MDD) and Bipolar Disorder Depression (BDD) are common psychiatric illnesses characterized by structural and functional brain alterations and

signs of neuroinflammation. In line with the neuroinflammatory pathogenesis of depressive syndromes (Mechawar N, Savitz J. Neuropathology of mood disorders: do we see the stigmata of inflammation? *Transl Psychiatry*. 2016;6(11):e946), recent studies have demonstrated how white matter (WM) microstructural impairments detected by Diffusion Tensor Imaging (DTI) are correlated to peripheral immunomarkers in depressed patients.

**Objectives:** In this context, the aim of our review is to report an updated overview of the evidence on the correlation between the blood immuno-markers changes and the brain WM disruptions in MDD and BDD patients.

**Methods:** Based on PRISMA 2020 guidelines (Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ*. 2021;372), we performed a systematic search on original DTI studies exploring the association between WM integrity, neuroimmune alterations and inflammation in patients affected by MDD or BDD.

**Results:** Concerning MDD, most of the reviewed studies provided evidence of a link between systemic immune dysregulation, detected through the elevation of peripheral markers (IL-1 $\beta$  and TNF- $\alpha$ ) or an altered ratio between proinflammatory and counterregulatory cytokines (IL-8/IL-10), and DTI alterations in specific WM tracts, such as the genu of corpus callosum and the IFOF. As for the BDD, we detected an increase of pro-inflammatory molecules (such as TNF- $\alpha$ , IL-8, IFN- $\gamma$  etc.) that correlated with DTI changes in different cerebral areas such as cingulum, forceps, corona radiata, corpus callosum, longitudinal fasciculus and internal capsule. Furthermore, other molecules seem to play a specific role in BDD pathogenesis, including counter-regulatory cytokines, kynurenine and specific lymphocyte classes, such as Th1 and Th17.

**Conclusions:** Taken together, these pathogenetic insights could outline an integrated clinical perspective to affective disorders, helping psychiatrists to develop novel biotype-to-phenotype models of depression and opening the way to tailored approaches in treatments.

**Disclosure of Interest:** None Declared

## EPP0987

### Treatment of Seasonal Affective Disorder. The efficacy of Light therapy

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**Introduction:** Seasonal affective disorder (SAD) is a type of depression that comes and goes in a seasonal pattern. Symptoms of SAD can include: a persistent low mood, a loss of pleasure or interest in normal everyday activities, irritability, feelings of despair, guilt and worthlessness, feeling lethargic (lacking in energy) and sleepy during the day, sleeping for longer than normal and finding it hard to get up in the morning, craving carbohydrates and gaining weight, difficulty concentrating.

**Objectives:** The purpose of this study was to evaluate the response to different therapeutic interventions of seasonal depression

**Methods:** A biographical review was performed using the PubMed platform. All relevant articles were found using the keywords: seasonal affective disorder, treatment, and winter depression.

**Results:** The main treatments are: lifestyle measures – including getting as much natural sunlight as possible, exercising regularly and managing your stress levels, light therapy – where a special lamp called a light box is used to simulate exposure to sunlight, talking therapies – such as cognitive behavioral therapy (CBT) or counseling, antidepressant medicine – such as selective serotonin reuptake inhibitors (SSRIs)

**Conclusions:** Light therapy can be a very effective treatment for SAD, with most seeing an improvement of symptoms within the first week. A powerful lamp that replicates natural light, high-quality light boxes are recommended as they allow patients to spend a shorter time (up to 30 minutes at a time) using them.

**Disclosure of Interest:** None Declared

## EPP0988

### Lower thyroid stimulating hormone concentrations linked to suicidal ideations among individuals with anxiety and mood disorders

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**Introduction:** Suicidal behavior is quite common in individuals with anxiety and mood disorders (AMD). One of the coexistence factors in suicidal behavior is thyroid dysfunction, however the results are still controversial (Shen et al. *J Affect Disord* 2019;(1) 180-185; Zhou et al., *Transl Psychiatry*. 2021;11(1):97). The role of thyroid function in suicidal ideation among individuals with comorbid AMD have not been thoroughly investigated.

**Objectives:** The aim of this cross-sectional study was to identify potential associations between thyroid function and suicidal ideation in a sample of individuals with anxiety and mood disorders.

**Methods:** This exploratory study comprised 77 consecutive individuals with AMD (age range 18-73 years, 76% were females) attending the Psychiatry Day care unit. All individuals have been evaluated for current psychiatric diagnoses, suicidal ideation using the Mini International Neuropsychiatric Interview [M.I.N.I. 7.0.2]) as well as for socio-demographic factors and for current psychotropic medication use. Severity of depression and anxiety symptoms have been evaluated using the Patient Health Questionnaire-9 (PHQ-9) and the General Anxiety Disorder-7 (GAD-7). The biochemical blood tests were performed for the concentrations of thyroxine (FT4), triiodothyronine (FT3) and thyroid stimulating hormone (TSH). The univariate and multivariable logistic regression analyses were used to assess the association between biochemical parameters and suicidal ideation.

**Results:** Of all study individuals with AMD – 56% have been identified as having current SI. There were not significant differences according to age, gender, education, BMI, smoking, depression and anxiety symptoms and current psychotropics use between

SI and non-SI individuals. Serum FT4, FT3 and TSH concentrations were within normal range. However individuals with SI had significantly lower TSH concentrations in comparison to the non-SI (1.54 (0.77) vs. 2.04 (1.22) IU/L, respectively;  $p = 0.049$ ), without significant differences in FT4 and FT3 concentrations. A multiple logistic regression, adjusting for sociodemographic factors and severity of mental symptoms revealed, that non-SI individuals with AMD were likely to have higher TSH levels than SI (odds ratio = 2.15 (95% CI 1.10–4.22;  $p = 0.027$ ).

**Conclusions:** Among individuals with AMD, lower levels of TSH concentrations have been associated with presence of suicidal ideation, independently of sociodemographic factors and severity of depression and anxiety.

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## EPP0989

### Safety and Tolerability of Intramuscular and Sublingual Ketamine for Psychiatric Treatment in the Roots to Thrive Ketamine Assisted Therapy Program

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**Introduction:** Ketamine has been increasingly used to treat mental health conditions yet there is a lack of safety data on intramuscular (IM) and sublingual (SL) dosing in a community setting. The Roots to Thrive Ketamine assisted Therapy (RTT-KaT) program is a 12-week program with 12 Community of Practice (CoP) group therapy sessions and three ketamine sessions.

**Objectives:** To provide preliminary data on RTT-KaT adverse events to subsequently inform safe use of IM and SL ketamine for the treatment of psychiatric disorders.

**Methods:** Retrospective chart review of the RTT-KaT Program on four cohorts ( $n=128$ ) between September 2020 to December 2021. Eligible patients include those with post-traumatic stress disorder, depression, generalized anxiety, burnout/adjustment disorder, substance use disorder, obsessive compulsive disorder, disordered eating, and disordered sleep. Baseline characteristics and adverse events were captured including medication administration before, during, and after RTT-KaT sessions. Chi-squared test with Yates' continuity correction was used to assess side effects in subgroups from ketamine administration.

**Results:** RTT-KaT was well tolerated with no loss to follow up. There were 351 IM (mean dose = 102.553mg) and 96 SL (mean dose = 276.667mg) sessions of ketamine. Of the 448 sessions, the prevalence of elevated blood pressure increased by 12.31% from baseline (36.85%), with all post-treatment elevations being transient. The prevalence of elevated blood pressure post-KaT session was also similar between IM (+11.69% from 37.71% baseline) and SL (+15.12% from 32.98% baseline) administration. Regarding