pharmacotherapy, while 20 received pharmacotherapy only. Blood samples were collected on the first and last days of hospitalization to measure serum TBARS. Sociodemographic and clinical data were recorded at admission and CGI (Clinical Global Impression), PANSS (Positive and Negative Symptom Scale) scales were applied. Analyses were conducted with SPSS v28. Non-normally distributed data (Shapiro–Wilk) were tested using Mann–Whitney U, Wilcoxon signed-rank, chi-square, Spearman correlation, and multiple linear regression; with significance at $p<0.05$. (The ethics committee approval number is TABED 1-24-306)

Results: Of the 38 patients, 18 received ECT plus medication, while the remaining 20 received medication alone. No significant differences were found between groups in terms of age, total duration of disorder, years of education, PANSS, CGI scores, or suicide history (all $p>0.05$). However, clozapine use was higher in the ECT group ($\chi^2=4.374$, $p=0.036$). TBARS levels showed no significant change from admission to discharge in either group (ECT group: 756.5 vs. 768.5 nmol/L; $Z=-1.136$, $p=0.256$; pharmacotherapy group: 644.0 vs. 693.5 nmol/L; $Z=-1.512$, $p=0.130$). There was also a similar percentage change in TBARS levels between groups (%9.6 vs. %15.3; $Z=-0.380$, $p=0.704$). Negative correlations were found between TBARS levels and both age ($\rho=-0.441$, $p<0.001$) and the duration of the disorder ($\rho=-0.333$, $p<0.05$). Regression analysis showed suicide history as the only significant predictor of TBARS levels ($\beta=-0.402$, $p=0.013$).

Conclusions: TBARS levels did not significantly change following either ECT or pharmacological treatment in schizophrenia inpatients, suggesting limited impact of ECT on oxidative stress as measured by lipid peroxidation. However, associations with age, illness duration, and history of suicide suggest potential relevance of oxidative stress markers in subgroups of patients.

Disclosure of Interest: None Declared

EPP365

Effect of Intermittent Theta-Burst Stimulation on Chronobiological Hypothalamic-Pituitary-Thyroid Axis Activity in Treatment-Resistant Depression

F. Duval*, M.-C. Mokrani, V. Danila, T. Weiss, F. Gonzalez Lopera and M. I. Tomsa

Pole 8/9 - APF2R, Centre Hospitalier, Rouffach, France
*Corresponding author.
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Introduction: The effects of intermittent theta-burst stimulation (iTBS), a form of repetitive transcranial magnetic stimulation, on the hypothalamic-pituitary-thyroid (HPT) axis are not well understood. This axis is frequently dysregulated in treatment-resistant depression (TRD). We previously demonstrated that the difference in thyrotropin (TSH) response to protirelin (TRH) between 23 h and 8 h on the same day (the ΔΔTSH test) is a highly sensitive chronobiological marker.

Objectives: To evaluate the impact of iTBS targeting the left dorsolateral prefrontal cortex (LDPFC) on chronobiological HPT activity in hospitalized TRD patients.

Methods: Twenty-three TRD inpatients with abnormal ΔΔTSH values (< 2.5 mU/L) and HAMD-17 scores ≥ 18 were compared to 23 matched healthy controls (HCs). Patients received 20 iTBS sessions (one per weekday over four weeks) while maintaining stable antidepressant treatment. The ΔΔTSH test and HAMD-17 scores were assessed pre- and post-treatment. Response was defined as a ≥ 50% reduction in HAMD-17.

Results: Baseline ΔΔTSH values were significantly lower in TRDs than in HCs ($p < 0.00001$, by U test). After iTBS treatment, patients showed a decrease in HAMD-17 scores ($p = 0.0001$, by T test) and an increase in ΔΔTSH values ($p = 0.001$, by T test)—although still remaining lower than those of HCs ($p = 0.03$, by U test). At endpoint, HAMD-17 scores correlated negatively with ΔΔTSH values (rho = -0.74, $p = 0.0004$). Fourteen patients (61%) normalized their ΔΔTSH, of whom 11 (78%) were responders; all patients who did not normalize their post-treatment ΔΔTSH (n = 9) were non-responders ($p = 0.0003$, by Fisher's Exact test).

Conclusions: While the underlying mechanisms remain to be clarified, this pilot study suggests that successful iTBS treatment may help restore normal chronobiological function of the HPT axis in TRD patients, and that such restoration is associated with clinical improvement.

Disclosure of Interest: None Declared

EPP366

Treating Depression in Dementia-patients with transcranial-pulse stimulation: an observational study

A. R. Günes[1]*, M. Rahimi[1], P. Wisniowski[1], M. Beglau[1], M. Köhne[2], C. Schmidt-Kraepelin[2] and U. Sprick[1]

[1]Center of Neurostimulation and [2]Alexius/ Josef Clinic, Neuss, Germany
*Corresponding author.
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Introduction: Depression plays a significant role in Alzheimer's-type dementia(AD) and affects approximately one in five patients[1] (Asmer MS *et al.* J Clin Psychiatry. 2018 Jul 31;79). On the other hand, depression is a risk factor for the development of dementia. Despite years of research and the discovery of numerous pathophysiological factors in their mutual influence, there is still no sufficiently reliable therapeutic approach[2] (Lyketsos CG *et al.* Alzheimers Dement.2011;7:5). As a non-invasive brain stimulation method, transcranial pulse stimulation (TPS) is a novel procedure based on shock-waves and whose effects on cognition in AD patients show promising results in current studies[3](Günes AR *et al.* Eur.J. N.2024;1468-1331), furthermore beneficial influence on affective symptoms were reported[4] (Sprick U *et al.* Brain Stimul.2025;439).

Objectives: In our observational study, we investigated the effects and safetiness of TPS on depressive symptoms in 100 patients who met the criteria for AD.

Methods: Each of the 100 patients (mean age: 73,5 years; gender ratio: 5:7 (m:f)) received a total of 6 treatments within 14 days (each session with 6000 pulses, energy level 0.25 mJ/mm² and frequency 4 Hz). On the basis of individual MRI scans neuro-navigation was used to apply pulses bilaterally in the frontal (including the dorsolateral prefrontal cortex) and parietal lobe as well as the precuneus area (Image 1). Written consent and the approval of an ethics committee were obtained for all patients. Before, during and immediately after the TPS intervention, the antidepressant medication of all patients remained unchanged. To assess the intensity of depressive symptoms the Beck Depression Inventory II (BDI-II) was used before the first and after the last treatment session.

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Results: 36 patients had a score above 5 points in BDI-II which represents the lower threshold with an average of 18,1 points within this group, which corresponds to a mild depression. The BDI-II total score in the pre-post comparison showed a reduction in the mean value from 18,1 to 5,9 points indicating a score almost below the lower threshold (Image 2). 26 patients reported temporary mild side effects such as fatigue or a local feeling of pressure in the treated areas. There were no serious adverse effects detected in any patient.

Image 1:

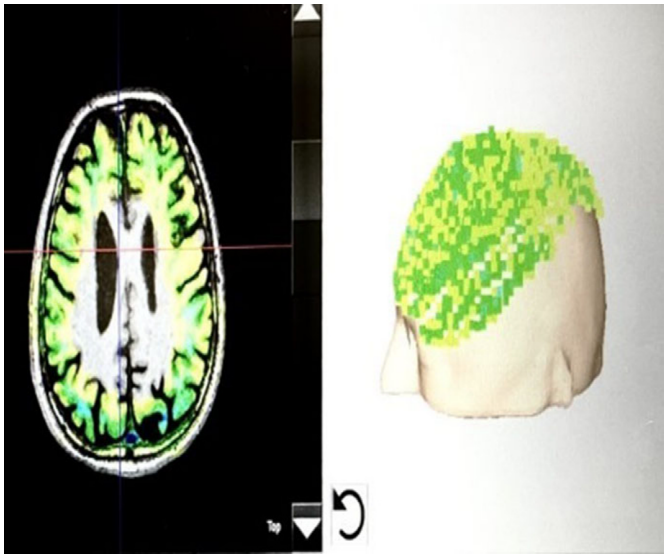
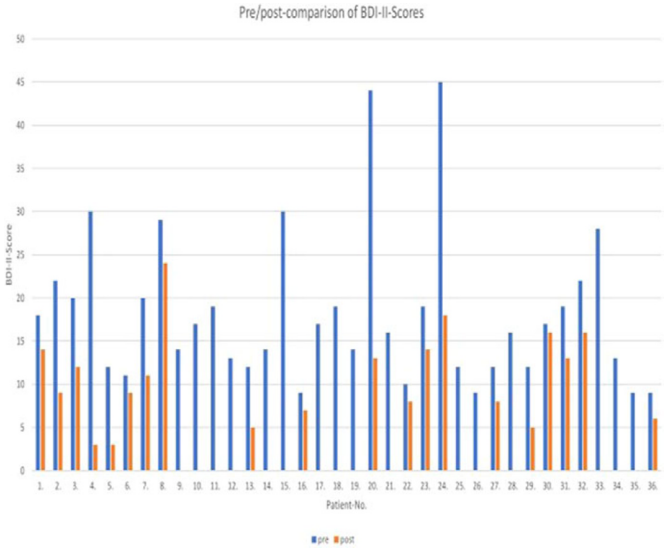


Image 2:



Conclusions: To our knowledge, this trial is the largest study to systematically investigate the effects of TPS on depressive symptoms in AD patients so far. The positive impact on depressive symptoms may be based in part on stimulation of the dorsolateral prefrontal cortex, which is a well established target region in the treatment of major depressive disorder using transcranial magnetic stimulation. TPS seems to be a safe approach to treat depressive symptoms in patients with AD. Further studies with a larger study population are necessary to confirm the safety and effectiveness of this novel method.

Disclosure of Interest: None Declared

EPP367

Exploring Serum BDNF as a Biomarker in Negative Symptoms of Schizophrenia following Neuromodulatory Intervention

R. Gupta[1]* and A. Sharma[2]

[1]Psychiatry, Institute of Mental Health, Rohtak and [2]Psychiatry, Central Institute of Psychiatry, Ranchi, India
*Corresponding author.
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Introduction: Schizophrenia with predominantly negative symptoms is highly disabling and lacks effective treatments. This gap underscores the need for biomarkers to guide novel approaches. Brain-Derived Neurotrophic Factor (BDNF), essential for neuronal survival and synaptic plasticity, is consistently reduced in schizophrenia, especially in those with prominent negative symptoms. Although its role as causative or epiphenomenal remains debated, its involvement in neurodevelopment and synaptic regulation makes it a strong candidate biomarker. Neuromodulatory interventions like rTMS and tDCS enhance neuroplasticity with emerging evidence in schizophrenia. The combination of HD-tDCS priming with iTBS offers a promising strategy targeting DLPFC hypoactivity, leveraging metaplasticity to potentiate BDNF-mediated plasticity. Investigating serum BDNF may provide insights into the biological underpinnings of neuromodulation & possibility of developing biomarker-guided precision psychiatry for schizophrenia.
Objectives: 1. To measure and compare serum BDNF levels before and after HD-tDCS (active/sham) priming followed by iTBS.
2. To examine the correlation between serum BDNF levels and clinical symptom severity, including PANSS and SANS scores.
Methods: A prospective hospital based double-blinded randomized controlled study with a sample size of 40, where the participants were divided into Active or Sham HD-tDCS primed iTBS stimulation groups for a total of 20 sessions over two weeks. Serum BDNF was quantified at baseline and after 4 weeks using Sandwich ELISA kits. Symptom severity was assessed with PANSS and SANS. Data were analyzed using paired and independent t-tests, along with correlational analyses.
Results: At baseline, serum BDNF did not differ significantly between active and sham groups. After 4 weeks, BDNF increased significantly in the active group, while a non-significant decline was observed in the sham group. Between-group comparison at 4 weeks favored the active group but did not reach statistical significance. Correlational analysis showed baseline serum BDNF negatively correlated with total PANSS scores. Post-treatment, higher serum BDNF levels correlated with lower PANSS total, PANSS negative subscale, and SANS domains including affective flattening and alogia. These findings suggest that increases in BDNF are linked to reductions in overall symptom severity.