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component was compared between the population aged over and under 65 years, the population aged \geq 65 years scored higher in most components. In contrast, daytime dysfunction scored higher in the population aged < 65 years.

Conclusions: Sleep quality tends to decrease with increasing age. Several factors, including physiological changes, underlying physical conditions, and psychosocial factors, may contribute to a decrease in sleep quality with age.

Disclosure of Interest: None Declared

EPP260

Sleep Deprivation's Effect on Circadian Genes Expression and Its Associtations with Depression Symtoms and Psychomotor Abilities

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Introduction: Sleep deprivation (SD) is a global health concern that impairs cognitive and psychomotor functions (PF). While it can temporarily improve mood, its effects on mood and connection to depressive symptoms (DS) remain unclear. These impacts may involve circadian rhythm gene regulation, though distinct evidence from human studies is lacking.

Objectives: To assess the impact of SD on the expression of circadian rhythm regulation genes and its associations with the alleviation of DS. Additionally, to explore the relationship between changes in gene expression and PF following SD.

Methods: Participants (n = 72) underwent a baseline sleep assesment by polysomnography (PSG), later being subjected to SD. In total, evaluation of mood And cognitive functions (Bimanual Eye-Hand Coordination Test - BEHCT) was conducted four times, pre/post PSG and SD. Moreover, circadian rhythm regulation genes expression: Circadian Locomotor Output Cycles Kaput (CLOCK), Brain and muscle Arnt-like protein-1 (BMAL1), Period Circadian Regulator 1 (PER1), Cryptochrome Circadian Regulator 1 (CRY1), Nuclear Receptor Subfamily 1 Group D Member 1 (NR1D1) and Neuronal PAS Domain Protein 2 (NPAS2) was evaluated. Participants were divided into respondents (RE, n = 49) and non-respondents (NR, n = 23) depending on changes in DS under the influence of SD by the Montgomery–Åsberg Depression Rating Scale evalluation.

Results: No relationship was found between BEHCT parameters and the studied genes in the entire study group. NRs exhibited a negative correlation in number of motor function errors in relation to all examined genes of CLOCK (r=-0.52, p=0.02), BMAL1 (r=-0.55, p=0.007), CRY1(r=-0.45, p=0.048), PER1(r=-0.6, p=0.023), and NR1D1 (r=-0.19, p=0.523) is except for NPAS2. Additionally, In NRs, the BEHCT error time negatively correlated with the PER1 and NR1D1 (r=-0.6, p=0.006; r=-0.52, p=0.045; respectively). In contrast, within the RE group, only NPAS2 expression showed a positive correlation with the number of errors (r= 0.35, p=0.049).

Conclusions: Reduced expression of CLOCK, BMAL1, CRY1, PER1, and NR1D1 is associated with impaired PF, only in individuals with worsening DS after SD. Increased NPAS2 expression appears to be an orgin of reduced PF results in the RE group. These genes, integral to the circadian system's feedback loop, may mediate the complex effects of SD on mood and cognitive function, warranting further investigation.

Disclosure of Interest: None Declared

Women, Gender and Mental Health

EPP261

Perinatal depression and psychiatric comorbidities during the life course: a Swedish nationwide register based study

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Introduction: Women with a history of major depression are at risk of perinatal depression (PND). The associations between PND and other types of psychiatric disorders are less clear, although a recent GWAS revealed genetic correlations with almost all psychiatric disorders.

Objectives: The aim of this study was to examine the association between PND and overall, and 17 type-specific, psychiatric disorders in a life course approach.

Methods: Leveraging Swedish nationwide health register and primary care data, we included all birthing women diagnosed with depression or prescribed for antidepressants during pregnancy or within a year postpartum, i.e., women with PND (n=122,720), during 2001-2022. Using incidence density sampling, we matched each case to 10 unaffected birthing women. We ascertained any diagnosis of psychiatric disorder over the lifetime from the National Patient Register. Using multivariable conditional logistic regressions, we estimated the association between PND and any, or subtypes of, psychiatric disorders dated before or after the PND diagnosis in a liftecourse approach. Moreover, we conducted a nested case control study to investigate the association of psychiatric disorders and subsequent PND, and a matched cohort study to investigate PND and subsequent psychiatric disorders.

Results: In preliminary results, at a mean age of 31.0, we found that PND was highly associated with any other psychiatric disorders (adjusted odds ratio (aOR)=9.02, 95%CI 8.9-9.2). The association remained when excluding depression (aOR=6.2, 95%CI 6.1-6.3), and was comparable for psychiatric disorders dated before PND diagnosis (aOR = 9.0), whereas attenuated for diagnoses dated after PND (OR= 3.9). Most pronounced association was noted for bipolar, personality disorders, depression, and anxiety. The association was stronger in primiparous women and in women born ourside of Europe (p-for interaction <0.001).

Conclusions: Throughout life course, PND is associated with psychiatric disorders, particularly with bipolar disorder, personality disorder, depression and anxiety. These findings may shed light on shared genetic/risk factors between PND and other psychiatric disorders.

Disclosure of Interest: None Declared