Cognitive Bias Modification (CBM)-training to focus attention on positive information is thought to improve emotional processing and depressive symptoms. Some studies imply reduced duration and occurrence of microstate D in MDD compared to healthy controls. However, the effect of CBM on microstates is still unclear.
Objectives: (1) To replicate previous findings that duration and occurrence of microstate D is reduced in patients with MDD compared to healthy controls in an independent sample and (2) to investigate the effect of an active CBM-training versus a control-training on microstates and its association with symptom improvements.
Methods: Thirty patients receiving outpatient treatment with MDD according to DSM V (aged 18-60) will be recruited in Essen and Aachen. The control group will consist of 30 healthy age-and-sex-matched participants. Psychological testing will be administered and all participants will be randomized to either an active or a control training. During the next visit, resting state EEG and a GoNoGo Task with positive, neutral and negative pictures will be measured. The participants will take a tablet home to undergo 10 sessions of CBM within 14 days. The training will be consisted of a dot-probe-task. In the active condition the probe will be more likely to appear behind a positive versus a neutral picture, while appearing randomly in the control condition. After 14 days, a second EEG will be recorded.
Results: Differences in duration and occurrence of microstate D between patients and healthy controls will be analyzed by conducting ANCOVAs with age and sex as covariates. ANCOVAs for repeated measurements will be calculated to study effects of time (pre- vs. post-training) and group (patients vs. healthy controls in active training; patients in active vs. patients in control-training), on duration and occurrence of microstate D.
Conclusions: CBM-training is proposed to be an effective treatment option for MDD patients, reflected in a reduced topographical bias of microstate D in EEG.

Disclosure of Interest: None Declared

EPV0407

Larks under pressure: The genetic background of the morning chronotype may contribute to depression in interaction with stress

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Introduction: Depression is a highly prevalent, multifactorial, complex disorder, its etiology is assumed to involve both genetic and environmental factors. Genetic factors, including biological clock genes such as *CLOCK* and *SIRT1*, have been linked to depression, particularly its symptom related sleep disturbances. Environmental factors also play a crucial role in the background of depression, particularly in interaction with genetic factors. Known environmental stress factors include stress caused negative life events or childhood adversities.
Objectives: This study aims to delve into the chronotype-specific impacts of genes previously correlated with circadian functionality on the pathomechanism of depression in interaction with environmental stress factors.
Methods: A genome-wide association study on the 'morning chronotype' phenotype was conducted with Plink2, utilizing data from the UK Biobank discovery sample (N = 139135). Using LDPred2we derived a polygenic risk score (PRS) for the NewMood Hungarian dataset (N = 1820). We performed pathway-specific analyses including genes implicated within the genetic pathway, drawing on prior research findings. Specifically, we selected the top genes (with a false discovery rate-corrected p-value < 0.05) from the "responders vs. non-responders" analysis conducted by Jerome C. Foo et al.Transl Psychiatry 2019; 9 343). We performed a main effect analysis investigating the pathway specific PRS's effect on BSI depression scores and interaction analyses using life course (number of negative life events in the past life) and recent (number of negative life events in the past year) stress scores to investigate how the interaction term predicts depression in our target sample.
Results: Our primary analysis revealed a nominally significant protective effect (beta = -20.90938, p = 0.070218). Subsequently, in the context of our interaction analysis, we identified significant risk associations, both with lifetime stress (beta = 13.7416, p = 0.0171) and recent stress (beta = 24.6034, p = 0.0038)
Conclusions: Our study unveiled a protective role in our primary analysis, juxtaposed with risk associations in our interaction analyses. This intriguing dichotomy underscores that this genetic pathway, associated with circadian dysregulation, exerts a protective influence in association with the morning chronotype. However, it transitions into a predisposing factor for depression when influenced by environmental stress factors.
Considering these findings, our study substantiates the hypothesis that both circadian genes and chronotype contribute to the pathogenesis and clinical manifestation of depression. Additionally, it underscores the pivotal role of stress as a contributing factor in the intricate pathogenesis of depression.

Disclosure of Interest: None Declared

EPV0408

Depression: Biological Non-Pharmacological Interventions. A Review.

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Introduction: Major depressive disorder stands as one of the most significant mental health issues in the general population. It impacts the patients' quality of life and increases both morbidity and mortality. Response and tolerability to available