interpersonal difficulties, and can adversely affect emotional well-being and intimate relationships.
Objectives: The aim of this review is to discuss the aetiology, diagnosis, and treatment of HSDD.
Methods: A comprehensive literature search was conducted using the electronic database PubMed. The keywords used for the search included "Hypoactive Sexual Desire Disorder", "treatment", and "aetiology and diagnosis".
Results: The search yielded a total of five systematic reviews. These studies concluded that the aetiology of HSDD involves a complex interaction of biological, psychological, and sociocultural factors. The diagnosis of this disorder should include a comprehensive sexual and medical history to rule out other causes. Treatment options for HSDD are multifaceted, incorporating both pharmacological and non-pharmacological approaches.
Conclusions: HSDD may be caused by biological factors such as a reduction in sexual excitation signals, an increase in sexual inhibition signals, or a combination of both. Testosterone plays a crucial role in initiating sexual activity, desire, and behaviour, through its influence on vaginal lubrication, sensation, and clitoral engorgement. Low oestrogen levels are associated with dyspareunia and changes in the vulvovaginal mucosa. Progesterone, serotonin, dopamine, and noradrenaline also play a role in the physiology of sexual desire. Psychological factors, particularly a lack of emotional intimacy, communication difficulties, negative body image perceptions and low self-esteem, can also reduce sexual desire. Depressive and anxiety disorders can significantly affect sexual desire. Sociocultural factors, such as religious beliefs and traditional values, can have a negative impact on sexuality. The diagnosis is made through a detailed clinical history, which may be supported by a screening tool, the Decreased Sexual Desire Screener (DSDS), as well as laboratory and imaging investigations. Identified modifiable factors, such as illicit substance abuse, sleep problems, medication use, and various medical and psychological factors, should be addressed first. For women without remaining modifiable factors who need psychological support, sex therapy, cognitive-behavioural therapy, and couples therapy are recommended. In premenopausal women, pharmacological treatment with flibanserin or bremelanotide may be considered. In postmenopausal women, hormonal therapy with testosterone may be considered off-label. The combination of psychological and pharmacological interventions is the most effective approach for HSDD. However, further studies are needed to better understand the pathological mechanisms of HSDD and to develop new therapeutic options.

Disclosure of Interest: None Declared

Addictive Disorders

EPP346

Exploring the effects of theta burst stimulation on craving severity and biomarkers in patients with gambling disorder

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Introduction: Gambling disorder, previously known as pathological gambling, is a behavior that significantly impairs functioning in personal, social, and occupational domains. Currently, there is no pharmacological treatment for gambling disorder, emphasizing the need for innovative treatment modalities. Imaging studies have identified a connection between prefrontal circuit dysfunction and behavioral disinhibition, supporting the potential use of non-invasive brain stimulation in treating gambling disorder.
Objectives: The purpose of this study is to investigate the effect of theta-burst stimulation (TBS) on gambling disorder.
Methods: The study duration is 2 weeks, with 10 sessions of TBS intervention. The intervention group will receive 1800 pulses of intermittent TBS at the left dorsolateral prefrontal cortex and 1200 pulses of continuous TBS at the pre-supplementary motor area during each session, while the control group will receive sham stimulation. Primary outcomes, including the Gambling Symptom Assessment Scale (G-SAS) and the Visual Analogue Scale (VAS) for craving, were administered at weeks 0, 2, 4, and 8, and the changes between the two groups were compared using generalized estimating equations. Secondary outcomes, including Beck Anxiety Inventory and Beck Depression Inventory, and serum brain-derived neurotrophic factor (BDNF), cortisol, and hsCRP, were measured at weeks 0, 4, and the changes between the two groups were compared using repeated measures ANOVA.
Results: A total of 33 patients with gambling disorder were randomly assigned in a 2:1 ratio to the intervention group (21 patients) and the control group (12 patients) on a double-blind basis. They were included in the preliminary analysis on an intention-to-treat basis. The VAS scores of the active group decreased more than those of the sham group (active group: 53.3 to 17.9, sham group: 37.5 to 15.3), but the difference did not reach statistical significance ($p = 0.13$). Compared to the sham group, the active group showed a decreasing trend in hsCRP ($p = 0.54$) and an increasing trend in free BDNF ($p = 0.34$), but neither reached a statistically significant difference.
Conclusions: Drawing definitive conclusions is limited by small sample size. Nevertheless, the initial results from this study suggest that the alterations in levels of gambling craving, hsCRP, and serum BDNF align with our hypothesis.

Disclosure of Interest: None Declared

EPP348

Cluster analysis of relapse risk factors in alcohol use disorder

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Introduction: The chronic relapsing nature of alcohol use disorder (AUD) makes treatment and recovery especially difficult. One of the most prevalent factors contributing to relapse is craving, which is a strong urge to drink alcohol. Moreover, alcohol withdrawal symptoms experienced during abstinence can trigger the urge to

drink alcohol. Furthermore, stress and anxiety can also elicit craving and increase the motivation to drink alcohol.
Objectives: Further understanding the risk of relapse would be crucial for the treatment of AUD. Thus, the aim of this study was to identify clusters within a sample of patients with AUD based on the different factors of relapse risk, and to compare these clusters based on the severity of alcohol use disorder, craving and anxiety.
Methods: The sample consisted of 114 patients diagnosed with AUD at the Department of Psychiatry, University of Szeged, Hungary between November 2022 and January 2024. The level of AUD was measured with the Alcohol Use Disorders Identification Test (AUDIT) (subscales: consumption, dependence, harmful consequences of alcohol use) and the Severity of Alcohol Dependence Questionnaire (SAD-Q) (subscales: physical withdrawal signs, affective withdrawal signs, withdrawal relief drinking, quantity and frequency of alcohol consumption, rapidity of reinstatement of withdrawal symptoms following abstinence). State and trait anxiety were measured with the State-Trait Anxiety Inventory (STAI-S, STAI-T). Craving was measured with the Multidimensional Alcohol Craving Scale (MACS). The risk of relapse was measured with the Alcohol Relapse Risk Scale (ARRS) (subscales: stimulus-induced vulnerability (SV), emotionality problems (EP), compulsivity for alcohol (CA), negative expectancy for alcohol (NE), positive expectancy for alcohol (PE), insight into mental condition (IM)).
Results: Two-step cluster analysis was performed with the six subscales of the ARRS as predictor variables. A two-cluster solution was found, in which SV proved to be the most important predictor. Independent sample t-tests for the two clusters revealed significant between-cluster differences on all subscales except for 'lack of negative expectancy for alcohol' ($p \geq 0.001$). Independent sample t-tests and Chi-square tests were performed to compare the two clusters on the basis of age, sex, the severity of AUD, craving and anxiety. Significant differences were found in almost all factors except for age, sex and the 'rapidity of reinstatement of withdrawal symptoms following abstinence' subscale of the SAD-Q ($p \geq 0.01$).
Conclusions: The first cluster with more defined signs for the risk of relapse was characterised by more severe AUD, craving, state and trait anxiety compared to the second cluster with milder signs for the risk of relapse. These results suggest that the risk of relapse is a complex phenomenon, which can be identified through the evaluation of several different factors, which may influence treatment choices.

Disclosure of Interest: None Declared

EPP350

Effect of CYP2C19*17 gene polymorphism on plasma levels of diazepam and nordiazepam in Turkish patients with Alcohol Withdrawal Syndrome

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Introduction: Alcohol withdrawal syndrome (AWS) is not a common medical condition in general population however it affects patients with alcohol use disorder (AUD) and causes severe complications when diagnosed late or left untreated. Diazepam is a benzodiazepine, which is used to treat various diseases such as insomnia, anxiety, muscle spasm, pain and AWS. Compared to other benzodiazepines, diazepam is more efficient to prevent delirium and decrease withdrawal due to its long half-life. Diazepam is metabolised to its main metabolite nordiazepam with the enzymes expressed by *CYP2C19* and *CYP3A4* genes. It has been reported that metabolic activity of the enzymes encoded by *CYP2C19* gene may be varied due to genetic polymorphisms leading a change in the efficiency of treatment via effecting the plasma level of drugs metabolised by *CYP2C19*.
Objectives: The aim of this study is to investigate whether *CYP2C19*17* gene polymorphism has an impact on plasma levels of diazepam and nordiazepam in the Turkish patients with AWS and under oral diazepam treatment.
Methods: The study included 50 male patients who were in withdrawal state and taking diazepam therapy. *CYP2C19*17* polymorphism was determined by PCR-RFLP method. Plasma levels of diazepam (DZP) and nordiazepam (NDZP) were detected by HPLC.
Results: Genotype frequencies were calculated as 66% for CC, 30% for CT and 4% for TT. Dose-normalized DZP and dose-normalized NDZP values were 0.049 µg/ml per mg/day and 0,056 µg/ml per mg/day, respectively. No statistical significance was observed in the levels of normalized DZP and NDZP when CC and CT+TT genotypes were compared (p= 0.073 and p=0.282, respectively).
Conclusions: The effect of CYP2C19*17 polymorphism on the plasma levels of DZP and NDZP following long term oral diazepam to treat patients with AWS was determined for the first time. With the help of current study, first data on Turkish population was obtained and may be useful for personalized therapy in the future.
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EPP351

Age-Related Cognitive Decline in Substance Use Disorder: Impact of Prolonged Substance Consumption

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Introduction: The consumption of alcohol, cannabis, cocaine, or heroin causes alterations in the central nervous system, affecting mood, perception, and behaviour. Despite the harmful effects of these substances, they remain widely used. Younger individuals tend to consume cannabis and cocaine, while older adults more commonly use alcohol and prescription medications. Ageing brings