ORIGINAL ARTICLE

The Efficacy of Daytime Polysomnography in Diagnosing Obstructive Sleep Apnea (OSA) at University Malaya Medical Centre (UMMC) – A Pilot Study

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ABSTRACT

Introduction: Obstructive Sleep Apnea (OSA) is a common sleep disease characterized by persistent upper airway blockages during sleep, resulting in interrupted sleep and oxygen desaturation. The gold standard for diagnosing OSA is Nighttime Polysomnography (NPSG), leading to prolonged waiting time due to high demand and limited overnight slots. Daytime polysomnography (DPSG) offers a more flexible and accessible alternative. Limited information exists on the application of DPSG in the diagnosis of OSA in Malaysia. Therefore, this pilot study aimed to assess the efficacy of DPSG in diagnosing OSA at the University Malaya Medical Centre (UMMC), Malaysia. **Methods:** Sixty patients, suspected of severe OSA, were diagnosed using polysomnography (PSG) at the Sleep Clinic of UMMC. The PSG reading was recorded either during daytime (n=30) or nighttime (n=30). Diagnostic parameters included the apnea-hypopnea-index (AHI), sleep architecture, and the Epworth Sleepiness Scale (ESS), which were compared between DPSG and NPSG. Results were analyzed using independent t-test, and Pearson correlation coefficient statistical tests. **Results:** NPSG exhibited significant sleep efficiency relative to DPSG ($p<0.05$); nevertheless, AHI and ESS scores revealed no significant differences between the two groups. Correlational study also revealed no significant link in AHI and ESS scores between DPSG and NPSG patients ($p>0.05$). **Conclusion:** The results indicate that there is no difference between using DPSG and NPSG in diagnosing OSA. This suggests that DPSG is an effective alternative to NPSG for diagnosing OSA.
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Keywords: apnea hypopnea index, sleep apnea syndrome, sleep disorders screening, daytime polysomnography, nighttime polysomnography

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INTRODUCTION

There are over 100 million individuals worldwide who suffer from sleep apnea, with obstructive sleep apnea (OSA) accounting for over 90% of all cases (1). OSA is caused by partial or total obstruction of the upper airway during sleep. Individuals who have OSA will experience a range of negative effects such as excessive daytime sleepiness, fatigue, non-restorative sleep, morning headaches, irritability, and memory loss. Untreated OSA can also lead to decreased productivity, occupational and motor vehicle accidents that lead to injuries, or worse, fatalities (2). If left untreated for long a period of time, OSA can cause depression, metabolic disorders, cardiovascular disease, and cognitive impairments (3).

In the United States, the prevalence of OSA is 33.9% among men and 17.4% among women, based on the criteria of an apnea–hypopnea index (AHI) greater than 5. When a level of AHI more than 15 is applied, the prevalence decreases to 13% in men and 6% in women, respectively (4). In Malaysia, the prevalence of high-risk OSA was found to be 54.4% based on questionnaire responses from 410 adults attending two primary care clinics in Miri, Sarawak. Significant factors associated with probable OSA included smoking, retirement status, and Chinese ethnicity (5). A following survey in Kuantan, Pahang, revealed a significant prevalence (32%) of individuals suffering with OSA, predominantly among men (54%), Malays (86.7%), and married individuals (79.4%) (6). The most common tool to

diagnose for OSA is by using NPSG, which is used for evaluating and monitoring physiological changes such as breathing and sleep level while the patient is asleep (7). PSG tests are usually conducted in a sleep center or laboratory. However, access to PSG is commonly used during nighttime, which leads to extensive waiting time (7). The lengthy waiting time is also caused by the increased demand because PSG is also utilized to diagnose other sleep disorders such as insomnia and night terror. At UMMC, the patients need to wait one year to be diagnosed using NPSG. Without any access to PSG, patients suspected of OSA could not be accurately diagnosed thereby preventing them from getting the right treatment. Therefore, to overcome this problem, DPSG can be offered as an alternative to diagnose for OSA. DPSG can be used to assess irregularities or problems related to sleep that might arise during irregular sleep patterns, such as in people with excessive daytime sleepiness (6). However, there is currently no information available on the use of DPSG in Malaysia. In Saudi Arabia, DPSG is known as an effective tool for diagnosing sleep disorders in patients experiencing excessive daytime sleepiness (EDS). It may help reduce the waiting time for patients requiring NPSG (8). Therefore, this study aimed to compare the efficacy of DPSG with the gold standard NPSG in diagnosing OSA at UMMC's sleep clinic.

METHODOLOGY

Research Design

This pilot study employed a cross-sectional design to evaluate the efficacy of DPSG in diagnosing OSA at UMMC. Two independent groups of patients were assessed: one group underwent DPSG, while a separate group underwent NPSG, which serves as the gold standard, through random selection. Both groups were studied in a controlled clinical environment, allowing for comparison of diagnostic outcomes between the two methods.

Study Population and Sample

The study population consisted of individuals who were referred to UMMC's sleep clinic for an examination of probable OSA. The inclusion criteria included adults aged 18-65, who exhibit symptoms suggestive of OSA, such as habitual snoring, witnessed apneas, or excessive daytime sleepiness. The exclusion criteria for this study encompass chronic renal illness, heart failure, resistant arterial hypertension, stroke, tonsillitis, and neurological problems. A total of 30 patients underwent a comprehensive DPSG recording and another 30 patients for NPSG. The main assessed variables consist of AHI for measuring the severity of OSA, sleep architecture for evaluating the distribution of various sleep stages, and the ESS values for assessing daytime sleepiness.

Ethical Approval

This pilot study received ethical approval from the Medical Research Ethics Committee, UMMC (MREC ID NO: 2023121-13071). All participants provided informed consent prior to inclusion, ensuring adherence to ethical guidelines and the protection of patients' rights and confidentiality. The study was conducted in accordance with the Declaration of Helsinki and relevant local regulations.

Polysomnography (PSG)

This study conducted comprehensive sleep study during the daytime or nighttime, using the standard polysomnographic equipment to monitor various physiological parameters. These include electroencephalogram (EEG) to measure brain activity, electrooculogram (EOG) to monitor eye movements, electromyogram (EMG) for muscle activity, electrocardiogram (ECG) for heart activity, respiratory effort, airflow, oxygen saturation, and body position. The patients suspected of suffering from OSA underwent a polysomnographic study conducted in a controlled environment, for approximately 6 hours. Sleep stages and respiratory event scoring were determined manually by qualified neurophysiology technologist based on the guidelines produced by the American Academy of Sleep Medicine (AASM).

Apnea-Hypopnea Index (AHI)

AHI is a standard metric used to quantify and assess the frequency of obstructive events during sleeping and identify the severity of OSA. These obstructive events include apnea, defined as a complete cessation of airflow lasting at least 10 seconds despite ongoing respiratory effort, and hypopnea, characterized by a partial reduction in airflow (typically ≥30%) for at least 10 seconds, accompanied by oxygen desaturation (≥3–4%) or arousal from sleep. Based on the AHI value, the level of OSA were classified as follows: i) AHI 5 per hour is normal; ii) AHI ≥ 5 and < 15 per hour are considered mild; iii) AHI ≥ 15 and < 30 are moderate, and AHI ≥30 is classified as severe OSA (9).

Epworth Sleepiness Scale (ESS)

In this study, ESS was measured using a self-administered questionnaire. The scale consisted of eight situations, and the respondent rates their likelihood of dozing off or falling asleep in each scenario. The total score ranged from 0 to 24, with higher scores indicating greater daytime sleepiness. The sum of the person's answers in each of the eight scenarios was used to get their overall ESS score. The score was then interpreted as follows: i) 0-9 = normal daytime sleepiness, ii) 10-15 = mild excessive daytime sleepiness, and iii) 16-24 = moderate to severe excessive daytime sleepiness (11).

Statistical Analysis

Statistical data analysis was performed using Graph Pad PRISM software version 10. Results were shown as mean ± SD. Comparisons between both groups of patients were performed using independent t-tests and Mann–

Whitney U-tests for normally and abnormally distributed data, respectively. Correlations were performed using Pearson correlation coefficient. Statistical significance was defined using a p-value less than or equal to 0.05 ($p \leq 0.05$).

RESULT

Patients Characteristics

A total number of 60 patients were recruited randomly for this study: DPSG (n=30) and NPSG (n=30). The majority of patients were males in daytime (70%) and nighttime (73%) (Table I). Analysis of racial profiling shows that the majority of patients were Malay (daytime: 57%; nighttime 63%), followed by Chinese (daytime: 27%; nighttime: 23%) and Indian (daytime: 16% nighttime: 14%). Many of the patients were young adults (daytime: 50%; nighttime: 40%) followed closely with the middle-aged adults (daytime: 27%; nighttime: 33%) and older adults (daytime: 23%; nighttime: 27%). Patients were also categorized according to their Body Mass Index (BMI). Result shows that 40% of DPSG patients and 53% of NPSG were obese. Statistical analysis indicated that there are no significant differences observed in age, BMI, or age between DPSG versus NPSG patients.

The final parameter is ESS. The mean values for ESS amongst patients are 12.10 (for DPSG) and 8.40 (for NPSG), suggesting mild excessive daytime sleepiness for DPSG (11). In contrast, the average score of 8.40 indicates that these NPSG patients have higher sleepiness on a daytime compared to healthy individuals. Although there is a variation in the ESS level, the difference is not statistically significant ($p > 0.05$).

Table I: Demographic and Clinical Characteristics of Participants in Polysomnography (PSG) Studies

Variable	Daytime PSG		Nighttime PSG		p-value
	Frequency (n)	Percentage (%)	Frequency (n)	Percentage (%)	
Gender					>0.05
Male	21	70	22	73	
Female	9	30	8	27	
Race					>0.05
Malay	17	57	19	63	
Chinese	8	27	7	23	
Indian	5	16	4	14	
BMI					>0.05
Normal	10	33	8	27	
Overweight	8	27	6	20	
Obese	12	40	16	53	
Age					>0.05
19-39 years (young adult)	15	50	12	40	
40-59 years (middle adult)	8	27	10	33	
60-89 years (older adult)	7	23	8	27	
ESS Mean Score	12.10 ± 3.87		8.40 ± 4.83		>0.05

PSG: polysomnography; BMI: body mass index; ESS: Epworth Sleepiness Scale

Polysomnography (PSG)

Polysomnography recorded brain activity, eye movements, oxygen saturation and heart rate to evaluate sleep pattern and identify obstructive sleep events (5). Results show that there is no significant difference between DPSG with NPSG following assessment of sleep stage from Non-Rapid Eye Movement (NREM) 1- 4 and Rapid Eye Movement (REM) (Table II). As for REM latency, the normal duration is between 70 to 110 minutes, which does not differ much from the readings attained from DPSG and NPSG ($p > 0.05$). There is also no significant difference between DPSG and NPSG for REM latency ($p > 0.05$). Other parameters such as heart rates, and lowest oxygen saturation level did not yield any significant difference between daytime versus nighttime PSG as well ($p > 0.05$). Interestingly, sleep efficiency was significantly higher during NPSG compared to DPSG ($p < 0.05$).

Table II: Comparison of Sleep Parameters between Daytime and Nighttime Polysomnography (PSG)

Variable	Daytime PSG		Nighttime PSG		p-value
	Mean	Std Dev	Mean	Std Dev	
Stage 1	27.40	21.34	19.50	15.03	>0.05
Stage 2	45.53	16.62	46.90	15.85	>0.05
Stage 3	6.27	6.28	6.25	6.01	>0.05
Stage 4	7.12	12.02	12.5	15.62	>0.05
REM	14.14	11.07	14.32	7.55	>0.05
REM latency	84.43	92.88	118.7	68.45	>0.05
Wake during sleep	57.08	54.53	36.60	43.27	>0.05
Sleep Efficiency	72.78	23.20	81.14	12.50	<0.05
AHI	41.64	30.70	47.91	28.80	>0.05
Highest heart rate	87.03	17.67	88.27	13.27	>0.05
Lowest heart rate	52.30	9.86	54.50	8.13	>0.05
Lowest SpO2	82.73	9.91	80.87	8.13	>0.05

AHI: Apnea-Hypopnea Index; REM: Rapid Eye Movement; SpO2: Oxygen Saturation Level

Analysis of the different severity levels of AHI and ESS for DPSG versus NPSG

To fully clarify the impact of daytime recording of sleep study versus nighttime, the effect of DPSG and NPSG on different AHI and ESS severity levels was analyzed. Result shows that the average value for the severe group (>30) in the AHI group for DPSG was 42.60±30.80 versus 48.18±29.27 for NPSG (Fig. 1). For the moderate group, the average value was 39.92±29.79 (DPSG) versus 46.74±32.82 (NPSG). Finally, the mild group shows an average reading of 38.81±33.97 (DPSG) and 48.18±29.27 (NPSG). None of the subjects in any category were classified as normal.

Meanwhile, in the assessment of ESS, the average value for severe group were 12.07±3.99 (DPSG) versus 10.40±5.19 (NPSG) (Fig. 2). In the moderate group, there were an average of 11.80±3.92 (DPSG) and 10.20±5.16 (NPSG). In the mild group, the average reading was 11.82±3.82 (DPSG) versus 3.79±4.57 (NPSG). Finally, in the normal group, there was an average number of

A)

	Daytime PSG			Nighttime PSG			
Characteristics	N	Mean	Std Dev	N	Mean	Std Dev	p-value
AHI							
Normal	0	0	0	0	0	0	>0.05
Mild	6	38.81	33.97	4	48.18	29.27	>0.05
Moderate	10	39.92	29.79	5	46.74	32.82	>0.05
Severe	14	42.60	30.79	21	49.70	28.96	>0.05

B)

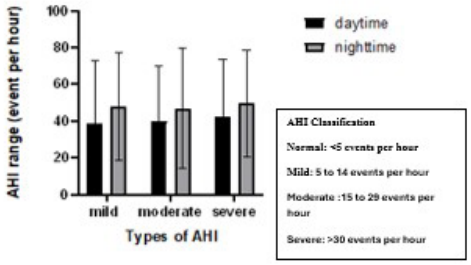

Fig. 1: Comparison of Apnea Hypopnea Index (AHI) between Daytime and Nighttime Polysomnography (PSG). (A) A table summarizing the mean of AHI values for different severity classifications (mild, moderate, and severe) based on daytime and nighttime PSG. No statistically significant differences (p>0.05) were observed between daytime and nighttime AHI values across all severity categories. (B) A bar graph illustrates the distribution of AHI values for mild, moderate, and severe sleep apnea during daytime (black bars) and nighttime (gray bars). Error bars represent standard deviations. The classification criteria for AHI severity are indicated in the inset box: normal (<5 events per hour), mild (5–14 events per hour), moderate (15–29 events per hour), and severe (≥30 events per hour).

A)

	Daytime PSG			Nighttime PSG			
Characteristics	N	Mean	Std Dev	N	Mean	Std Dev	p-value
ESS							
Normal	8	11.70	4.13	19	8.40	4.83	>0.05
Mild	8	11.82	3.82	3	8.79	4.57	>0.05
Moderate	9	11.80	3.92	4	10.20	5.16	>0.05
Severe	5	12.07	3.99	4	10.40	5.19	>0.05

B)

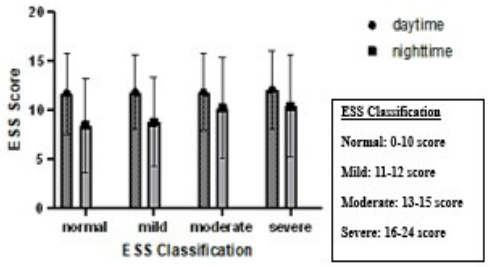

Fig. 2: Comparison of Epworth Sleepiness Scale (ESS) Scores between Daytime and Nighttime Polysomnography (PSG). (A) A table summarizing the mean ESS scores for different severity classifications (normal, mild, moderate, and severe) based on daytime and nighttime PSG. No statistically significant differences (p>0.05) were observed between daytime and nighttime ESS scores across all severity categories. (B) A bar graph illustrates the distribution of ESS scores for different severity levels of sleep apnea during daytime (black bars) and nighttime (gray bars). Error bars represent standard deviations. The classification criteria for ESS severity are indicated in the inset box: normal (0–10 score), mild (11–12 score), moderate (13–15 score), and severe (16–24 score).

11.70±4.13 DPSG patients in contrast with 8.4±4.83 for NPSG. Despite these differences, they are not statistically significant (p>0.05).

Correlation analysis of AHI and ESS between daytime PSG and nighttime PSG

Correlation analysis of AHI between DPSG and NPSG shows that there is no significant correlation for AHI values between DPSG and NPSG patients (p>0.05) (Table III). Furthermore, only about 0.012% of the variability in DPSG outcomes can be explained by nighttime AHI values (R = 0.0001172). These findings confirm that there is no correlation for AHI values between DPSG with NPSG.

Meanwhile, correlation analysis of ESS between DPSG and NPSG also showed there was no significant correlation (p>0.05) (Table IV). The Pearson correlation coefficient indicates a non-significant negative linear relationship, meaning that changes in nighttime ESS

scores are not reliably associated with changes in daytime ESS scores. Furthermore, only 3.15% of the variability in daytime ESS scores could be explained by nighttime ESS values (R = 0.03153), suggesting that other factors are likely more influential in determining daytime sleepiness.

Table III: Correlation Between Daytime and Nighttime Apnea-Hypopnea Index (AHI) in Polysomnography (PSG).

AHI category	95% confidence interval	r	R^2	p-value
All patients	-0.3697 to 0.3508	-0.01082	0.0001172	0.9547

Table IV: Correlation Between Daytime and Nighttime Epworth Sleepiness Scale (ESS) in Polysomnography (PSG).

ESS category	95% confidence interval	r	R^2	p-value
All patient	-0.5055 to 0.1952	-0.1776	0.03153	0.3479

DISCUSSION

Patients Characteristics

The mean ages of patients undergoing DPSG or NPSG are very similar, with no significant difference ($p>0.05$). This study found that age is not a confounding factor and the slight age variation between groups is unlikely to affect the comparative analysis of PSG results. The ESS score, which measures daytime sleepiness, is higher in the DPSG group compared to the NPSG group, indicating that patients undergoing DPSG report higher levels of sleepiness. The specific molecular mechanisms connecting nocturnal oxygen desaturation with EDS remain unidentified. A prominent hypothesis proposes that intermittent hypoxemia during sleep causes neural cell injury and apoptosis through the convergence of inflammatory and oxidative stress pathways. This damage is believed to specifically affect wake-promoting regions in the central nervous system, resulting in the development of sleepiness (9).

Although the difference is not statistically significant ($p>0.05$), it shows a trend towards higher daytime sleepiness in the DPSG group. This could be due to the circadian influence, where patients are naturally more alert at night, affecting their daytime sleepiness scores. The BMI is higher in the NPSG group, but the difference is not statistically significant ($p>0.05$). This suggests that both groups have comparable BMI, although a slightly higher BMI in the nighttime group might indicate a trend that could be explored further. BMI is an important factor in OSA, as higher BMI is associated with increased risk and severity of the condition (10).

Apnea Hypopnea Index (AHI)

There is no statistically significant difference between DPSG and NPSG ($p > 0.05$) for all AHI levels. This suggests that the severity of OSA, as indicated by AHI, remains consistent regardless of the time of sleep study, indicating the reliability of both daytime and nighttime PSG in assessing OSA severity. The similarity in AHI values between DPSG and NPSG implies that the severity of OSA remains relatively stable regardless of when the sleep study is conducted. The stability in OSA severity observed between DPSG and NPSG suggests that factors such as circadian rhythms or variations in sleep architecture may not significantly influence the frequency of apneas and hypopneas, as also noted by Suhaila Al-Jawder and Bahammam (2009) in their evaluation of DPSG (8).

Epworth Sleepiness Scale (ESS)

The comparison of ESS between DPSG and NPSG shows no statistically significant differences across all severity categories (normal, mild, moderate, and severe). This suggests that the timing of the PSG study does not significantly influence the level of daytime sleepiness, as measured by ESS. The ESS is a widely used tool for assessing daytime sleepiness and is considered valid and reliable. The consistency in ESS scores between DPSG and NPSG indicates that the ESS is robust and provides consistent results regardless of the timing of the sleep study. The consistent ESS scores between DPSG and NPSG suggest that treatment decisions based on ESS assessments can be made without concern for differences in the timing of the sleep study. Although the ESS is a simple and reliable method for measuring persistent daytime sleepiness in adults, daytime studies on patients with relatively low ESS scores (12.1 ± 3.87) were successfully conducted. The findings are comparable to the study by by Suhaila Al-Jawder and Bahammam in 2009 (8). Despite their study involving inpatients and sleep-deprived individuals, similar ESS scores were recorded.

Correlation of Apnea Hypopnea Index (AHI) with Epworth Sleepiness Scale (ESS)

The correlation finding of AHI during DPSG and NPSG suggests that there is no significant correlation between the severities of OSA measured at different times of the day, as indicated by the high p-value and the confidence interval below zero. Therefore, there is an unlikely meaningful relationship between AHI values obtained during DPSG and NPSG. Contrary to earlier findings by Persson and Svanborg (1996) (12), who reported a significant correlation between DPSG and NPSG results and concluded that sleep deprivation worsens OSA, our study found no meaningful linear relationship between AHI values measured at different times of the day. The absence of significant correlation in our findings may reflect the influence of sleepiness-related diurnal variation or other physiological factors that differ between daytime and nighttime sleep.

In regards to the ESS values related to nocturnal hypoxemia, this variation is not consistent or significant enough to establish a robust relationship between daytime and nighttime ESS scores (12). Different individuals may experience varying patterns of sleepiness, influenced

by factors such as lifestyle, sleep habits, and underlying health conditions. Other factors, such as alterations in oxygenation may contribute to nocturnal hypoxemia (13). Another factor is sleep fragmentation, which might independently contribute to an increased risk of hypersomnolence in patients with OSA syndromes. These individual differences may contribute to the absence of a significant correlation between daytime and nighttime ESS scores.

CONCLUSION

This pilot study indicates that DPSG may serve as an effective alternative to NPSG in diagnosing OSA. Key diagnostic parameters, including AHI and ESS scores, showed no significant differences between groups. Although NPSG demonstrated higher sleep efficiency, other sleep architecture and cardiovascular measures were comparable. These findings support the potential role of DPSG in clinical practice, particularly when NPSG is not feasible. Further large-scale studies are recommended to validate these results.

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REFERENCES

1. Benjafield AV, Ayas NT, Eastwood PR, Heinzer R, Ip MSM, Morrell MJ, et al. Estimation of the global prevalence and burden of obstructive sleep apnoea: a literature-based analysis. Lancet Respir Med. 2019;7(8):687–98.
2. Faria A, Allen AH, Fox N, Ayas N, Laher I. The public health burden of obstructive sleep apnea. Sleep Sci (Sao Paulo, Brazil). 2021;14(3):257–65.
3. Sharma S, Mahajan S, Mahajan K. Obstructive sleep apnea and coronary artery disease: An unholy nexus or a holy alliance? Lung India. 2022;39(5):460.
4. Surani S, Taweesedt P. Obstructive sleep apnea: new perspective. Medicina (Kaunas). 2022;59(1):75.
5. Ching SM, Singh R, Azmi FSB, Chong KL, Ong CRS, Ayob NAB, et al. Prevalence and factors associated with probable obstructive sleep apnea among patients with hypertension in two primary care clinics in Miri, Sarawak, Malaysia. Ir J Med Sci. 2024;193(1):375–82.
6. Md Aris MA, Amir Hamzah AF, Abdullah F, Asha'ari ZA. Prevalence of high risk for obstructive sleep apnoea and its risk factors among adults attending government primary health clinics in Kuantan. IIUM Med J Malaysia. 2021;20(1).
7. Teplitzky, T. B., Zauher, A. J., & Isaiah, A. (2023). Alternatives to Polysomnography for the Diagnosis of Pediatric Obstructive Sleep Apnea. Diagnostics (Basel, Switzerland). 2023;13(11):1956.
8. Al-Jawder S, Bahammam A. Utility of daytime polysomnography for in-patients with suspected sleep-disordered breathing. Neurol Neurochir Pol. 2009;43(2):140–7.
9. Budhiraja, R., Javaheri, S., Parthasarathy, S., Berry, R. B., & Quan, S. F. The association between obstructive sleep apnea characterized by a minimum 3 percent oxygen desaturation or arousal hypopnea definition and hypertension. JCSM. 2019;15(09): 1261–1270.
10. Mou J, Zhou H, Huang S, Feng Z. The impact of comprehensive healthy lifestyles on obstructive sleep apnea and the mediating role of BMI: insights from NHANES 2005–2008 and 2015–2018. BMC Pulmonary Medicine. 2024 Dec 4;24(1).
11. Guo Q, Song W, Li W, Zeng C, Li Y, Mo J, et al. Weighted Epworth sleepiness scale predicted the apnea-hypopnea index better. Respir Res. 2020;21(1).
12. Persson HE, Svanborg E. Sleep deprivation worsens obstructive sleep apnea. Comparison between diurnal and nocturnal polysomnography. Chest. 1996;109(3):645–50.
13. Bédard M, Montplaisir J, Richer F, Malo J. Nocturnal hypoxemia as a determinant of vigilance impairment in sleep apnea syndrome. Chest. 1991;100(2):367–70.