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THE ROLE OF VITAMIN B12 SUPPLEMENTATION IN THE ASSOCIATION BETWEEN DEPRESSION SYMPTOMS AND DAYTIME SLEEPINESS

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Introduction: A substantial body of literature has demonstrated an association between depression and sleep disturbances. While depressive symptoms are strongly associated with limited sleep duration and quality, high depressive symptomology has been also linked to symptoms of hypersomnolence. Vitamin B12 supplementation is associated with reduced risk of depression and the enhancement of pharmacological treatment for depression, but less is known about the role of vitamin B12 on sleep disturbances associated with depressive symptoms. Thus, our current study examined vitamin B12 intake as a moderator of depression and daytime sleepiness in a national sample of adults.
Methods: The sample consisted of 5,553 adults who completed the 2017-18 National Health and Nutrition Examination Survey (M age=49.8, SD=18.6; 51.7% female). Participants reported on prescription and nonprescription dietary supplement use in the last 30 days, sleep habits and disorders adapted from the Munich Chronotype Questionnaire, and completed the Patient Health Questionnaire-9 for depression screening. All measures were administered by trained interviewers. A moderation analysis was performed using R statistical programming. The analysis controlled for age, gender, ethnicity, sleep duration, and other B-complex vitamins (e.g., B1, B2, B3, and B6).
Results: Depressive symptoms were significantly associated with greater daytime sleepiness (b=.06, p <.001). Furthermore, there was a significant interaction between depressive symptoms and vitamin B12 consumption (b=-.002, p=.003, R2=.12). Higher vitamin B12 consumption buffered the relationship between depressive symptoms and daytime sleepiness. In contrast, a stronger positive relationship between depressive symptoms and daytime sleepiness was observed among participants with lower Vitamin B12 consumption. Thus, the findings suggest that consuming vitamin B12 may be beneficial for counteracting daytime sleepiness associated with depression.
Conclusion: Findings from the current study suggest that vitamin B12 supplementation provides a small, but significant buffering effect on the relationship between depressive symptoms and daytime sleepiness. Although existing pharmacological and behavioral interventions for sleep and depression are clinically effective, vitamin B12 intake may be an additional modifiable behavior that could increase prognosis of treatment. Given the very modest interaction effect, further
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OBESITY-INDUCED BREATHING VARIABILITY DURING SLEEP IS NOT ENTIRELY ATTRIBUTED TO APNEAS AND SLEEP FRAGMENTATION

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0**Introduction:** Obesity is a major cause of sleep-disordered breathing (SDB). Conventional metrics of SDB can be confounded by the effects of obesity on oxygenation and lack of standard definitions. Sleep fragmentation is frequently observed in obese individuals, but whether it occurs independently of SDB remains unknown. Quantitative analysis of ventilation may delineate the effects of obesity on breathing patterns and sleep fragmentation. We aimed to examine the effects of obesity on respiratory patterns during sleep and the relationship between obesity-related respiratory variability and sleep fragmentation.
Methods: Sleep recordings were performed in 15 lean C57BL/6J and 17 diet-induced obese (DIO) mice on the same genetic background. We applied Poincaré analysis of minute ventilation (VE) during sleep to estimate the breathing variability. Arousals were classified as respiratory when associated with ≥30% drops in VE from baseline.
Results: Breathing variability was significantly higher in DIO mice during NREM sleep, but not during REM sleep. Obesity-induced breathing variability could not be entirely attributed to apneas or arousals. Sleep fragmentation was 45% greater in DIO mice. Respiratory arousals comprised 15% of the arousals in both strains. Breathing variability was inversely associated with sleep fragmentation regardless of obesity.
Conclusion: Obesity increased respiratory variability during NREM sleep, which was not fully attributed to apneas and macrosleep architecture. Obesity caused sleep fragmentation that was not entirely explained by SDB severity. Our quantitative analysis of VE identified differences in breathing variability in obesity that were not captured by traditional SDB metrics, which may be applicable for human SDB.
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