ICSD3 criteria. Statistical analysis: general descriptive statistics, ANOVA, Wilcoxon signed-rank test and Mann-Whitney were performed using SigmaStat.
Results:A total of 44/34% BeCOVID/POSTCOVID women and 56/66% men. Chronic disease 46/88% (hypertension 64/70%; mellitus diabetes 24/22%, dyslipidemia 32/52%; heart disease 10/18%, glaucoma 18/8%), psychiatric disease previous 10/8%. Regarding sleep disorders, a significant increase of chronic insomnia 16/46% (P = <0,001). No significant difference in other sleep disorders, obstructive sleep apnea 88/92%, rest leg syndrome 20/24%, periodic leg movement (PLM) disorders 48/32%, REM sleep behaviors disorders 8/8%, Circadian rhythms disorders 2/4%. When comparing polysomnography significant difference were observed in changes of phases number (P=0.004), no significant changes in others sleep architecture parameters such as sleep latency, REM sleep latency, efficiency, total sleep time, proportion of sleep stages (N1, N2, N3 y REM), wake after sleep onset, arousals index,PLM index or apnea-hypopnea index (AHI)were observed. Moderate or severe clinical insomnia in 42/54%. Significant increase of anxiety state (P = 0,008) and trait (P = <0,001) and sleepiness (P = 0,008). No significant difference in depression (P = 0,125).
Conclusion: The COVID-19 pandemic has brought us relevant changes in sleep disorders in older people, as a rise in chronic insomnia and generalized anxiety disorder.

CHRONOTYPE ADVANCE AS A PREDICTOR OF SUBSEQUENT COGNITIVE FUNCTION AND BRAIN VOLUME REGRESSION: A 4-YEAR LONGITUDINAL COHORT STUDY

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Introduction: Maintaining adequate sleep quantity and quality is vital for many aspects of overall health and neurocognitive function in the elderly. Accumulating evidence suggests that there exists a bidirectional link between circadian rhythm deterioration and neurodegeneration. We aimed to investigate the impact of circadian rhythm disruption on structural and functional brain deteriorations in a late adulthood population.
Materials and Methods: We analyzed data from 1874 participants (aged 49–80 years, women 50.3%) in the Korean Genome and Epidemiology Study. Midsleep time on free days corrected for sleep debt on workdays (MSFsc) at baseline was adopted as a marker of chronotype in late adulthood and used to categorize the participants into three groups. "Advancers" were defined as those falling in the bottom third of MSFsc (earlier than 2:15 am), while "delayers" were defined as those falling in the top third (later than 3:30 am). The relationships of chronotype with longitudinal change in cognitive function and gray matter volume (GMV) over four years were investigated.
Results: The average MSFsc at baseline was 2:45 am, and the earlier chronotype was linearly associated with lower right entorhinal GMV ($p = 0.001$) in addition to the poor visual memory function test scores ($p < 0.001$). Compared to baseline, follow-up GMVs were all reduced with advancing age regardless of brain lobes. In longitudinal analysis, the earlier chronotype was significantly associated with more faster atrophy in the temporal lobe ($p = 0.018$). Advancers exhibited significantly decreased scores in the digit-symbol coding test and categorical verbal fluency test. Compared to the practice effect in the delayer group, there was no definite change in verbal memory test score in the advancer group.
Conclusion: Given the long preclinical period, it is imperative to find a clinically useful indicator for screening high-risk groups in the preclinical stages of neurodegenerative disorders. We suggest that chronotype in midlife measured using a questionnaire can be a practical and valuable indicator for selecting a target group that should be closely monitored for further neurodegenerative disorder development.
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EFFECTS OF INTERACTION BETWEEN SEX AND APOE GENOTYPE ON SLEEP-STAGE SPECIFIC EXPRESSION OF OBSTRUCTIVE SLEEP APNEA AND SLEEP-DEPENDENT MEMORY

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Introduction: Alzheimer's disease (AD) is a world-wide healthcare crisis among older adults resulting in cognitive decline and dementia. It is therefore imperative to understand how AD risk factors interact to impact cognition in healthy older adults at risk for AD. Sex and apolipoprotein E (APOE) genotype are among the most impactful risk factors for developing late onset AD. Sleep disturbance, such as the presence of obstructive sleep apnea (OSA) is also impacted by sex and APOE genotype, and is a risk factor for developing AD. However, it remains unclear how these risk factors interact and how they may disrupt episodic memory, a cognitive ability particularly sensitive to AD. Here, we tested the hypothesis that sex and APOE genotype (ε4 carrier/non-carrier) would interact to impact the expression of OSA events across non-rapid eye movement (NREM) and REM sleep stages and sleep-dependent consolidation of episodic memory.
Materials and Methods: Fifty-eight cognitively unimpaired, older adults (mean±SD; 61.4±6.3 years, 38 female, 15 *APOE* ε4 carriers) underwent in-laboratory polysomnography during their habitual time. Participants encoded 88 word-pairs in the evening prior to sleep. Encoding was followed by an immediate recall test, and a delayed recall test occurred in the morning following sleep. Memory consolidation was computed by comparing performance accuracy across testing sessions (post-sleep − pre-sleep memory accuracy). Sleep architecture (TST: total sleep time, time and percent spent in each sleep stage N1, N2, N3 and REM sleep) and OSA severity (apnea-hypopnea index, AHI, measured during REM and NREM sleep stages) were quantified. Univariate ANOVA and post hoc testing was implemented to examine interacting effects of sex and *APOE* genotype on OSA expression and sleep-dependent memory.
Results: A significant sex×*APOE* genotype interaction predicted sleep-dependent memory consolidation (p=0.04). Post hoc analysis revealed a trend among *APOE* ε4 carriers, such that sleep-dependent memory consolidation was reduced in men as compared to women (p=0.06). Associations between OSA severity and sleep-dependent memory consolidation also differed by sex and *APOE* genotype. Specifically, among *APOE* ε4 non-carriers, AHI during REM sleep was negatively associated with sleep-dependent memory (r=-0.44, p=0.02) in women but not men (r=0.25, p=0.39). Further, these two correlation coefficients were significantly different (z-score= -1.98, p=.05). Among *APOE* ε4 carriers, REM sleep duration was negatively associated with sleep-dependent memory in men (r=-0.89, p=0.04) but not women (r=-0.51, p=0.16). However, these two correlations coefficients were not significantly different (z-score=-1.05, p=0.29)