

**Methods:** Literature review on adult ADHD and comorbid substance abuse.

**Results:** A 43-year-old male who consulted in the Emergency Department due to auditory hallucinosis in the context of an increase in his daily cocaine use. There were not delusional symptoms associated and judgment of reality was preserved. Treatment with olanzapine was started and the patient was referred for consultation. In psychiatry consultations, he did not refer sensory-perceptual alterations anymore, nor appeared any signals to suspect so, and he was willing to abandon cocaine use after a few appointments. He expressed some work concerns, highlighting that in recent months, in the context of a greater workload, he had been given several traffic tickets for “distractions.” His wife explained that he had always been an inattentive person (he forgets important dates or appointments) and impulsive, sometimes interrupting conversations. In the Barkley Adult ADHD Rating Scale he scored 32 points.

He was diagnosed with adult ADHD and treatment with extended-release methylphenidate was started with good tolerance and evolution, with improvement in adaptation to his job and social environment. Since then, the patient has moderately reduced the consumption of drugs, although he continues to use cocaine very sporadically.

**Conclusions:** Early detection of ADHD and its comorbidities has the potential to change the course of the disorder and the morbidity that will occur later in adults. Comorbidity in adult ADHD is rather the norm than the exception, and it renders diagnosis more difficult. The most frequent comorbidities are usually mood disorders, substance use disorders, and personality disorders. Treatment of adult ADHD consists mainly of pharmacotherapy supported by behavioral interventions. When ADHD coexists with another disorder, the one that most compromises functionality will be treated first and they can be treated simultaneously. The individual characteristics of each patient must be taken into account to choose the optimal treatment.

**Disclosure of Interest:** None Declared

## EPP0424

### Clinical overlap between functional neurological disorders and autism spectrum disorders: a preliminary study

D. Goeta<sup>1\*</sup> and B. Demartini<sup>2,3</sup>

<sup>1</sup>U.O. di Psichiatria, Presidio San Carlo; <sup>2</sup>U.O. di Psichiatria 52, Presidio San Paolo, ASST Santi Paolo e Carlo and <sup>3</sup>Aldo Ravelli Research Center for Neurotechnology and experimental brain therapeutics, University of Milan, Milan, Italy

\*Corresponding author.

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**Introduction:** Functional neurological disorders (FNDs) and autism spectrum disorders (ASDs) share common features in terms of deficits in emotion regulation and recognition, sensory sensitivity, proprioception and interoception. Nevertheless, few studies have assessed their overlap.

**Objectives:** Aims of the present study were: (i) to assess the prevalence of autistic traits in a sample of adult patients with FNDs

and (ii) to assess the prevalence of FNS in a sample of adult individuals with ASDs without intellectual disabilities; in this sample, we also evaluated the presence of a possible association between sensory sensitivity and FNS.

**Methods:** We recruited 21 patients with FNDs, 30 individuals with ASDs without intellectual disabilities and 45 neurotypical adults (NA). Participants completed: the Autism Quotient (AQ); the Ritvo Autism Asperger Diagnostic Scale-Revised (RAADS-R); and a questionnaire assessing functional neurological symptoms (FNS). ASDs participants also completed the Sensory Perception Quotient-Short Form (SPQ-SF35), assessing sensory sensitivity.

**Results:** In the FNDs sample, no patient scored above the clinical cut-off at the AQ and the 19% scored above the cut-off at the RAADS-R, a prevalence similar to the one we found in NA (15.6%; both  $p > 0.05$ ). The 86.7% of participants with ASDs reported at least one FNS, a prevalence significantly higher than the NA one (35.6%,  $p < 0.001$ ). In the ASDs sample, tactile hyper-sensitivity was found to be a risk factor for functional weakness (OR = 0.74,  $p = 0.033$ ) and paraesthesia (OR = 0.753,  $p = 0.019$ ).

**Conclusions:** In conclusions, FNDs individuals did not present autistic traits more than NA, but ASDs individuals presented a higher number of FNSs than NA; this rate was associated with higher sensory sensitivity, especially in the touch domain.

**Disclosure of Interest:** None Declared

## EPP0425

### Quality of life and psychosocial adjustment: the moderator role of anxiety symptoms in persons with temporal lobe epilepsy due to hippocampal sclerosis

E. M. Lima\*, J. Gois, S. Vincentiis and K. D. R. Valente

<sup>1</sup>Department of Psychiatry, University of Sao Paulo, Sao Paulo, SP, Brazil

\*Corresponding author.

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**Introduction:** Persons with temporal lobe epilepsy due to hippocampal sclerosis (TLE-HS) have a high frequency of psychiatric disorders, namely depression and anxiety. In addition, poor quality of life (QOL) and impairments in psychosocial adjustment are frequently reported. Despite the increasing number of studies aiming to identify predictors of poorer QOL, the role of depressive and anxiety symptoms remains poorly understood.

**Objectives:** This study aimed to evaluate: (1) if psychosocial adjustment predicts worse QOL; (2) the relationship between psychosocial adjustment and QOL by concurrently examining the role of depressive and anxiety symptoms.

**Methods: Participants:** Thirty-five persons with TLE-HS ranging from 18 to 60 years old (mean age 39.82 SD 9.05; 20 men 57.14%) followed in a tertiary outpatient center underwent neurologic and psychiatric assessments.

**Assessments:** The psychiatry interview was performed using the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders (SCID). Psychosocial adjustment was assessed with the total score of the Social Adjustment Scale (SAS) (SAS Overall). The QOL was evaluated by the total score of the Quality of Life in Epilepsy-31 Inventory (Overall QOLIE-31), the most

frequently used instrument in assessing QOL in epilepsy. The Beck Depression Inventory (BDI) and the State-Trait Anxiety Inventory (STAI-X) were used to assess depressive and anxiety symptoms, respectively.

**Analyses:** Prediction analysis was performed to evaluate the impact of psychosocial adjustment on QOL (simple linear regression). Simple moderation models were used to examine the moderation effect of depressive and anxiety symptoms on the association between PA and QOL (Figure 1). We used SPSS (version 29 IBM Corp) and PROCESS Macro (version 4.1. for SPSS) to perform regression and moderation analyses (Figures 2 and 3), respectively.

**Results:** Poor psychosocial adjustment (higher scores on SAS) impacted on poor QOL (lower scores on QOLIE-31) ( $R=0.39$ ;  $R^2=0.15$ ; adjusted  $R^2=0.12$ ;  $B=-0.39$ ;  $t=-2.28$ ;  $p=0.03$ ). The severity of anxiety symptoms (Trait and State; coefficient=-0.64;  $t=-2.01$ ;  $p=0.05$  and coefficient=-1.17;  $t=-2.20$ ;  $p=0.03$ , respectively), but not the severity of depressive symptoms (coefficient=0.77;  $t=1.37$ ;  $p=0.18$ ), moderated the relationship between psychosocial adjustment and QOL.

**Image:**

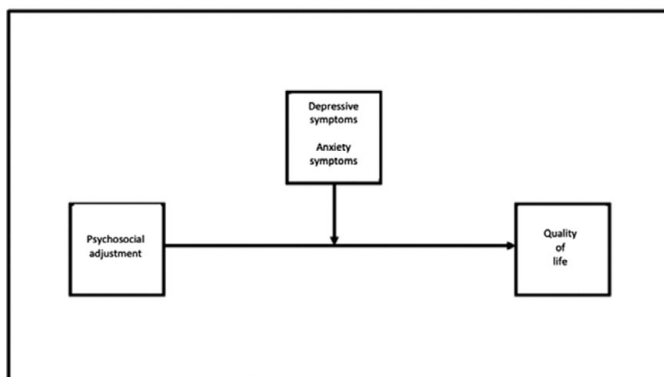
Figure 1. Variables of moderation model studied

Predictor (X)	Outcome (Y)	Moderators* (W)
Psychosocial Adjustment (Social Adjustment Scale score)	Quality of Life (Quality of Life in Epilepsy-31 Inventory score)	Depressive symptoms (Beck Depression Inventory score)  Anxiety symptoms (State-Trait Anxiety Inventory State score and State-Trait Anxiety Inventory Trait score)

\*It was considered separately, for the Models 1, 2 and 3

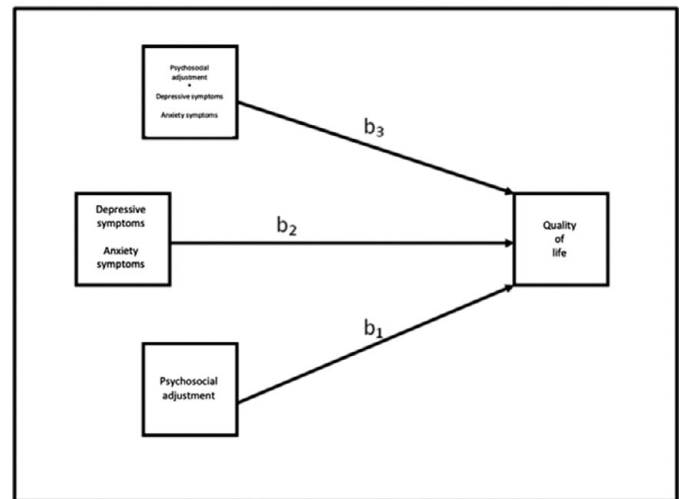
**Image 2:**

Figure 2. Moderation model diagram (PROCESS, Model 1) adopted



**Image 3:**

Figure 3. Moderation statistical diagram (PROCESS, Model 1) adopted



**Conclusions:** Psychosocial adjustment is a predictor of QOL in TLE-HS. Anxiety symptoms moderate this relationship between psychosocial adjustment and QOL. Consequently, higher anxiety symptoms are associated with worse psychosocial adjustment and quality of life.

**Disclosure of Interest:** None Declared

COVID-19 and related topics 04

EPP0427

Relationships of eeg and immunological parameters in depressive patients who survived COVID-19

A. F. Iznak<sup>1\*</sup>, E. V. Iznak<sup>1</sup>, E. V. Damyanovich<sup>1</sup>, S. A. Zozulya<sup>2</sup> and I. V. Oleichik<sup>3</sup>

<sup>1</sup>Laboratory of Neurophysiology; <sup>2</sup>Laboratory of Neuroimmunology and <sup>3</sup>Department of endogenous disorders, Mental Health Research Centre, Moscow, Russian Federation

\*Corresponding author.

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**Introduction:** Coronavirus infection affects the CNS and modulates the immune system. The associated processes of neuroplasticity play an important role in the pathogenesis of depression.

**Objectives:** The aim of the study is to identify the relationships between EEG and immunological parameters in depressive patients who recovered from coronavirus infection, in order to clarify the features of neuroimmune interaction after suffering COVID-19.

**Methods:** 30 female patients aged 16-25 enrolled in the study were admitted for treatment during the pandemic in 2020-2022 ("COVID" group). Previously, they had been ill with COVID-19 in a mild or asymptomatic forms on a background of depressive state (F31.3-4, F21.3-4 + F34.0, according to ICD-10) from 3 months to 2 years before the examination.