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Comparison of Sleep Architecture in Individuals Aged 65 to 80 Years With and Without Mild Cognitive Impairment Using Multi-Channel Sleep Monitoring

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Background: This study aimed to compare sleep architecture using the apnea-hypopnea index (AHI) and wake after sleep onset (WASO) in 150 participants aged 65 to 80 years with and without mild cognitive impairment (MCI), using polysomnography, actigraphy, and home sleep monitoring, and assessed cognitive performance using the Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment (MoCA).


Material/Methods: A total of 150 participants were enrolled, including 75 patients with MCI and 75 cognitively healthy controls. Sleep architecture was evaluated using polysomnography, actigraphy, and home sleep monitoring systems. Cognitive function was assessed using MMSE and MoCA. Differences in sleep parameters, including AHI and WASO, were compared between groups. Pearson correlation and multiple linear regression analyses were performed to evaluate associations between sleep parameters and cognitive scores.

Results: Compared with controls, the MCI group showed significantly reduced slow-wave sleep duration (22.6 ± 14.6 vs 45.3 ± 16.7 min; $P < 0.01$), increased WASO ($42.5 \pm 7.1\%$ vs $29.1 \pm 5.9\%$; $P < 0.01$), and higher AHI (18.7 ± 6.2 vs 5.3 ± 2.0 ; $P < 0.01$). Slow-wave sleep duration was positively correlated with MMSE ($r = 0.72$; $P < 0.01$) and MoCA ($r = 0.68$; $P < 0.01$), whereas WASO was negatively correlated with MMSE ($r = -0.65$; $P < 0.01$) and MoCA ($r = -0.63$; $P < 0.01$). Multiple linear regression analysis demonstrated that slow-wave sleep duration remained independently associated with cognitive performance ($\beta = 0.41$; $P < 0.001$).

Conclusions: Alterations in sleep architecture, particularly reduced slow-wave sleep and increased nocturnal wakefulness, are significantly associated with poorer cognitive performance in older adults with MCI. Multi-channel sleep monitoring may facilitate early identification of sleep-related cognitive impairment and support clinical evaluation.

Keywords: Brain • Cognitive Dysfunction • Mild Cognitive Impairment • Multichannel Analysis • Sleep Apnea, Obstructive • Sleep Monitoring

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Introduction

Cognitive impairment represents a spectrum ranging from mild cognitive decline to severe dementia, with early stages often presenting subtle and frequently undetected deficits [1]. The global prevalence of cognitive impairment and dementia is increasing rapidly with population aging, creating a significant public health challenge [2]. The early identification of modifiable risk factors associated with cognitive decline is therefore essential to delay disease progression and improve quality of life in older adults [1]. Among the potential contributors to cognitive decline, sleep disturbances have emerged as an important and potentially modifiable factor [2]. Sleep plays a critical role in memory consolidation, emotional regulation, and overall cognitive functioning [2]. Accumulating evidence suggests that alterations in sleep architecture, particularly reductions in slow-wave sleep (SWS) and increased sleep fragmentation, may be associated with impaired cognitive performance and early neurodegenerative processes [2,3].

Recent epidemiological and clinical studies have reported associations between poor sleep quality and subjective cognitive issues in young adults [3], increased vascular and respiratory comorbidities affecting cognition in older adults [4], and delayed functional recovery after stroke [5]. These findings indicate that sleep disturbances can influence cognitive function across different populations and clinical conditions [3-5]. Technological advances in sleep monitoring have enabled more detailed assessment of sleep-cognition relationships [6]. Multi-channel sleep monitoring, integrating polysomnography, actigraphy, and home-based sensors, allows objective evaluation of sleep architecture under laboratory and real-world conditions [7,8]. Previous studies using these techniques have demonstrated their potential for detecting sleep disorders such as obstructive sleep apnea (OSA) and for characterizing sleep patterns associated with neurological and systemic diseases [6-8]. In addition, meta-analyses have shown that improving sleep quality through non-pharmacological interventions can lead to improvements in cognitive outcomes in individuals with mild cognitive impairment (MCI) [8,9]. These observations highlight the potential importance of sleep assessment in identifying individuals at risk for cognitive decline [8,9].

OSA is one of the most common sleep-related disorders associated with cognitive impairment [10]. OSA is characterized by repeated airway obstruction during sleep, leading to intermittent hypoxia, sleep fragmentation, and alterations in sleep architecture [11]. These physiological disturbances have been linked to neurocognitive dysfunction and can contribute to neurodegenerative processes [12,13]. Neuroimaging studies have further demonstrated associations between OSA and structural brain changes, including gray-matter loss, altered neural connectivity, and neurovascular dysregulation [14-16].

In addition, demographic and clinical factors such as age, sex, and cardiovascular comorbidities may influence the cognitive impact of sleep-disordered breathing [17-20].

Despite increasing evidence linking sleep disturbances with cognitive impairment, important questions remain unresolved [2,8,9]. In particular, the specific sleep architecture features most strongly associated with cognitive performance in older adults with MCI remain incompletely characterized, and the potential value of multi-channel sleep monitoring for identifying clinically relevant sleep-cognition relationships requires further investigation [7,8]. Therefore, the objective of the present study was to evaluate the association between sleep architecture parameters measured using multi-channel sleep monitoring and cognitive performance in older adults with MCI. We hypothesized that alterations in sleep architecture, particularly reduced slow-wave sleep and increased sleep fragmentation, would be associated with poorer cognitive performance as measured by standard neuropsychological assessments.

Material and Methods

Ethics Approval

The study protocol was reviewed and approved by the Ethics Committee of the Second Affiliated Hospital of Xiamen Medical College, Fujian, China (approval No. 2025136). All procedures conformed to the Declaration of Helsinki (revised 2013) and applicable national regulations. Written informed consent was obtained from all participants prior to inclusion.

Study Design and Participants

This cross-sectional study investigated the association between multi-channel sleep monitoring parameters and cognitive function in older adults. A total of 150 participants aged 65 to 80 years were recruited from community health clinics in Xiamen, Fujian Province, China, using convenience sampling among individuals attending routine health examinations. Each participant was informed of the study purpose and procedures and of their right to withdraw at any time. Personal identifiers were removed to ensure confidentiality, and all data were stored in coded electronic files accessible only to authorized investigators, to maintain participant confidentiality. Participants received modest compensation for their time. Neuropsychological assessments and multi-channel sleep monitoring were conducted at baseline for the primary cross-sectional analysis. A 6-month follow-up assessment was conducted for observational monitoring of participants; however, the present study primarily analyzed baseline cross-sectional associations between sleep parameters and cognitive performance. The flow diagram of the study is presented in **Figure 1**.

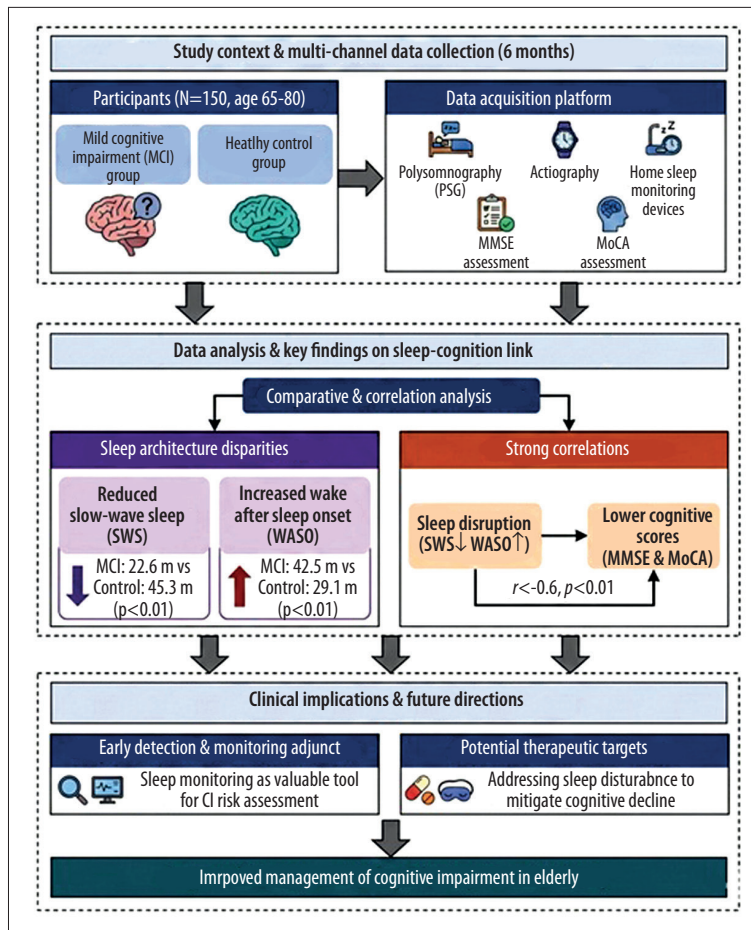


Figure 1. Study design and key findings.

Multi-channel sleep monitoring (polysomnography, actigraphy, and home monitoring) and cognitive assessments (Mini-Mental State Examination [MMSE] and the Montreal Cognitive Assessment [MoCA]) were performed in elderly participants. Individuals with mild cognitive impairment (MCI) showed reduced slow-wave sleep (SWS), increased wake after sleep onset (WASO), and lower cognitive scores compared with controls.

The primary objective of this study was to evaluate the relationship between sleep architecture parameters and cognitive performance in older adults with MCI.

Inclusion and Exclusion Criteria

Exclusion criteria included any history of neurological disorders (eg, dementia or stroke), psychiatric illness (eg, depression or schizophrenia), or serious systemic diseases that could affect sleep or cognition. Participants using sleep-related medications or diagnosed with severe sleep apnea or chronic insomnia were also excluded to ensure data integrity. Additional exclusion criteria included severe cardiovascular disease, substance abuse, untreated major psychiatric illness, and inability to complete sleep monitoring or cognitive assessments.

Multi-Channel Sleep Monitoring Procedures

Comprehensive sleep assessment was performed using polysomnography, actigraphy, and home-based sleep monitoring devices. Overnight polysomnography was conducted in a controlled sleep laboratory to record electroencephalogram, electro-oculogram, electromyogram, heart rate, and respiratory activity,

enabling classification of non-rapid eye movement and rapid eye movement stages, including SWS. Participants also wore wrist actigraphy devices throughout the study to record movement and activity patterns under natural conditions. Actigraphy provided estimates of total sleep time, wake after sleep onset (WASO), and sleep fragmentation. Additionally, portable electroencephalogram systems and pulse oximeters were used at home to continuously monitor brain activity and oxygen saturation, allowing ecological evaluation of sleep quality. The apnea-hypopnea index was calculated as the number of apnea and hypopnea events per hour of sleep and was used as a primary indicator of sleep-disordered breathing severity [21]. WASO was defined as the cumulative duration of wakefulness occurring after initial sleep onset and was used as a marker of sleep fragmentation [21]. The sleep monitoring devices used in this study have been validated in previous clinical sleep research and were calibrated according to manufacturer specifications prior to data collection to ensure measurement accuracy. Sleep parameters from polysomnography, actigraphy, and home monitoring systems were synchronized using standardized time-stamped recordings and integrated to generate a composite sleep profile for each participant.

Sleep Parameters and Definitions

The primary sleep parameters analyzed included total sleep time, sleep efficiency, SWS duration, sleep onset latency, WASO, and the proportions of rapid eye movement and deep sleep. Sleep efficiency was defined as the ratio of total sleep time to total time in bed, expressed as a percentage [22]. The sleep fragmentation index was derived from actigraphy data and reflected the frequency of nocturnal awakenings per hour [22]. Data from the 3 modalities were integrated to construct a comprehensive profile of each participant's sleep architecture for correlation with cognitive outcomes.

Cognitive Assessment and Diagnostic Criteria

Cognitive function was assessed using 2 validated instruments: the Mini-Mental State Examination (MMSE) and the Montreal Cognitive Assessment (MoCA) [23]. The MMSE is a 30-point screening tool assessing orientation, memory, attention, language, and visuospatial ability. Scores of 24 to 30 are considered normal, while lower scores indicate mild (20-26), moderate (10-20), or severe (<10) cognitive impairment [24]. The MoCA is a 30-point screening instrument designed to detect MCI and early dementia, covering domains including memory, attention, language, visuospatial skills, and executive function. A score of 26 or higher is considered normal [25]. The validity and reliability of the MMSE and MoCA as screening tools for cognitive impairment have been widely established in clinical research. MCI was diagnosed according to established clinical criteria, including subjective cognitive symptoms, objective cognitive impairment based on MoCA scores (≤ 26), largely preserved activities of daily living, and absence of dementia. Cognitive evaluations were conducted by trained clinicians to ensure diagnostic consistency. Both assessments were administered at baseline as part of the cross-sectional evaluation. Follow-up assessments were conducted for observational monitoring but were not included in the primary statistical analysis of the present study.

Statistical Analysis

Data were analyzed using SPSS version 25.0 (IBM Corp, Armonk, NY, USA). The normality of continuous variables was assessed using the Shapiro-Wilk test before performing parametric statistical analyses. Pearson correlation coefficients were calculated to assess relationships between key sleep parameters (SWS duration, WASO, and sleep efficiency) and cognitive scores (MMSE and MoCA). Independent-samples *t* tests compared continuous variables between the MCI and control groups, while chi-square tests assessed categorical variables.

Multiple linear regression analysis was applied to evaluate the predictive influence of sleep parameters on cognitive

performance, adjusting for age, sex, education level, and continuous positive airway pressure (CPAP) use as a potential confounding factor. The model was defined as:

$$\text{Cognitive Score} = \beta_0 + \beta_1 (\text{SWS Duration}) + \beta_2 (\text{WASO}) + \beta_3 (\text{Sleep Efficiency}) + \epsilon$$

In this model, the cognitive score represents either the MMSE or MoCA value, reflecting overall cognitive function. SWS duration denotes the time spent in SWS (minutes), WASO indicates the percentage of wake time after sleep onset, and sleep efficiency represents the ratio of total sleep time to time spent in bed, expressed as a percentage. The term β_0 refers to the intercept, β_1 - β_3 denotes the regression coefficients for each predictor variable, and ϵ represents the random error term. Multicollinearity among predictors was evaluated using variance inflation factor values. Missing data were minimal (<5%) and were handled using complete-case analysis. Pearson correlation analysis was selected to evaluate linear relationships between sleep parameters and cognitive scores, whereas multiple linear regression analysis was used to determine independent associations after adjustment for potential confounders. A 2-tailed $P < 0.05$ was considered statistically significant. Descriptive statistics summarized demographic and baseline characteristics of the study population. The sample size of 150 participants was considered adequate to detect moderate correlations between sleep parameters and cognitive outcomes with sufficient statistical power.

Results

Baseline Demographic and Clinical Characteristics

A total of 150 participants were enrolled, including 75 with MCI and 75 age-matched healthy controls. The mean \pm SD age was 72.3 ± 4.2 years, with no significant difference between groups ($P = 0.46$). Sex distribution was balanced (52% men in both groups). These similarities indicate that the 2 groups were comparable with respect to major demographic characteristics. Baseline cognitive assessments showed a clear group separation. The MCI group exhibited significantly lower MMSE and MoCA scores (MMSE, 24.2 ± 3.1 vs 29.1 ± 0.9 ; MoCA, 22.6 ± 4.2 vs 28.7 ± 1.0 ; both $P < 0.01$; **Figure 2A, 2B**). Boxplots illustrate broader score dispersion in the MCI group, confirming mild but measurable cognitive decline. Demographic and clinical data are summarized in **Table 1**. No significant group differences were observed in body mass index, hypertension, diabetes, education level, or smoking history. However, the MCI group had shorter sleep duration (6.3 ± 1.1 h vs 7.0 ± 1.0 h; $P = 0.02$), higher apnea-hypopnea index scores (18.7 ± 6.2 vs 5.3 ± 2.0 ; $P < 0.01$), and more frequent CPAP use (22.7% vs 2.7%; $P < 0.01$). These findings suggest that sleep-disordered breathing was more prevalent among participants with cognitive impairment.

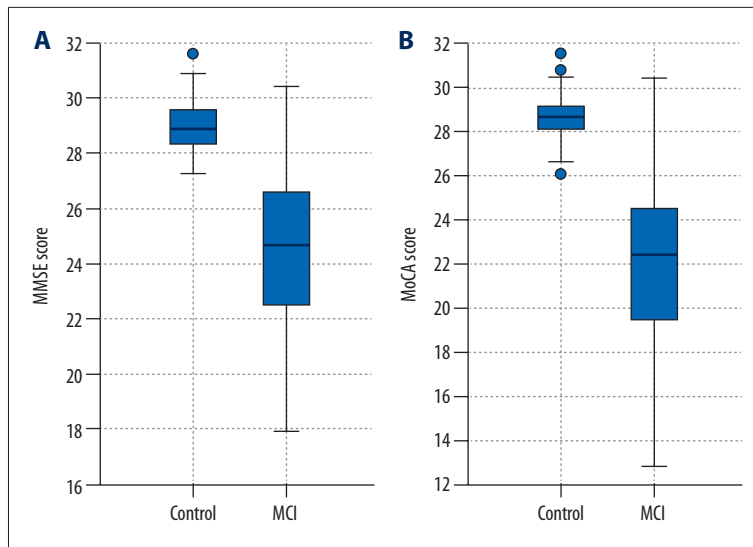


Figure 2. Cognitive performance in study groups. (A) Mini-Mental State Examination (MMSE) and (B) Montreal Cognitive Assessment (MoCA) scores were significantly lower in the mild cognitive impairment (MCI) group than in controls ($P<0.01$).

Table 1. Baseline demographic, clinical, and cognitive characteristics of participants.

Variable	MCI group (n=75)	Control group (n=75)	P value
Age (years, mean±SD)	72.5±4.3	72.1±4.1	0.46
Sex (male,%)	52	52	1.00
MMSE score	24.2±3.1	29.1±0.9	<0.01
MoCA score	22.6±4.2	28.7±1.0	<0.01
Body mass index (kg/m ²)	27.4±3.2	26.8±3.0	0.23
Hypertension (%)	61	59	0.74
Diabetes mellitus (%)	22	20	0.78
Education level (years)	13.1±2.7	14.3±3.2	0.15
Smoking history (%)	12	10	0.67
Sleep duration (hours/night)	6.3±1.1	7.0±1.0	0.02
Apnea-hypopnea index	18.7±6.2	5.3±2.0	<0.01
CPAP usage (n,%)	17 (22.7%)	2 (2.7%)	<0.01

MCI, mild cognitive impairment; MMSE, Mini-Mental State Examination; MoCA, Montreal Cognitive Assessment; CPAP, continuous positive airway pressure.

Comparison of Sleep Architecture Parameters

Multi-channel sleep monitoring revealed notable alterations in sleep architecture in the MCI group. Total sleep time was significantly shorter (5.9±1.3 h vs 7.2±1.2 h; $P<0.01$), while WASO was substantially higher (42.5±7.1% vs 29.1±5.9%; $P<0.01$). Sleep onset latency was also prolonged (25.3±12.8 min vs 18.5±9.7 min; $P=0.03$). SWS duration in the MCI group was nearly 50% less than in the control group (22.6±14.6 min vs 45.3±16.7 min; $P<0.01$; **Figure 3**). Sleep efficiency was lower in the MCI group (78.2±6.3% vs 83.4±5.9%; $P<0.01$), and the sleep fragmentation index was elevated (19.4±4.7 vs 12.8±3.9 episodes per hour; $P<0.01$). Collectively, these findings indicate

more fragmented and less restorative sleep patterns among individuals with MCI. Detailed comparisons of sleep parameters between groups are presented in **Table 2**. The association between SWS duration and cognitive scores is illustrated in **Figure 4**, and the relationship between WASO and MoCA scores is shown in **Figure 5**.

Correlation Between Sleep Parameters and Cognitive Function

Pearson correlation analysis demonstrated significant associations between sleep quality indicators and cognitive performance. SWS duration showed a positive association with both

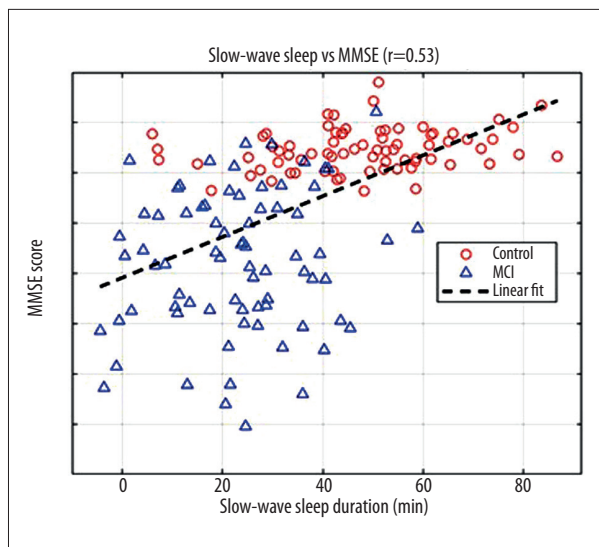


Figure 3. Association between slow-wave sleep duration and Mini-Mental State Examination (MMSE) score shows a positive correlation ($r=0.53$; $P<0.01$).

the MMSE ($r=0.72$; $P<0.01$) and MoCA ($r=0.68$; $P<0.01$), whereas WASO correlated negatively with MMSE ($r=-0.65$; $P<0.01$) and MoCA ($r=-0.63$; $P<0.01$). Total sleep time and sleep efficiency were also positively associated with cognitive scores (Table 3). Although the observed correlations were relatively high, multicollinearity diagnostics indicated stable model estimates (variance inflation factor <2), suggesting that the associations were not attributable to collinearity among predictors. A correlation heatmap (Figure 6) illustrates these relationships,

Table 2. Comparison of sleep architecture parameters between mild cognitive impairment (MCI) and control groups assessed by multi-channel sleep monitoring.

Sleep parameter	MCI group (n=75)	Control group (n=75)	P value
Total sleep time (hours)	5.9±1.3	7.2±1.2	<0.01
Wake after sleep onset (WASO,%)	42.5±7.1	29.1±5.9	<0.01
Sleep onset latency (minutes)	25.3±12.8	18.5±9.7	0.03
Slow-wave sleep duration (minutes)	22.6±14.6	45.3±16.7	<0.01
Rapid eye movement (REM) Sleep (minutes)	90.3±22.1	95.6±21.7	0.28
Light sleep duration (minutes)	218.9±45.2	230.4±41.8	0.12
Sleep efficiency (%)	78.2±6.3	83.4±5.9	<0.01
Sleep fragmentation index (episodes h ⁻¹)	19.4±4.7	12.8±3.9	<0.01
Percentage of deep sleep (%)	10.3±3.5	17.2±4.1	<0.01
Percentage of REM sleep (%)	18.3±4.7	21.7±5.2	0.02
Number of awakenings (per night)	4.7±1.9	3.2±1.4	0.04
CPAP usage (%)	23.5	3.2	<0.01

CPAP, continuous positive airway pressure.

highlighting positive associations between SWS and sleep efficiency with cognitive scores and negative associations between WASO, sleep fragmentation, and cognitive performance.

Multiple Linear Regression Analysis of Cognitive Performance

Multiple linear regression analysis was conducted to determine whether sleep parameters independently predicted cognitive performance after adjustment for age, sex, education level, and CPAP use. As shown in Table 4, SWS duration demonstrated a significant positive association with cognitive scores ($\beta=0.41$; 95% CI, 0.25-0.58; $P<0.001$). In contrast, WASO showed a significant negative association with cognitive performance ($\beta=-0.36$; 95% CI, -0.52 to -0.19; $P<0.001$). Sleep efficiency also remained positively associated with cognitive outcomes ($\beta=0.21$; 95% CI, 0.04-0.38; $P=0.015$). The overall regression model explained a substantial proportion of variance in cognitive scores ($R^2=0.56$; adjusted $R^2=0.54$). Multicollinearity diagnostics indicated acceptable variance inflation factor values (<2), suggesting stable model estimates.

Integrated Visualization of Sleep-Cognition Relationships

A 3-dimensional scatter plot (Figure 7) illustrates the combined relationship between SWS duration, WASO, and MMSE scores. Participants with longer SWS and lower WASO tended to demonstrate higher cognitive scores. Radar visualization further confirmed global reductions in restorative sleep parameters in the MCI group. A concise summary of the key

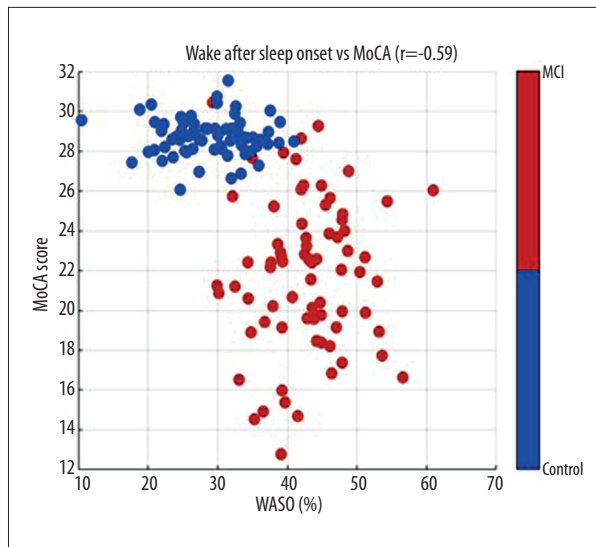


Figure 4. Association between wake after sleep onset (WASO) and Montreal Cognitive Assessment (MoCA) score shows negative correlation between WASO (%) and MoCA score ($r=-0.59$; $P<0.01$).

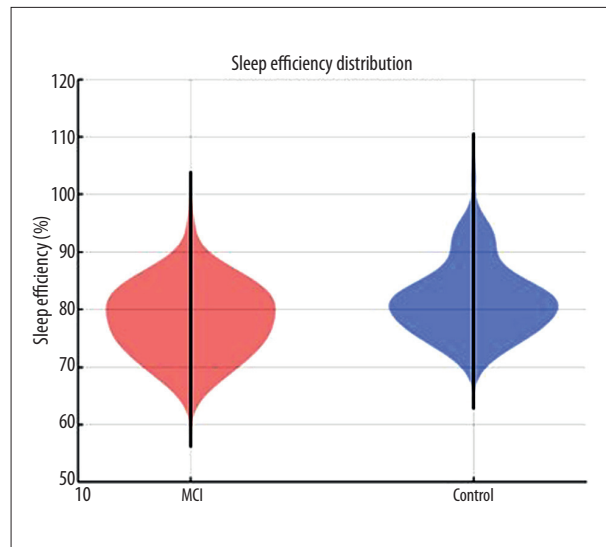


Figure 5. Sleep efficiency in study groups. Sleep efficiency was significantly lower in the mild cognitive impairment (MCI) group than in the control group ($P<0.01$).

Table 3. Correlation between sleep parameters and cognitive performance measured with the Mini-Mental State Examination (MMSE) and the Montreal Cognitive Assessment (MoCA).

Sleep parameter	MMSE (r)	MoCA (r)	P value
Slow-wave sleep duration (minutes)	+0.72	+0.68	<0.01
Wake after sleep onset (%)	-0.65	-0.63	<0.01
Total sleep time (hours)	+0.59	+0.57	<0.01
Sleep efficiency (%)	+0.75	+0.71	<0.01
Sleep onset latency (minutes)	-0.60	-0.57	0.02
Light sleep duration (minutes)	-0.45	-0.41	0.04
REM sleep duration (minutes)	-0.50	-0.47	0.02
Sleep fragmentation index (episodes h ⁻¹)	-0.55	-0.52	0.01
Percentage of deep sleep (%)	+0.60	+0.58	<0.01
Number of awakenings (per night)	-0.51	-0.48	0.03
CPAP usage (%)	-0.37	-0.35	0.10

REM, rapid eye movement; CPAP, continuous positive airway pressure.

sleep-cognition associations identified through multi-channel monitoring is presented in **Table 5**, highlighting the sleep parameters that demonstrated the strongest relationships with MMSE and MoCA scores. Radar visualization (**Figure 8**) further confirmed global reductions in restorative sleep parameters in the MCI group. Overall, reduced SWS and increased nocturnal wakefulness emerged as the sleep characteristics most strongly associated with poorer cognitive performance in this cohort.

Discussion

In this clinical study based on multi-channel sleep monitoring, we found that older adults with MCI displayed significant alterations in sleep architecture compared with age-matched controls, and that these abnormalities were closely associated with cognitive performance. The MCI group demonstrated shorter total sleep time, longer sleep onset latency, reduced sleep efficiency, higher WASO, and markedly reduced SWS. In

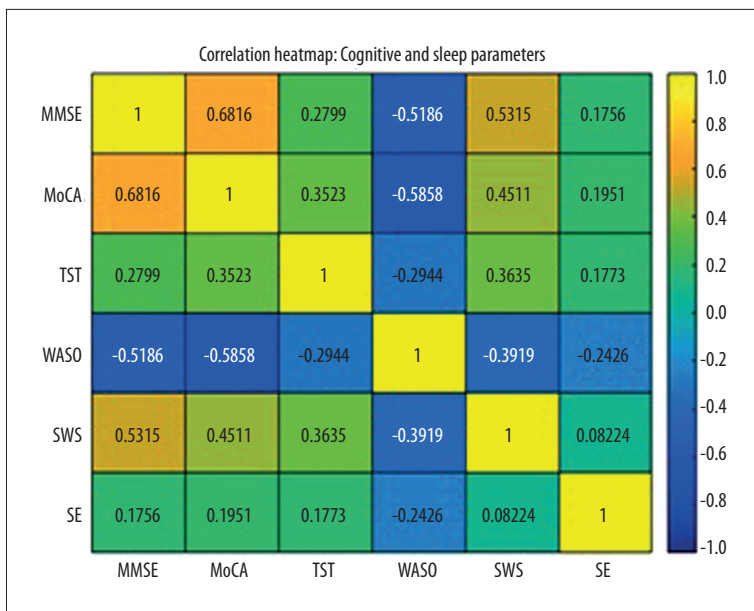


Figure 6. Correlation heatmap of sleep parameters and cognitive scores. Slow-wave sleep (SWS) and sleep efficiency (SE) correlated positively with cognitive scores, whereas wake after sleep onset (WASO) showed negative correlations. MMSE – Mini-Mental State Examination; MoCA, Montreal Cognitive Assessment; TST, total sleep time; SWS, slow-wave sleep.

Table 4. Multiple linear regression analysis of sleep parameters associated with cognitive performance.

Predictor variable	β	95% CI	Standardized β	P value	VIF
Slow-wave sleep duration (minutes)	0.41	0.25 to 0.58	0.46	<0.001	1.32
Wake after sleep onset (%)	-0.36	-0.52 to -0.19	-0.39	<0.001	1.28
Sleep efficiency (%)	0.21	0.04 to 0.38	0.24	0.015	1.19

Model statistics: $R^2=0.56$; adjusted $R^2=0.54$; $F=21.7$; $P<0.001$. VIF, variance inflation factor.

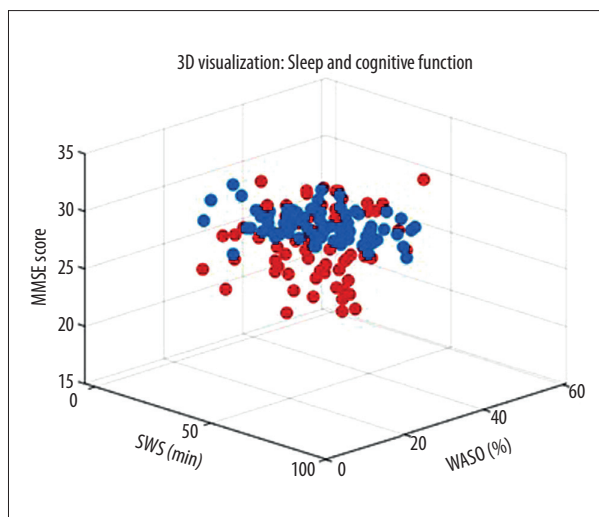


Figure 7. Three-dimensional relationship between sleep parameters and cognition. Shorter slow-wave sleep (SWS) duration and higher wake after sleep onset (WASO) were associated with lower Mini-Mental State Examination (MMSE) scores, particularly in participants with mild cognitive impairment (MCI).

addition, higher apnea-hypopnea index values and more frequent CPAP use were observed among participants with MCI. Cognitive scores (MMSE and MoCA) were significantly lower in the MCI group, indicating mild but clinically meaningful impairment. These findings suggest that disrupted sleep architecture, particularly reduced deep sleep and increased fragmentation, is closely associated with poorer cognitive performance in older adults. These findings are consistent with previous population-based and clinical studies showing that sleep disturbances and short sleep duration are associated with worse cognitive outcomes and higher prevalence of cognitive impairment in aging populations [14,26,27]. In the present study, SWS duration and sleep efficiency showed the strongest positive correlations with MMSE and MoCA scores, whereas WASO, sleep fragmentation, and nocturnal awakenings were negatively associated with cognitive performance. These observations support the hypothesis that restorative sleep stages, particularly SWS, may play an important role in maintaining cognitive function in later life. Prior research has similarly demonstrated that reduced SWS and disrupted rest-activity rhythms are linked to early neurodegenerative changes in amnesic MCI and preclinical Alzheimer disease [12,15,16,27].

Table 5. Summary of associations between sleep parameters and cognitive performance identified by multi-channel monitoring.

Key sleep metrics	Cognitive association	Direction of effect	Statistical significance (P)
Slow-wave sleep (minutes)	Strong link with MMSE/MoCA	Positive	<0.01
WASO (%)	Correlates with reduced cognitive scores	Negative	<0.01
Sleep efficiency (%)	Predicts higher MMSE/MoCA	Positive	<0.01
Sleep fragmentation index	Associated with worse attention and memory	Negative	0.01
Deep sleep (%)	Related to improved global cognition	Positive	<0.01

MMSE, Mini-Mental State Examination; MoCA, Montreal Cognitive Assessment; WASO, wake after sleep onset.

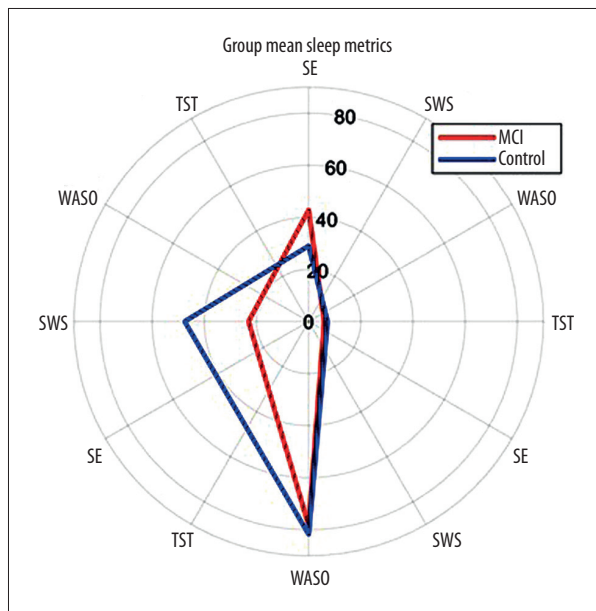


Figure 8. Radar plot illustrating differences in mean sleep parameters, including total sleep time (TST), slow-wave sleep (SWS), wake after sleep onset (WASO), and sleep efficiency (SE), between the mild cognitive impairment (MCI) and control groups.

Sleep-disordered breathing may represent one potential mechanism linking sleep disruption and cognitive impairment. In our cohort, the MCI group exhibited higher apnea-hypopnea index values and a greater proportion of CPAP users, suggesting a higher burden of OSA, which has been widely associated with intermittent hypoxia, repeated arousals, vascular dysfunction, and impaired metabolic clearance, all of which can contribute to neurodegenerative processes and cognitive decline [20,28-30]. Although CPAP therapy is known to improve sleep architecture and may support cognitive performance in some patients, the modest correlations observed in this study indicate that CPAP use alone may not fully normalize sleep disturbances in individuals with MCI. Previous clinical studies have

reported heterogeneous cognitive responses to CPAP therapy, likely influenced by treatment adherence, disease severity, and comorbid conditions [31,32]. These findings highlight the complexity of the relationship between sleep-disordered breathing and cognitive impairment in aging populations. The present study also highlights the potential value of objective multi-channel sleep monitoring for characterizing sleep disturbances in individuals with cognitive impairment. While subjective sleep concerns are common in older adults, objective monitoring methods, such as polysomnography and actigraphy, provide more precise measurements of sleep architecture and fragmentation. Recent research has demonstrated the feasibility of wearable and home-based monitoring systems for evaluating sleep patterns in older adult populations and patients with neurological disorders [8,29,33,34]. Our integrated approach combining polysomnography, actigraphy, and home monitoring devices enabled comprehensive characterization of sleep patterns and may represent a practical strategy for identifying sleep abnormalities associated with cognitive impairment. From a clinical perspective, these findings suggest that sleep disturbances may represent a potentially modifiable factor associated with cognitive decline. Previous systematic reviews have reported that behavioral sleep interventions, including cognitive behavioral therapy for insomnia, sleep hygiene programs, and optimization of OSA treatment, can improve sleep quality in older adults and may also benefit cognitive function and quality of life [7-9]. Identification of individuals with pronounced reductions in SWS, low sleep efficiency, and elevated nocturnal wakefulness may therefore help clinicians identify patients who could benefit from targeted sleep interventions.

Several limitations should be considered when interpreting the results of this study. First, the cross-sectional design prevents conclusions about causal relationships between sleep disturbances and cognitive decline. It remains unclear whether sleep abnormalities contribute to cognitive impairment or whether neurodegenerative changes disrupt sleep regulation; existing

evidence suggests a bidirectional relationship between these processes [27]. Longitudinal studies with repeated sleep and cognitive assessments are therefore needed to clarify temporal relationships. Second, the study population consisted of older adults within a relatively narrow age range, which may limit the generalizability of the findings to other age groups or populations. Third, although multi-channel monitoring provides detailed sleep information, variability in home recording conditions and night-to-night sleep fluctuations may introduce measurement variability. In addition, certain methods used in this study, including actigraphy and home-based monitoring systems, may be less precise than laboratory-based polysomnography, which represents a limitation of the measurement methods. Future research should expand on these findings through larger, multicenter longitudinal studies that integrate sleep monitoring with neuroimaging and biomarker analysis. Combining physiological sleep metrics with structural brain imaging and emerging biomarkers of neurodegeneration may improve early detection of individuals at high risk for cognitive decline. Additionally, interventional trials examining whether targeted treatment of sleep disturbances can improve cognitive outcomes in individuals with MCI would provide important insights into the clinical relevance of sleep-based therapeutic strategies. Overall, our findings demonstrate that older adults with MCI exhibit characteristic alterations in sleep architecture, including reduced SWS and increased sleep fragmentation, which are significantly associated with cognitive performance. These results support the potential role of objective sleep monitoring as a complementary tool for identifying sleep abnormalities linked to cognitive impairment and highlight the importance of addressing sleep disturbances in the clinical management of older adults at risk for cognitive decline.

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Conclusions

This study demonstrates that older adults with MCI experience significant alterations in sleep architecture, including reduced SWS, increased WASO, and lower sleep efficiency. These sleep parameters were significantly associated with poorer cognitive performance, as reflected by MMSE and MoCA scores, highlighting the close interrelationship between sleep quality and cognitive health. Altered sleep architecture, particularly reduced SWS and increased nocturnal wakefulness, is significantly associated with poorer cognitive performance in older adults with MCI. Objective multi-channel sleep monitoring may provide a useful approach for identifying sleep abnormalities linked to cognitive impairment in older adults. However, because this study used a cross-sectional design, causal relationships between sleep disturbances and cognitive decline cannot be established. Further longitudinal studies are needed to determine whether improving sleep architecture may influence cognitive outcomes in aging populations.

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Patient Permission/Consent Declarations

Written informed consent was obtained from all participants prior to inclusion.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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