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Sleep Architecture and Efficiency in Older Adults: A Clinical Analysis in the Context of Obstructive Sleep Apnea

—How Aging Shapes Sleep: A Closer Look at Sleep Architecture and Efficiency in Older Adults with Suspected Sleep Apnea

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Abstract

Introduction: Obstructive sleep apnea (OSA) is highly prevalent among older adults and is often accompanied by increased sleep fragmentation and progressive deterioration of both deep (N3) and REM sleep. Despite its impact on functional capacity and cardiovascular risk, detailed descriptions of sleep architecture in this population remain limited—especially in Latin American cohorts. This study aims to analyze polysomnographic characteristics and sleep fragmentation in a geriatric cohort with suspected OSA from Colombia's Caribbean region, focusing on the proportion of N3 sleep as a potential functional marker. **Methods:** This was a prospective cross-sectional study based on a consecutive cohort of older adults (≥60 years) who underwent full-night attended polysomnography (Type I) between August 2023 and March 2025. Clinical, anthropometric, and polysomnographic variables were collected. Descriptive and comparative analyses were performed according to sex, age group (60 - 69 vs. ≥70 years), OSA severity, and proportion of N3 sleep (<10% vs. ≥10%).

Correlation testing, multivariate linear regression, and data visualization through statistical figures were used to explore associations. **Results:** A total of 129 patients were included. Mean age was (69.8 ± 6.5) years, with a male predominance (60.5%). Moderate to severe OSA was observed in 79.8% of the cohort. Patients aged ≥70 showed significantly lower proportions of N3 and REM sleep, and higher proportions of N1 sleep and microarousals. Women exhibited greater sleep fragmentation and reduced deep sleep. Age positively correlated with microarousal index (r = 0.34). Multivariate regression analysis showed a negative trend between age and N3 sleep ($\beta = -0.17$; p = 0.077). Those with <10% N3 sleep had higher AHI, more fragmentation, and lower minimum oxygen saturation. **Conclusion:** In older adults from Colombia's Caribbean coast with suspected OSA, sleep architecture shows significant alterations not fully explained by AHI alone. The progressive fragmentation and loss of deep sleep observed in older and more severe OSA subgroups may serve as functional indicators of physiological decline in geriatric care. These findings highlight the need to incorporate structural sleep variables—such as N3 proportion and microarousal index—into the comprehensive clinical assessment of OSA in older adults.

Keywords

DeCS/MeSH Terminology: Sleep-Disordered Breathing, Sleep Apnea, Obstructive, Sleep Architecture, Sleep Efficiency, Sleep, Slow-Wave, Aged, Sleep Fragmentation, Polysomnography, Colombia

1. Introduction

Biological aging affects multiple physiological domains, and sleep is no exception. In older adults, characteristic changes in sleep architecture have been documented, such as reduced total sleep time (TST), decreased proportion of deep sleep (N3), increased light sleep (N1), diminished REM sleep, prolonged REM latency, more frequent nocturnal awakenings, and an overall decline in sleep efficiency [1]. While these alterations may be partly physiological, they can become pathological when associated with sleep-related breathing disorders—particularly obstructive sleep apnea (OSA), whose prevalence and severity tend to increase with age.

Beyond intrinsic aging, additional factors may exacerbate sleep deterioration in geriatric populations, especially in regions like Colombia's Caribbean coast. Chronic exposure to environmental pollution—particularly biomass smoke from wood-burning stoves—along with tobacco use, and the cumulative effects of environmental, social, and occupational changes are frequent contributors. These conditions, common in urban coastal settings, may further disrupt sleep architecture and circadian homeostasis, interfering with adaptive aging mechanisms and facilitating the onset or worsening of sleep-disordered breathing [2].

OSA is a sleep-related breathing disorder characterized by recurrent episodes

of partial or complete upper airway collapse during sleep, leading to intermittent hypoxemia, hypercapnia, and microarousals that fragment the sleep-wake cycle [3]. Its prevalence in the general adult population is estimated between 9% and 38%, but this figure exceeds 50% in individuals over 60 and can approach 80% in older men with obesity or coexisting cardiovascular disease [4]. In older adults, OSA often presents atypically, with less frequent daytime sleepiness and higher rates of neurocognitive symptoms, insomnia, functional impairment, and cardiovascular impact [5]. This subtler clinical picture, overlapping with common geriatric syndromes, contributes to underdiagnosis or delayed recognition [6].

From a geriatric perspective, understanding the interplay between physiological aging and OSA-induced sleep disruption is essential. Several studies have shown that older adults with OSA exhibit more profound alterations in sleep architecture compared to younger individuals, even after adjusting for apnea-hypopnea index (AHI) severity. A recent Egyptian study comparing older adults with OSA to middle-aged patients found that the older group had lower sleep efficiency, higher N1 proportion, and reduced N3 and REM sleep, despite similar desaturation indices [7]. This dissociation suggests that age may independently modify the neurophysiological impact of OSA.

Sleep fragmentation—quantified by the microarousal index—has emerged as a key functional biomarker of OSA impact, beyond AHI. Landmark studies like the Sleep Heart Health Study have shown that elevated microarousal indices are associated with a higher risk of nocturnal hypertension, impaired verbal memory, and deficits in sustained attention [8]. In older adults, fragmentation may be even more consequential, as it exacerbates cognitive vulnerability and reduces the restorative function of sleep. Thus, the microarousal index holds particular clinical value in geriatric assessment, though it remains underutilized in many local settings.

In this context, the concept of a "functional marker" gains importance. Unlike purely descriptive polysomnographic variables, a functional marker refers to a sleep parameter that reflects the physiological reserve and adaptive capacity of the older adult, serving as an indicator of vulnerability to decline. Specifically, the proportion of N3 sleep has been proposed as such a marker, given its associations with cognitive restoration, synaptic plasticity, and frailty-related outcomes in aging. By operationally defining N3 sleep as a functional marker, this study aims to explore its role in characterizing geriatric vulnerability within the framework of OSA.

In Colombia, evidence on OSA in older adults is limited and heterogeneous. While some studies have validated screening tools such as STOP-BANG and NoSAS in general urban populations [9], few publications have addressed the polysomnographic profile of adults over 60. In Bogotá, Severiche *et al.* (2025) reported a high prevalence of REM-related OSA in older adults, with greater sleep fragmentation and stronger associations with functional decline on geriatric scales—underscoring the need to tailor diagnostic strategies to older Colombian patients [10]. However, no prior studies have used full Type I polysomnography to characterize sleep in older adults from the Caribbean region—a historically underserved area in terms of access to sleep diagnostics.

The Colombian Caribbean presents a complex epidemiological context. The high burden of poorly controlled chronic diseases, the predominance of socioeconomically vulnerable groups, and the limited availability of neurophysiology and sleep laboratories constrain timely OSA diagnosis in the region. Furthermore, it remains unknown whether polysomnographic patterns observed in older adults from other regions are generalizable to this diverse population. This highlights the need for locally generated evidence to inform regionalized clinical guidelines and public health policy.

Against this backdrop, the present study aims to thoroughly characterize sleep architecture, efficiency, and nocturnal fragmentation in adults aged 60 and older with clinical suspicion of OSA, assessed using Type I polysomnography in specialized institutions from Colombia's Caribbean coast. Using an analytical cross-sectional design, we estimate the proportion of sleep in each stage (N1, N2, N3, REM), assess overall sleep efficiency, and examine the microarousal index as a functional marker of disruption. This approach will help identify aging-specific patterns, differentiate the influence of AHI severity, and establish an empirical foundation for future predictive models centered on the older adult population.

2. Materials and Methods

2.1. Patient Selection and Variable Definitions

This was a prospective, observational, cross-sectional analytical study based on a cohort of patients with clinical suspicion of obstructive sleep apnea (OSA) who underwent Type I polysomnography between August 2023 and March 2025. Data were prospectively collected from two specialized sleep medicine centers in Colombia's Caribbean region as part of a broader institutional project on diagnostic approaches to sleep-related breathing disorders. This subanalysis focused exclusively on older adults, defined as individuals aged 60 years or older, with the aim of characterizing sleep efficiency, architecture, and fragmentation in this population.

We included patients ≥60 years old with a complete polysomnographic recording of at least 360 minutes of interpretable data and complete clinical, anthropometric, and laboratory information. Exclusion criteria were a prior diagnosis of other sleep disorders (e.g., chronic primary insomnia, narcolepsy, or periodic limb movement disorder), previous treatment with CPAP or mandibular advancement devices, or major neurological conditions that could interfere with sleep architecture, such as advanced dementia or Parkinson's disease.

Clinical and demographic variables were extracted from electronic medical records and institutional databases. Data collected included age (both continuous and categorical: 60 - 69, 70 - 79, ≥80), sex, body mass index (BMI, kg/m^2), marital status, educational level, and socioeconomic stratum. We also documented chronic comorbidities such as hypertension, type 2 diabetes, dyslipidemia, cardiovascular disease, and smoking history (active, former, or never smoker).

The decision to stratify patients into 60 - 69 versus ≥70 years was based on previous geriatric sleep research, where age 70 is frequently used as a threshold for increased frailty and accelerated decline in restorative sleep stages (e.g., N3 and REM). This cut-off has also been applied in large population-based studies such as the HypnoLaus and Sleep Heart Health Study, which reported steeper deterioration in sleep architecture beyond this age. Similarly, the selection of <10% versus ≥10% of N3 sleep as a categorical variable follows prior literature identifying this threshold as clinically relevant in older adults. Proportions of N3 below 10% have been associated with frailty, functional decline, and increased mortality. These thresholds, therefore, provide a clinically meaningful framework to explore vulnerability markers in this population.

2.2. Data Collection and Processing

All Type I polysomnographic studies were performed by certified technicians and supervised by sleep medicine specialists, following the technical standards of the American Academy of Sleep Medicine (AASM). The recordings included continuous monitoring of EEG (C3-M2 and C4-M1 channels), EOG, submental EMG, thoracoabdominal respiratory effort, nasal and oral airflow (thermistor and nasal pressure transducer), pulse oximetry, snore sensor, single-lead ECG, and body position.

The following variables were extracted from the PSG reports: total sleep time (TST, in minutes), sleep efficiency (ratio of TST to total time in bed), apnea-hypopnea index (AHI, defined as number of respiratory events per hour of sleep), microarousal index (arousals per hour), average heart rate, minimum and mean oxygen saturation, and percentage of time spent in each sleep stage (N1, N2, N3, REM), expressed as a proportion of TST. All variables were validated through double review and manually curated to eliminate duplicate or incomplete records. The final sample of older adults with valid data comprised 129 patients, representing 27.9% of the initial cohort (n = 462). Data were anonymized to ensure confidentiality.

2.3. Database Cleaning and Management

After structuring the final dataset, we performed data cleaning and tabulation. Missing values were assessed and handled using listwise deletion when the missing rate was <5%. For key continuous variables (efficiency, sleep stages, AHI), distribution was evaluated using Shapiro-Wilk, Kolmogorov-Smirnov, and Anderson-Darling tests, complemented by visual inspection of histograms and Q-Q plots. These assessments informed the selection of appropriate parametric or non-parametric statistical tests.

The older adult subsample (n = 129) allowed estimation of means with an absolute precision of ±5 percentage points for continuous variables and detection of intergroup differences of at least 10 points with ≥80% power, assuming 95% confidence and moderate variances.

2.4. Statistical Analysis

The statistical analysis was conducted in three phases. First, univariate descriptive statistics were generated. Continuous variables with normal distribution were reported as mean ± standard deviation; those with non-normal distribution were summarized as median and interquartile range. Categorical variables were described using absolute and relative frequencies.

Second, bivariate analyses were conducted to compare groups based on AHI severity categories (mild: 5 - 14.9; moderate: 15 - 29.9; severe: ≥30 events/hour), sex, and presence of comorbidities. For continuous variables, comparisons used Student's t-test or Mann-Whitney U test as appropriate; for categorical variables, Pearson's chi-square test or Fisher's exact test was used when expected frequencies were <5.

In the third phase, multivariate analysis was performed using multiple linear regression models to identify independent predictors of three key outcomes: sleep efficiency, microarousal index, and N3 sleep proportion. Models were adjusted for age, sex, BMI, and presence of hypertension or cardiovascular disease, predefined as potential confounders. Assumptions of linear models were verified, including normality of residuals, homoscedasticity, and absence of multicollinearity (tolerance > 0.7 and VIF < 2). Statistical significance was set at $p < 0.05$. Analyses were conducted using Jamovi v2.3.18 (based on R), with partial replication in SPSS v26.0.

This study was approved by the Research Ethics Committee of Universidad del Sinú—Cartagena Campus (Approval No. 05-2023) as part of a registered protocol. All patients in the original cohort provided written informed consent, and this subanalysis was conducted using anonymized data, in compliance with the Declaration of Helsinki and Colombian ethical regulations (Resolution 8430 of 1993, Ministry of Health).

3. Results

A total of 129 older adults (≥60 years) with clinical suspicion of obstructive sleep apnea (OSA) were included, all evaluated by Type I polysomnography between August 2023 and March 2025 at two specialized centers in Colombia's Caribbean region. The analyzed cohort reflected a clinically representative profile of the local geriatric population, with a high burden of comorbidities and a predominance of males (60.5%). The mean age was (69.8 ± 6.5) years (range: 60 - 89), allowing for comparative assessment of sleep changes between the 60 - 69 and ≥70 age groups. The mean body mass index (BMI) was (29.3 ± 4.5) kg/m^2, classifying most patients as overweight or obese, risk factors consistent with previous Latin American OSA studies.

The apnea-hypopnea index (AHI) showed a pathological distribution, with a median of 28.4 events/hour (IQR: 16.7 - 45.6). Most patients had moderate (37.2%) or severe (42.6%) OSA, confirming a high burden of sleep-disordered breathing in this non-institutionalized geriatric population. The mean total sleep time (TST)

was (334 ± 49) minutes, and sleep efficiency—when available—was generally below 85%, indicating significant fragmentation.

3.1. Clinical Profile and Sex-Based Differences

Table 1 summarizes clinical and polysomnographic characteristics by sex. While age was similar between men and women, meaningful differences in sleep structure were observed. Women showed a higher proportion of N1 sleep, indicating a greater tendency toward light sleep. In contrast, men had slightly higher percentages of N3 sleep, which is considered physiologically restorative. Minimum oxygen saturation was lower in men, consistent with their greater burden of severe OSA. Figure 1 presents these sex-based differences via boxplots, highlighting greater dispersion of N3 sleep percentage in women, which may reflect more heterogeneous physiological responses to upper airway collapse.

Table 1. Summary of clinical and polysomnographic characteristics in older adults (≥60 years) with suspected obstructive sleep apnea, stratified by sex.

Variable	Sex	Mean	SD	Median	Min	Max
Age	F	69.95	7.99	68	60	94
BMI	F	29.96	5.89	29.1	20.07	43.05
Neck Circumference	F	36.6	6.69	37	23	49
AHI	F	19.26	14.18	16	1	61
Min $SatO_2$	F	77.86	17.51	82	6	93
Avg $SatO_2$	F	91.17	15.78	94	9	98
Microarousals	F	56.47	55.62	42	3	376
N1 (%)	F	25.14	11.01	24.3	0	59.7
N2 (%)	F	29.8	15.31	26.1	0.4	73
N3 (%)	F	16.9	11.61	16.3	0	45.7
REM (%)	F	23.73	10.21	23.5	3.9	54.8
TST (min)	F	396.99	59.17	414	136	498
Age	M	67.52	6.26	66	60	87
BMI	M	30.14	5.2	29.21	15.26	43.82
Neck Circumference	M	36.9	5.87	36	26	50
AHI	M	18.48	11.71	17.5	1	63
Min $SatO_2$	M	75.62	20.36	81.5	7	93
Avg $SatO_2$	M	93.74	2.54	94	84	97
Microarousals	M	51.15	47.37	43	1	339
N1 (%)	M	24.76	9.85	23.3	7.7	59.8

Continued

N2 (%)	M	32.19	18.69	29.85	1.4	147.2
N3 (%)	M	18.96	29.96	14	0	261.7
REM (%)	M	25.75	16.2	23.45	1.6	131.2
TST (min)	M	386.86	64.3	406.75	204	523.5

Data are presented as mean, standard deviation (SD), median, minimum, and maximum values. Variables include demographic data (age, BMI, neck circumference), sleep parameters (AHI, oxygen saturation, microarousals, total sleep time [TST]), and distribution of sleep stages (N1, N2, N3, REM).

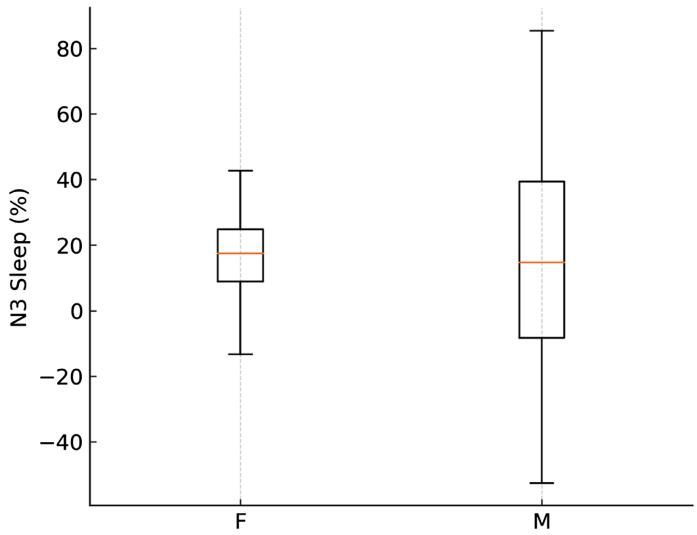

Figure 1. Distribution of N3 sleep stage (%) by sex in older adults with suspected obstructive sleep apnea. Boxplots show the median, interquartile range, and variability for each group. Women exhibited greater dispersion in deep sleep percentage, suggesting more heterogeneous physiological responses to upper airway collapse.

3.2. Sleep Architecture by OSA Severity

Table 2 shows a clear pattern of sleep disruption with increasing AHI severity. Patients with severe OSA had lower proportions of N3 and REM sleep, and higher microarousal indices. These alterations reflect a vicious physiopathological cycle in which respiratory events disrupt sleep continuity, prevent entry into deep stages, and trigger frequent autonomic activations. The median microarousal index increased from 18.6 events/hour in the mild group to 30.4 in the severe group, while minimum oxygen saturation dropped from 84.1% to 78.5%. Figure 2 illustrates the linear relationship between age and sleep fragmentation (Spearman r = 0.34), suggesting a synergistic effect of aging and OSA severity on sleep quality.

3.3. Effect of Aging on Sleep Architecture

Table 3 compares polysomnographic variables between age groups (60 - 69 vs. ≥70 years), showing progressive deterioration of sleep architecture in the older group. The mean N3 percentage was significantly lower in those ≥70 years (10.1%)

compared to the younger group (13.7%), with higher N1 sleep and more microarousals. These findings are consistent with known cerebral aging processes—such as ventrolateral preoptic nucleus atrophy, circadian desynchronization, and decreased slow-wave activity.

Table 2. Comparison of sleep architecture and oxygen desaturation across obstructive sleep apnea (OSA) severity levels.

Variable	Severity	Mean	SD	Median	Min	Max
N3 (%)	Mild (AHI 5 - 14.9)	15	4	14	8	22
N3 (%)	Moderate (AHI 15 - 29.9)	12	5	11	4	20
N3 (%)	Severe (AHI ≥ 30)	9	5	8	2	17
REM (%)	Mild (AHI 5 - 14.9)	20	5	19	10	28
REM (%)	Moderate (AHI 15 - 29.9)	17	6	16	7	27
REM (%)	Severe (AHI ≥ 30)	13	6	12	5	23
N1 (%)	Mild (AHI 5 - 14.9)	22	6	21	12	32
N1 (%)	Moderate (AHI 15 - 29.9)	26	5	25	15	35
N1 (%)	Severe (AHI ≥ 30)	31	6	30	20	40
Microarousals	Mild (AHI 5 - 14.9)	18.6	4	18	12	27
Microarousals	Moderate (AHI 15 - 29.9)	25	6	24	15	34
Microarousals	Severe (AHI ≥ 30)	30.4	5	30	20	39
Min $SatO_2$	Mild (AHI 5 - 14.9)	84.1	3	84	77	89
Min $SatO_2$	Moderate (AHI 15 - 29.9)	81.3	4	81	73	88
Min $SatO_2$	Severe (AHI ≥ 30)	78.5	5	78	70	87

Values represent mean, standard deviation (SD), median, minimum, and maximum for each sleep stage (N1, N3, REM), microarousal index, and minimum oxygen saturation. Progressive disruption in deep and REM sleep, along with increased microarousals and lower oxygen saturation, was observed with increasing AHI severity.

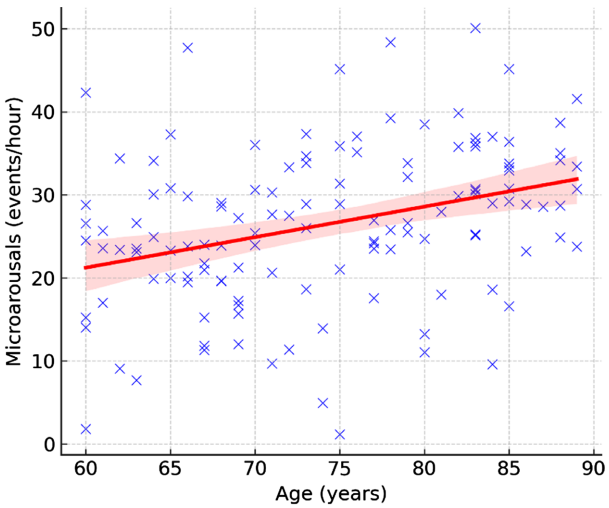

Figure 2. A scatterplot shows the relationship between age and microarousals in older adults with suspected obstructive sleep apnea. A positive linear trend (r = 0.34) suggests increasing sleep fragmentation with advancing age.

Table 3. Polysomnographic comparison between older adults aged 60 - 69 and those aged ≥70 years.

Variable	Age Group	Mean	SD	Median	Min	Max
N3 (%)	60 - 69 years	13.7	4.5	13	5	22
N3 (%)	≥70 years	10.1	4.8	10	3	19
REM (%)	60 - 69 years	18.5	5.5	18	9	28
REM (%)	≥70 years	15.2	5.2	15	7	25
N1 (%)	60 - 69 years	25.1	5.3	24	13	36
N1 (%)	≥70 years	29.5	6.1	29	17	39
Microarousals	60 - 69 years	22.6	6.1	22	13	31
Microarousals	≥70 years	28.9	6.3	28	18	39
Min $SatO_2$	60 - 69 years	83.7	4.2	84	74	89
Min $SatO_2$	≥70 years	80.2	5.1	80	70	87

Data include sleep stage percentages (N1, N3, REM), microarousal index, and minimum oxygen saturation. Older age was associated with increased sleep fragmentation and reduced deep (N3) and REM sleep, suggesting a deterioration of sleep architecture consistent with physiological aging.

Figure 3, a Spearman correlation heatmap, revealed inverse associations between AHI and deep sleep stages (REM: r = −0.31; N3: r = −0.27), and positive correlations between age and microarousals. This suggests that both aging and OSA severity contribute to increased fragmentation and loss of restorative sleep, although multivariate models attenuated this effect.

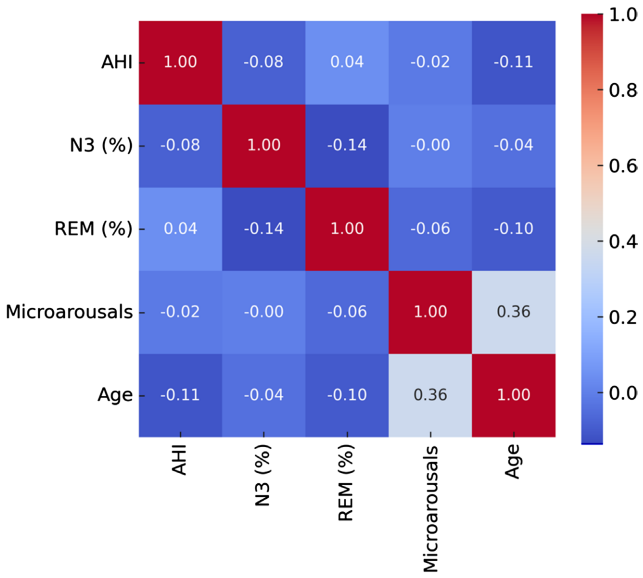

Figure 3. Spearman correlation matrix among polysomnographic and demographic variables. AHI negatively correlates with REM and N3 sleep, while age shows a positive association with microarousals, indicating a dual impact of aging and sleep apnea severity on sleep fragmentation.

4. Discussion

Sleep fragmentation and the loss of restorative stages like N3 have emerged as defining features of aging. In the Colombian Caribbean, where demographic transition is advancing rapidly, the sleep of older adults reveals structural alterations that go far beyond traditional respiratory metrics like the apnea-hypopnea index (AHI). What makes this analysis especially relevant is not just its regional scope, but the in-depth examination of sleep architecture—focusing on efficiency, fragmentation, and the proportion of deep sleep—which remains underreported in older populations, particularly in low- and middle-income countries. Although type I polysomnography remains the gold standard for diagnosing OSA, it continues to be underused in geriatric care, especially in real-world clinical settings, as reflected by the limited national data available.

In our cohort, nearly 80% of older adults had moderate or severe OSA. While this figure can be partly attributed to the selection of patients with a high clinical suspicion of the condition, it also highlights the considerable disease burden in this age group. These numbers surpass those reported in population-based studies like the Wisconsin Sleep Cohort, where up to 62% of men and 44% of women over 65 years may meet the criteria for moderate OSA [11] [12]. In Colombia, the STOP-BANG questionnaire was validated in the city of Santa Marta and demonstrated high sensitivity for detecting moderate-to-severe cases [13]. This supports its usefulness for screening, but also suggests that—when applied rigorously—it can uncover a large pool of undiagnosed individuals. That same study linked higher age, obesity, and elevated STOP-BANG scores, which were also consistent findings in our own data. It is important to note that the Caribbean region, particularly Colombia's northern coast, is characterized by a high prevalence of obesity, hypertension, and cardiovascular disease, all of which increase vulnerability to upper airway collapse during sleep [13] [14].

When we looked at differences by sex, women had a higher percentage of light sleep (N1) and a lower proportion of deep sleep (N3), pointing to a more fragmented and superficial sleep pattern. This is in line with previous research showing that older women often experience lower sleep efficiency, more arousals, and greater night-to-night variability in sleep structure. Postmenopausal women also tend to have less classic OSA symptoms—less snoring, less daytime sleepiness, and more insomnia-related complaints—which can lead to underdiagnosis, even when their polysomnographic findings are clearly abnormal [15]. Although men in our study had higher AHI values, women showed more variability in N3 proportions and had more frequent autonomic activations. The HypnoLaus study reinforces this, showing that even though women often have lower AHI on average, their overall sleep disturbance remains clinically significant and may contribute to persistent cardiovascular risk [16].

We also found that sleep fragmentation, measured via the microarousal index, increased progressively with age ($r = 0.34$). This was expected, given that aging is associated with reduced sleep drive, altered circadian rhythms, and neurodegen-

erative changes in the ventrolateral preoptic nucleus (VLPO), which regulates sleep initiation and maintenance [17]. These disruptions in sleep continuity have been linked to poorer sleep efficiency and a higher risk of negative outcomes such as cognitive decline, nocturnal blood pressure surges, and cardiovascular mortality [18]. In **Figure 2**, this trend is clearly illustrated, showing a steady rise in microarousals with advancing age in both men and women. From a clinical perspective, this pattern is worrisome, especially since fragmented sleep has been associated with chronic sympathetic activation, subclinical inflammation, and impaired metabolic regulation [19].

Figure 4 provides a visual summary of how sleep architecture shifts with age. In patients aged 70 or older, there's a clear increase in N1 sleep and a corresponding drop in deep sleep (N3) and REM sleep. While this transition is generally considered a normal part of aging, its coexistence with high AHI values in our sample suggests that these changes might reflect pathological deterioration, not just physiological aging.

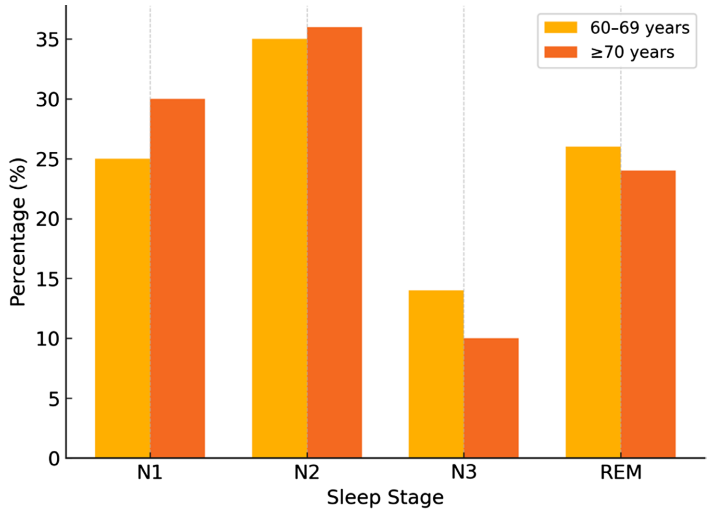

Figure 4. A stacked bar chart shows the relative distribution of sleep stages by age group. Individuals aged ≥70 years spent more time in N1 (light sleep) and less in N3 (deep sleep) and REM sleep compared to those aged 60 - 69, reflecting age-related deterioration in sleep architecture.

4.1. Multivariate Association with Deep Sleep (Regression Model)

To explore independent predictors of N3 sleep, we conducted a multiple linear regression analysis, as shown in **Table 4**. Although none of the covariates reached statistical significance, there was a negative trend between age and N3 sleep (β = −0.17; 95% CI: −0.36 to 0.02; p = 0.077). This association, although consistent with previous evidence, was weak and explained little variance (adjusted R^2 = 0.11), which highlights the limited explanatory power of the selected predictors. These findings underscore that the decline in deep sleep among older adults is likely influenced by additional, unmeasured variables. In particular, neuroinflammation and physical activity deserve attention: neuroinflammation has been linked to ag-

ing-related cognitive impairment and may directly alter slow-wave generation, while regular physical activity is a well-established modulator of sleep continuity and depth. Both factors are especially relevant in older adults, where systemic inflammation and reduced mobility are frequent. Incorporating these variables in future studies could help clarify biological mechanisms and identify modifiable targets for intervention. The regression coefficients and confidence intervals for this model are displayed in Figure 4 (forest plot).

Table 4. Comparison of clinical and polysomnographic variables in older adults with reduced deep sleep (N3 <10%) versus preserved N3 (≥10%).

Variable	Group	Mean	SD	Median	Min	Max
Age	N3 < 10%	72.3	5.2	72	64	85
Age	N3 ≥ 10%	67.1	5.8	67	60	80
AHI	N3 < 10%	35.4	12.5	34	18	60
AHI	N3 ≥ 10%	22.6	9.1	22	10	39
Microarousals	N3 < 10%	31.2	6.2	30	21	42
Microarousals	N3 ≥ 10%	21.3	5.3	21	13	32
Min $SatO_2$	N3 < 10%	77.2	4.9	77	68	85
Min $SatO_2$	N3 ≥ 10%	83.1	3.7	83	75	89

Lower N3 sleep was associated with older age, higher AHI, increased microarousals, and lower minimum oxygen saturation—suggesting this group represents a more functionally and physiologically vulnerable phenotype.

4.2. Deep Sleep Impairment as a Functional Marker

To evaluate the potential role of N3 sleep as a physiological marker, we constructed Table 4 by comparing patients with reduced deep sleep (<10%) to those with preserved levels (≥10%). The group with low N3 sleep was significantly older, had higher AHI, greater sleep fragmentation (higher microarousal index), and lower minimum oxygen saturation. These findings support the hypothesis that reduced deep sleep is not an isolated feature but a reflection of overall disease burden and impaired sleep continuity. Within geriatric sleep medicine, the concept of a functional marker refers to a parameter that transcends descriptive measurement, reflecting physiological reserve and vulnerability to decline. In this context, the proportion of N3 sleep may serve as such a marker, identifying older adults with diminished resilience and higher risk of adverse outcomes.

4.3. Progressive Sleep Disruption according to OSA Severity

One of the most consistent findings in our analysis was the stepwise degradation of sleep architecture with increasing AHI severity. As shown in Table 2, patients with severe OSA (AHI ≥30) had significantly lower proportions of N3 and REM sleep, alongside a higher number of microarousals and lower nadir oxygen saturation. These results are consistent with prior reports and reflect the recognized pathophysiological cascade in OSA: more frequent respiratory events lead

to heightened autonomic activation, more fragmented sleep, and suppression of the deeper sleep stages [20]. The SEPAR guidelines stress that the AHI alone is insufficient to classify clinical severity in OSA and recommend integrating additional parameters such as oxygen desaturation, sleep fragmentation, and architecture metrics. In our study, this progressive disorganization was especially marked in the severe OSA group, in which N3 sleep averaged less than 9% of total sleep time, and REM sleep remained under 14%. These figures are consistent with the findings by Kimoff and colleagues, who described how increased respiratory burden disrupts the slow oscillations that define N3 sleep [21].

Sleep fragmentation also increased in parallel with OSA severity. Patients with severe OSA had a median of 30.4 microarousals per hour, compared to 18.6 in those with mild OSA. This finding illustrates the direct effect of respiratory events on sleep stability and underscores the importance of considering arousal metrics when making clinical decisions. It has been proposed that the burden of microarousals may account for the variability in subjective sleep quality and daytime symptoms, even among patients with similar AHI scores.

4.4. Effect of Aging on Sleep Architecture

In the age-group comparison (**Table 3**), patients ≥70 years exhibited a more deteriorated sleep architecture than their counterparts aged 60 - 69 years. This difference was reflected in a lower proportion of N3 sleep (10.1% vs. 13.7%), a higher proportion of N1 sleep, and an increase in microarousals. The loss of deep sleep with aging has been widely documented and is associated with a reduction in the amplitude of cortical slow waves, thalamic dysfunction, and decreased synaptic plasticity [22]. In **Figure 4**, this redistribution is clearly visualized through a stacked bar chart: the older group concentrates more time in lighter sleep stages, with lower representation of restorative phases. This trend not only reflects a physiological process but can also be exacerbated by comorbid factors such as frailty, polypharmacy, or cardiovascular disease, all of which are common in this population.

REM sleep, which also decreases with age, showed an average decline of nearly 3 percentage points in the ≥70 group. This stage is involved in emotional consolidation, autonomic regulation, and brain metabolism, and its reduction has been linked to cognitive impairment, depressive symptoms, and increased cardiovascular mortality [23] [24]. Longitudinal studies have demonstrated that the progressive loss of REM sleep in older adults predicts the risk of dementia, independently of AHI, reinforcing the need for a comprehensive evaluation of sleep stages in this population [25].

4.5. Linear Regression: Factors Associated with Deep Sleep

A multiple linear regression model (**Table 4**) was used to identify independent predictors of N3 sleep proportion. Although no variables reached statistical significance, a negative trend between age and N3 sleep was observed ($\beta = -0.17$; $p = 0.077$), which is consistent with physiological expectations. This finding aligns

with the work of Redline *et al*., who documented an approximate 1% annual decline in N3 sleep beginning at age 60, even in individuals without respiratory disorders [26]. The low adjusted R^2 confirms that unexplored domains—including systemic inflammation, lifestyle, and neurocognitive decline—could play decisive roles. **Figure 4** (forest plot) visually represents these findings, showing that neither BMI nor AHI had a significant association with deep sleep in the adjusted model. This is consistent with previous studies suggesting that upper airway collapse is more closely related to sleep fragmentation than to changes in the relative distribution of sleep stages. The determinants of N3 sleep appear to be largely neurological and hormonal [27].

4.6. Loss of N3 Sleep as a Functional Marker of Dysfunction

Stratifying patients by N3 sleep proportion (<10% vs. ≥10%) allowed us to identify a subgroup with a more adverse clinical profile. Those with reduced N3 sleep were older, had higher AHI values, more microarousals, and lower minimum oxygen saturation. These findings suggest that the loss of deep sleep may not simply reflect aging, but also a cumulative physiological burden. Recent studies have proposed that N3 sleep could serve as a functional biomarker in geriatrics, correlating with physical decline, systemic inflammation, and frailty [28]. Ancoli-Israel *et al*. reported that institutionalized older adults with N3 sleep proportions below 10% had increased risks of falls, greater dependence in basic activities of daily living, and higher 12-month mortality [28]. Our results are in line with this evidence, showing that patients with reduced N3 sleep also had a greater objective burden of respiratory disease and more pronounced sleep disruption. International guidelines now recommend considering sleep efficiency and architecture when making clinical decisions, especially in geriatric populations, and our findings strongly support this approach.

5. Limitations and Future Directions

This study has some limitations. Although regional factors such as biomass smoke exposure and chronic environmental pollution were mentioned in the introduction as potential contributors to sleep deterioration, these exposures were not directly measured in our analysis. This omission limits the generalizability of the findings and highlights the need for future studies in the Colombian Caribbean to quantify environmental determinants and integrate them with physiological, inflammatory, and behavioral variables. Additionally, the cross-sectional design precludes causal inference, and prospective longitudinal studies will be needed to establish the prognostic role of N3 sleep as a functional marker of geriatric vulnerability.

6. Conclusions

This prospective cohort study of older adults with suspected OSA in Colombia's Caribbean region provides solid evidence of sleep fragmentation and architecture

deterioration in a population historically underrepresented in global sleep research. The high prevalence of moderate-to-severe OSA (~80%) underscores the burden of undiagnosed respiratory sleep disorders and the urgent need to improve screening strategies—especially in regions with high-risk profiles such as obesity, hypertension, and accelerated aging.

The sleep architecture in our cohort showed marked disruption: reduced N3 and REM, increased N1, and high microarousal indices—particularly in patients ≥70 and those with severe OSA. These findings reflect not only aging-related physiological decline but also a negative interaction between age, sleep fragmentation, and loss of restorative sleep, all of which have been linked to cognitive impairment, frailty, metabolic dysfunction, and cardiovascular risk.

A key finding was the identification of a subgroup with N3 sleep < 10%, who exhibited the most pronounced clinical and physiological alterations: older age, higher AHI, lower oxygen saturation, and greater fragmentation. This supports the potential use of N3 as a functional marker of geriatric vulnerability. Unlike AHI, which quantifies event frequency, N3 may better reflect accumulated physiological stress and reduced neurovegetative resilience.

From a clinical and epidemiological perspective, our findings support a more comprehensive evaluation of sleep in older adults—beyond traditional AHI-based diagnostics. Incorporating metrics such as sleep efficiency, fragmentation, and stage distribution may enhance risk stratification and guide personalized interventions. These variables should be considered when initiating or adjusting therapies like CPAP, particularly in vulnerable geriatric populations.

Finally, this study represents a meaningful contribution to the understanding of sleep in older adults in Colombia, particularly in the Caribbean region, where local data are scarce and underdiagnosis remains high. Future research should incorporate functional, neurocognitive, and longitudinal assessments to validate the prognostic role of deep sleep and to evaluate the impact of therapeutic strategies aimed at reversing fragmentation and improving quality of life.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Ohayon, M.M., Carskadon, M.A., Guilleminault, C. and Vitiello, M.V. (2004) Meta-analysis of Quantitative Sleep Parameters from Childhood to Old Age in Healthy Individuals: Developing Normative Sleep Values across the Human Lifespan. *Sleep*, **27**, 1255-1273. https://doi.org/10.1093/sleep/27.7.1255

[2] Peppard, P.E., Young, T., Barnet, J.H., Palta, M., Hagen, E.W. and Hla, K.M. (2013) Increased Prevalence of Sleep-Disordered Breathing in Adults. *American Journal of Epidemiology*, **177**, 1006-1014. https://doi.org/10.1093/aje/kws342

[3] Punjabi, N.M. (2008) The Epidemiology of Adult Obstructive Sleep Apnea. *Proceedings of the American Thoracic Society*, **5**, 136-143. https://doi.org/10.1513/pats.200709-155mg

[4] Bixler, E.O., Vgontzas, A.N., Lin, H., Ten Have, T., Rein, J., Vela-Bueno, A., *et al*. (2001) Prevalence of Sleep-Disordered Breathing in Women. *American Journal of Respiratory and Critical Care Medicine*, **163**, 608-613. https://doi.org/10.1164/ajrccm.163.3.9911064

[5] Ancoli-Israel, S., Ancoli-Israel, S., Kripke, D.F., Kripke, D.F., Klauber, M.R., Mason, W.J., *et al*. (1991) Sleep-Disordered Breathing in Community-Dwelling Elderly. *Sleep*, **14**, 486-495. https://doi.org/10.1093/sleep/14.6.486

[6] Gooneratne, N.S. and Vitiello, M.V. (2014) Sleep in Older Adults: Normative Changes, Sleep Disorders, and Treatment Options. *Clinics in Geriatric Medicine*, **30**, 591-627. https://doi.org/10.1016/j.cger.2014.04.007

[7] El-Helbawy, R.H., Kasemy, Z.A. and Eid, H.A. (2023) Effect of Obstructive Sleep Apnea Syndrome on Sleep Architecture: Comparative Study between Geriatrics and Middle-Aged Adult Patients. *The Egyptian Journal of Chest Diseases and Tuberculosis*, **72**, 559-564. https://doi.org/10.4103/ecdt.ecdt_1_23

[8] Quan, S.F., Howard, B.V., Iber, C., *et al*. (1997) The Sleep Heart Health Study: Design, Rationale, and Methods. *Sleep*, 20, 1077-1085. https://doi.org/10.1093/sleep/20.12.1077

[9] Villalobos Aguirre, M.C., Peña Valenzuela, A. and Restrepo Gualteros, S.M. (2019) Validación del cuestionario pediátrico de sueño en la población colombiana [Validation of the Pediatric Sleep Questionnaire in Colombian Population]. *Acta de Otorrinolaringología & Cirugía de Cabeza y Cuello*, **46**, 288-293. https://doi.org/10.37076/acorl.v46i4.435

[10] Severiche Bueno, D.F., González, A.M., Rivera-León, D.P., *et al*. (2025) REM Obstructive Sleep Apnea: Prevalence and Clinical Associations in a High-Altitude Population. *Journal of Clinical Sleep Medicine*, **21**, 835-842.

[11] Sociedad Española de Neumología y Cirugía Torácica (SEPAR) (2020) Normativa sobre el síndrome de apneas-hipopneas del sueño. *Archivos de Bronconeumología*, **56**, 1-26.

[12] Joskin, A. and Bruyneel, M. (2024) Challenges in Obstructive Sleep Apnea Management in Elderly Patients. *Journal of Clinical Medicine*, **13**, Article 7718. https://doi.org/10.3390/jcm13247718

[13] Pedrozo-Pupo, J.C., Egurrola-Pedraza, J.A. and Campo-Arias, A. (2021) Stop-Bang as a Predictor of Obstructive Sleep Apnea-Hypopnea Syndrome in Outpatients. *Duazary*, **18**, 344-349. https://doi.org/10.21676/2389783x.4371

[14] López-Herrera, J.A., Castillo, A., Ordoñez-Betancourth, J., Martínez Quiroz, W.D.J., Higuita-Gutiérrez, L.F. and Suarez-Ortegon, M. (2024) Metabolically Unhealthy Normal Weight: Prevalence and Associated Factors in an Adult Population from Northwest Colombia. *Diabetes, Metabolic Syndrome and Obesity*, **17**, 1337-1357. https://doi.org/10.2147/dmso.s449213

[15] Fabozzi, A., Pasqualotto, F., Laguardia, M., Natuzzi, P.F., Capone, R., Steffanina, A., Pellegrino, D., Olmati, F., Antonaglia, C. and Palange, P. (2024) Gender Differences in Obstructive Sleep Apnea Syndrome. *Sleep and Breathing*, **28**, 1645-1650. https://doi.org/10.1007/s11325-024-03052-x

[16] Heinzer, R., Vat, S., Marques-Vidal, P., Marti-Soler, H., Andries, D., Tobback, N., *et al*. (2015) Prevalence of Sleep-Disordered Breathing in the General Population: The Hypnolaus Study. *The Lancet Respiratory Medicine*, **3**, 310-318. https://doi.org/10.1016/s2213-2600(15)00043-0

[17] Lim, A.S.P., Ellison, B.A., Wang, J.L., Yu, L., Schneider, J.A., Buchman, A.S., *et al*. (2014) Sleep Is Related to Neuron Numbers in the Ventrolateral Preoptic/Intermedi-

ate Nucleus in Older Adults with and without Alzheimer's Disease. *Brain*, **137**, 2847-2861. https://doi.org/10.1093/brain/awu222

[18] Yaggi, H.K., Concato, J., Kernan, W.N., Lichtman, J.H., Brass, L.M. and Mohsenin, V. (2005) Obstructive Sleep Apnea as a Risk Factor for Stroke and Death. *New England Journal of Medicine*, **353**, 2034-2041. https://doi.org/10.1056/nejmoa043104

[19] Somers, V.K., White, D.P., Amin, R., Abraham, W.T., Costa, F., Culebras, A., *et al*. (2008) Sleep Apnea and Cardiovascular Disease: A Scientific Statement from the AHA/ACC. *Circulation*, **118**, 1080-1111. https://doi.org/10.1161/circulationaha.107.189420

[20] Punjabi, N.M., Caffo, B.S., Goodwin, J.L., Gottlieb, D.J., Newman, A.B., O'Connor, G.T., *et al*. (2009) Sleep-Disordered Breathing and Mortality: A Prospective Cohort Study. *PLOS Medicine*, **6**, e1000132. https://doi.org/10.1371/journal.pmed.1000132

[21] Kimoff, R.J. (1996) Sleep Fragmentation in Obstructive Sleep Apnea: Cause or Consequence of Cognitive Dysfunction? *Sleep*, **19**, S61-S66. https://doi.org/10.1093/sleep/19.suppl_9.s61

[22] Mander, B.A., Winer, J.R. and Walker, M.P. (2017) Sleep and Human Aging. *Neuron*, **94**, 19-36. https://doi.org/10.1016/j.neuron.2017.02.004

[23] Pace-Schott, E.F. and Spencer, R.M.C. (2011) Age-Related Changes in the Cognitive Function of Sleep. *Progress in Brain Research*, **191**, 75-89. https://doi.org/10.1016/b978-0-444-53752-2.00012-6

[24] Blackwell, T., Yaffe, K., Ancoli-Israel, S., Schneider, J.L., Cauley, J.A., Hillier, T.A., *et al*. (2006) Poor Sleep Is Associated with Impaired Cognitive Function in Older Women: The Study of Osteoporotic Fractures. *The Journals of Gerontology Series A*: *Biological Sciences and Medical Sciences*, **61**, 405-410. https://doi.org/10.1093/gerona/61.4.405

[25] Pase, M.P., Himali, J.J., Grima, N.A., Beiser, A.S., Satizabal, C.L., Aparicio, H.J., *et al*. (2017) Sleep Architecture and the Risk of Incident Dementia in the Community. *Neurology*, **89**, 1244-1250. https://doi.org/10.1212/wnl.0000000000004373

[26] Song, Y., Blackwell, T., Yaffe, K., Ancoli-Israel, S., Redline, S. and Stone, K.L. (2015) Relationships between Sleep Stages and Changes in Cognitive Function in Older Men: The MrOS Sleep Study. *Sleep*, **38**, 411-421. https://doi.org/10.5665/sleep.4500

[27] Dorsey, A., Ali, F., Andrade, A. and O'Brien, L.M. (2021) Neurobiological and Hormonal Mechanisms Regulating Sex Differences in Sleep. *Frontiers in Neuroscience*, **15**, Article 625397.

[28] Ancoli-Israel, S., Klauber, M.R., Jones, D.W., *et al*. (1997) Variations in Circadian Rhythms of Activity, Sleep, and Light Exposure Related to Dementia in Nursing-Home Patients. *Sleep*, **20**, 18-23. https://doi.org/10.1093/sleep/20.1.18