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Age-related changes in sleep architecture: Effects of body mass index, sex, and mental health in community-dwelling adults using at-home polysomnography

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ABSTRACT

Background: Sleep is a critical component of human health, influencing cognitive, physical, and psychological well-being. Sleep architecture changes significantly with age. Odds Ratio Product (ORP) is a novel continuous index of sleep depth, which provides insights into age-related changes in sleep depth.
Objectives: This study investigates the interaction of sex, body mass index (BMI) and mental on age-related changes in sleep architecture, measured with ORP in community-dwelling adults using at-home polysomnography (PSG).
Methods: We measured sleep architecture using ORP in 195 middle-aged to older (>50 years) sedentary but healthy adults and a younger cohort (<35 years, n = 34). All sleep nights utilized MY scoring (Cerebra Medical, Winnipeg Manitoba) to calculate traditional sleep architecture values, average ORP in different sleep stages and percent of time in different ORP deciles. Differences between groups were assessed using ANOVA models. Mental health was assessed using standardized questionnaires linear regression models were used to report the relationship between age and sleep architecture, ORP, and interactions between sex, BMI, anxiety and depression.
Results: Significant interactions were present for body mass index, deep sleep deciles, and the awake stage.

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Conclusions: Confirming previous findings, we report reduced deep sleep, increased light sleep and more full wakefulness in middle-aged to older adults, consistent with decreased sleep pressure with aging. Higher body mass index was associated with reduced age-related changes in sleep architecture.

1. Introduction

Age-related changes in sleep architecture, as measured by the Rechtschaffen and Kales (R&K) criteria, have been documented extensively in the past [1–4].These include earlier bed time, rise time, delayed sleep onset, decreased total sleep time, increased wake time, more sleep fragmentation, lower arousal threshold, less time in slow wave sleep (SWS), and decreased EEG delta power [1]. The mechanisms of these age-related changes have been debated and include circadian rhythm changes, increased comorbidities and medication use in older people, age-related decrease in delta wave amplitude, among others [1,2]. Given the association of poor sleep quality with multiple health outcomes [5–7], identification of the mechanism(s) of these age-related changes in sleep quality is desirable.

Apart from the earlier bed and rise times, all the reported changes above are consistent with age-related decrease in sleep pressure with a consequent decrease in sleep depth across the night. Differences in sleep depth are conventionally evaluated from the percent of time spent in SWS, EEG delta power, and changes in arousal threshold. Time in SWS and delta power are highly dependent on the number, duration and amplitude of delta waves. Recent work has shown that delta waves are special events that occur in deep sleep, but their numbers or amplitude do not correlate with sleep depth when the latter is assessed from likelihood of spontaneous arousals [8]. The same study [8] also showed that sleep becomes progressively deeper during stage 2, reaching SWS levels even in the absence of delta waves. In fact, most of the changes in sleep depth occur before SWS is reached [8]. Time in SWS also does not reveal level of vigilance during awake stage, which is an important marker of sleep pressure [9–11]. Likewise, EEG delta power is of little value as a continuous measure of sleep depth as it is exquisitely sensitive to delta waves [8]. Serious limitations have also been raised [8] to the use of noise stimuli for measuring arousal threshold, as used to document age-related decrease in arousal threshold [12]. These, and the limitations on the use of time in SWS and EEG delta power as continuous measures of sleep depth make it clear that reduction in overall sleep depth with age has not been clearly demonstrated in previous studies that utilized conventional sleep scoring.

The odds ratio product (ORP) is a validated continuous metric of sleep depth and sleep propensity calculated from the relationship of EEG powers in different frequencies to each other [8,13–15]. It is measured in consecutive 3-s epochs throughout the night. It is reported in a variety of graphical and numerical ways including in 3-s epochs to determine the dynamic behavior of sleep depth in different situations (e.g., in response to noise) [16] following arousal [17], as average values in 30-s epochs [9,14], as average values in the entire night or in different sleep stages, where it distinguishes different levels of vigilance in stage wake (ORP 1.75–2.50) and different levels of sleep depth (.00–1.75) in the same sleep stage [9,14], or as percent of total recording time in different ORP deciles (ORP-architecture) [10]. Differences in sleep pressure result in changes in deep sleep (Time in ORP$<$0.50, deciles 1 and 2) and time in full wakefulness (time in ORP$>$2.25; decile 10). Thus, sleep deprivation is followed by a leftward shift in the histogram with more time in deciles 1 and 2 and less time in decile 10 [10], while reduction in sleep drive (e.g., second half vs. first half of the night, or presence of hyperarousal) results in a rightward shift with opposite changes. By contrast, disorders that fragment sleep are associated with reduction in both deep sleep and full wakefulness while circadian disorders result in increases in both deep sleep and full wakefulness deciles, and so on [10].

In an earlier study on a large community-based cohort (Sleep Heart Health Study (SHHS), n = 5781) ORP in stages wake, NREM sleep, REM sleep, and TRT was reported to increase progressively from participants aged 40–55 to participants aged 75–90 years [9]. In a subsequent analysis that used the ORP architecture approach (deciles histogram) % of time in deep sleep (ORP$<$0.50) in participants with no OSA or insomnia (n = 1704) decreased progressively from age 18–39 to $>$70 years, while % of time in full wakefulness (ORP$>$2.25; Decile 10) increased over the same age range [10].

Whether the age-related changes in sleep depth are affected by BMI, gender or mental health is unclear. A large body of literaure has explored the effects of BMI on sleep architecture [18–24], however the literature examining effects of BMI on ORP architecture is limited. A prior ORP-based analysis found no association between BMI and ORP deciles [10], but did not examine this relationship across age. The effects of sex on age-related changes in ORP are unclear, though prior work suggests there are sex differences in ORP deciles with deep sleep and drowsy-wake properties, independent of age [10]. Anxiety and depression are associated with reduced time spent in traditional stage 3 and REM sleep [25,26]. However, the relationship between these conditions and ORP remains unexplored, with research limited to youth with psychiatric or learning disorders [27].

The objectives of the current study are to confirm results of earlier studies that used ORP to report changes in ORP architecture with age. We also aim to focus on changes in sleep depth, both by presenting ORP architecture and ORP changes within stages. Lastely we examine interactions between age-related changes in sleeep depth with BMI, sex and self-reported mental health.

2. Methods

2.1. Participants

Study participants were drawn from a distinct group of younger research subjects [28] and two studies, Brain in Motion I (BIM I) and Brain in Motion II (BIM II) that recruited middle-aged to older adults [29,30]. All participants resided in the community and underwent at-home polysomnography. Participants were recruited from the city of Calgary, Alberta through printed posters and newspaper advertisements. The noted studies were all approved by the University of Calgary Institutional Conjoint Health Research Ethics Board (REB16-1027, REB14-2284 and REB16-1199) [28–30]. A total of two hundred and twenty-nine participants were then divided into two age groups: younger adults (18–35 years) and middle-aged to older adults (50 years and over). The age range criteria for inclusion of participants in the study of younger adults [28] was 18–35 years and over 50 years for middle-aged to older adults [29,30].

2.2. Eligibility criteria

The inclusion criteria for the younger group included: residing in Calgary $>$1 year and no prior diagnosis of obstructive sleep apnea (OSA), no current use of medications affecting vascular function, and no history of cardiorespiratory disease, hypertension, or smoking history [28].

The eligibility criteria for BIM I included: being 55–80 years of age, sedentary, having a BMI of less than 35 kg/m^2, no diagnosis of an active cardiovascular or cerebrovascular disease, no obstructive airway disease that would preclude the ability to safely exercise, being a non-smoker for at least 12 months, no major surgery or trauma in the past six months, no debilitating neurological disorders (e.g., Multiple Sclerosis or Alzheimer disease), and a Montreal Cognitive Assessment (MoCA) score of at least

24 points [29,31].

The eligibility criteria for the BIM II randomized controlled trial (RCT) included: sedentary adults (age 50–80 years) who had one or more cardiovascular disease risk factors, a family history of Alzheimer disease and related dementias (ADRD), or no dementia but subjective cognitive symptoms. Exclusion criteria included dementia or severe cognitive deficits, absence of cardiovascular disease risk factors or a history of ADRD, severe asthma, developmental disability, and terminal illness with a life expectancy of less than a year, history of stroke or serious cardiovascular condition, participation in another clinical trial, and coexisting medical or neurological conditions. Participants were required to provide informed consent, and medical clearance was requested from their family physician to participate in the study. The Physical Activity Readiness Questionnaire (PAR-Q+) was completed by all participants prior to exercise testing [30].

BIM I and BIM II shared several common inclusion criteria including being sedentary, free from cardiovascular/cerebrovascular disease which would preclude the participants' ability to exercise, MoCA score >24, postmenopausal status for women (i.e., no menses within the 12 months prior to enrolling in the study) and being over the age of 50. The sample characteristics of younger and middle-aged to older group is presented in Table 1. For increased clarity, and to provide justification for combining BIM-I and BIM-II into one group, the sample characteristics of the middle-aged to older group are separated by study (BIM-I and BIM-II) in Table S1 and sleep characteristics in Table S2.

2.3. Study design

This cross-sectional study used level 2 at-home PSG. Data were taken from each participant's first PSG session during the study period and no participants were used as duplicate data points. Only participants in BIM I and BIM II completed the Centres for Epidemiological Studies Depression Scale (CES-D) and Beck Anxiety Inventory (BAI) [30].

2.4. Measurements

2.4.1. Objective measures of sleep

Polysomnographic data were collected using two PSG systems: Embletta MPR (Natus Medical Inc., Pleasanton, CA) and Prodigy 2 (© 2022 Cerebra). For the Embletta system, a sleep technician set up the equipment in participants' homes [32]. When using the Prodigy 2 system a research associate demonstrated the instrumentation of Cerebra Sleep System to the participants (~1 h in length) and participants self instrument the equipment within 1–2 days. Brain and body activity were recorded using electroencephalogram (EEG), electrooculogram (EOG), electromyogram (EMG), electrocardiogram (ECG), and pulse oximeter. All studies utilized a semi-automated scoring system, in which sleep was initially scored automatically, with subsequent manual review and the option for minor edits by technologists (MY scoring; Cerebra Medical, Winnipeg, Manitoba) [33]. The outcomes obtained included, apnea-hypopnea index, periodic limb movements, percentage of total sleep time (TST) spent in traditional sleep stages: awake, stage 1, stage 2, stage 3, REM, and percentage of total recording time (TRT) in NREM or awake. In addition, the PSG analyses also included the Odds Ratio Product (ORP, see below). ORP can be reported as an average value within a given sleep stage, as a percentage of TRT spent in a given decile. The calculation of ORP has been reported in-depth previously, a brief description is provided below (*see Odds-Ratio Product)* [15].

2.4.2. Body mass Index and sex

BMI was acquired during anthropometric assessments and was calculated using weight (kg)/height $(m)^2$. Sex was collected as self-reported sex assigned at birth.

2.4.3. Mental health questionnaires

The CES-D was only utilized in BIM I and BIM II. The CES-D measures the frequency of depressive symptoms with a score ranging from 0 to 60. A score above 15 suggests the possible presence of depression, with higher scores indicating greater symptom severity [34]. Self-report anxiety symptoms were assessed using the Beck Anxiety Inventory (BAI) in BIM I and BIM II cohorts. The BAI evaluates the severity of common anxiety symptoms experienced over the past week. Scores above 21 suggest the possible presence of clinically relevant anxiety and a higher total score means more severe anxiety symptoms. No mental health questionnaires were collected in the younger sample.

2.5. Odds-Ratio Product

Calculation of ORP has been reported in-depth previously [15]. Briefly, Fast Fourier Transform is applied to each non-overlapping 3-s epoch. Total power is calculated within 4 frequency ranges: delta (.3–2.3 Hz), theta (2.7–6.3 Hz), alpha-sigma (7.3–14.0 Hz), and beta (14.3–35.0 Hz). The power in each frequency is assigned a rank (0–9) based on its location within the range of powers observed in 56 clinical reference polysomnograms [14,15]. The 4 ranks are concatenated in a 4-digit number, from left to right relative to each other, producing 10 000 different bin numbers (0000–9999). The probability of each bin number occurring in epochs scored wake, or during arousals is determined from a look-up table based on manual scoring of the same 56 clinical polysomnograms. This probability (0–100 %) is divided by 40 (% of epochs in development files scored as awake), converting the range to an ORP range of 0–2.5 where 0 refers to a pattern that never occurs during wake epochs or arousals while 2.5 refers to patterns that are never seen during sleep [15]. Each PSG contains 800–1000 30-s epochs. From these epochs, an equal number of ORP values spanning the entire range (0–2.5) are generated. Here we report average ORP values in different sleep stages and percent of recording time spent in each ORP decile. ORP deciles provide a useful way to identify the likely mechanism of differences in ORP-based sleep architecture [10] see (Introduction and Supplement). Deciles 1 and 2 (ORP<0.50) represent very deep and deep sleep, respectively. Decile 3 (ORP .50–.75) indicates moderately deep sleep, while decile 4 (ORP .75–1.00) signifies light sleep. Deciles 5 to 7 (ORP 1.00–1.75) are transitional states with increasing features of wakefulness. Deciles 8 and 9 (ORP 1.75–2.25) correspond to periods generally scored as wake but with some sleep features (drowsy awake), and decile 10 (ORP 2.25–2.50) represents full wakefulness [10,15].

2.6. Statistical analysis

Participant characteristics were compared using one-way ANOVA. Levene's test for equality of variances assessed the assumption of homogeneity. One-way analyses of variance (ANOVA) were conducted to compare sleep variables across groups. ANOVA was used instead of multiple t-tests to manage the increased risk of Type I error. Tukey's Honest Significant Difference (HSD) was used to control for multiple comparisons.

Linear regression analyses were conducted to examine the effects of age on sleep architecture and to explore potential interaction effects between age, sex, BMI and mental health variables (CES-D, BAI) on sleep depth. A set of hierarchical regression models was performed. In the first step, age was entered as the sole predictor of sleep architecture. In the second step, AHI and sex were added to assess whether they explained additional variance in sleep outcomes beyond age. To examine moderation effects, additional linear regression models were conducted, including interaction terms between age and each predictor variable (sex, BMI, CES-D, BAI). Individual deciles and traditional sleep stages were entered as dependent variables. Each model included age, AHI, and a moderator variable along with an interaction term between age and the moderator. To visualize the interaction between age and BMI, BMI was stratified using the sample mean and SD (27.6 ± 4.9). This resulted in three groups, low BMI (<22.7 kg/m^2), average BMI (22.7–32.5 kg/

m^2), and high BMI (>32.5 kg/m^2).

All analyses were performed using SPSS software (IBM SPSS Statistics for Windows, Version 29.0.2.0; Armonk, NY: IBM Corp). Statistical significance for all tests was set at $p < .05$. To visually assess the normality of residuals, Q-Q plots, a histogram with a normal distribution curve, and homoscedasticity of residuals were inspected. An example of plots used for inspection is presented in the supplemental material. All models met the necessary assumptions to an acceptable degree.

3. Results

3.1. Participants

The younger (18–35 years) sample consisted of 34 adults with a mean age (SD) of 26.3 (3.8) years (19 [55.8 %] were female). The middle-aged to older (>50 years) sample consisted of 74 participants from the BIM I study (mean (SD) age of 68.5 (5.9) years; 34 [45.9 %] female) and 121 participants from the BIM II study (mean age (SD) 63.6 (6.1) years; 78 [64.4 %] female) [28–30]. The younger sample was collected with the Embletta sleep system (100 %), whereas in the middle-aged to older sample, 65 were collected using the Prodigy 2 sleep system (33.3 %) and 130 were collected using the Embletta sleep system (66.6 %). All 195 middle-aged to older participants were included in an interaction analysis between BMI, sex and age. Of those, 135 (n = 42 BIM I, 93 BIM II) middle-aged to older participants completed the BAI and were included in the interaction analysis between BAI and age. 136 (n = 41 BIM I, 95 BIM II) middle-aged to older participants completed the CES-D and were included in the interaction analysis involving CES-D and age. Fig. 1 displays the flow chart for sample sizes included in each analysis.

Table 1 summarizes participant characteristics. The middle-aged to older (>50 years) adults had higher age, BMI, Waist-to-hip ratio, body fat percentage, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure, and AHI ($p < .05$). Additionally, the middle-aged to older (>50 years) group had lower VO_2peak ($p < .05$).

Table S1 summarizes the individual characteristics of the middle-aged to older cohorts which were included in the linear regression analyses. Adults from the BIM II sample had higher BMI (kg/m^2, 28.4 ± 5.5 *vs*. 26.4 ± 3.4), body fat percentage (%, 35.5 ± 7.4 *vs*. 30.8 ± 6.4), and proportion of female participants (%, 64.5 *vs*. 45.6). The BIM I sample had greater age (years, 68.5 ± 5.9 *vs*. 63.6 ± 6.1). There were no significant differences between the groups' waist-to-hip ratio (WHR), VO_2peak, systolic blood pressure (SBP), diastolic blood pressure (DBP), AHI retired status, smoking status, presence of metabolic syndrome or usage of medication that could affect sleep. Table S2 presents the sleep characteristics between middle-aged to older adult cohorts. BIM II participants had higher % of time in stage 3 (%, 11.2 ± 12.0 *vs* 4.8 ± 6.4) and time in stage 3 (minutes, 38.2 ± 42.0 *vs* 18.3 ± 22.4). BIM II participants had lower time in stage 1 (minutes, 47.6 ± 26.1 *vs* 58.6 ± 31.6), stage 2 (minutes, 192.4 ± 55.1 *vs* 222.3 ± 51.8) and lower % of time in stage 2 (%, 57.1 ± 10.8 *vs* 61.2 ± 10.6). The middle-aged to older groups did not differ in % of time in stage 1, nor in average ORP values within sleep stages or in %TRT in ORP deciles.

Table 1
Participant Characteristics of 34 (19 Female) Younger (18–35 years of age) and 195 (112 Female) Middle-Aged to Older Adults (>50 years of age).

	Younger (<35)	Older (>50)	*p-value*	η^2
Age (years)	26.3 ± 3.8	65.5 ± 6.4	<.001	.838
BMI (kg/m^2)	23.7 ± 3.2	27.6 ± 4.9	<.001	.080
WHR	.82 ± .05	.91 ± .09	<.001	.118
Body Fat %	25.6 ± 8.1	33.7 ± 7.4	<.001	.128
VO_2peak (ml/kg/min)	46.6 ± 9.2	26.7 ± 5.8	<.001	.552
SBP (mmHg)	112.5 ± 10.1	127.4 ± 18.1	<.001	.089
DBP (mmHg)	74.4 ± 8.4	75.5 ± 9.2	.015	.026
MAP (mmHg)	85.1 ± 7.6	92.8 ± 10.4	<.001	.070
AHI (events/hour)	5.4 ± 3.2	22.0 ± 18.7	<.001	.104

Notes. BMI, Body mass index; $VO_{2peak,}$ maximal rate of oxygen consumption/aerobic capacity; WHR, waist: hip ratio; SDP; systolic blood pressure, DBP; diastolic blood pressure, MAP; mean arterial pressure, AHI; apnea hypopnea index. Data are presented as Means ± SD (mean ± standard deviation), *p*-values are results from one-way ANOVA. Eta-squared (η^2) = small (.01), moderate (.06), or large (>.14) effect size.

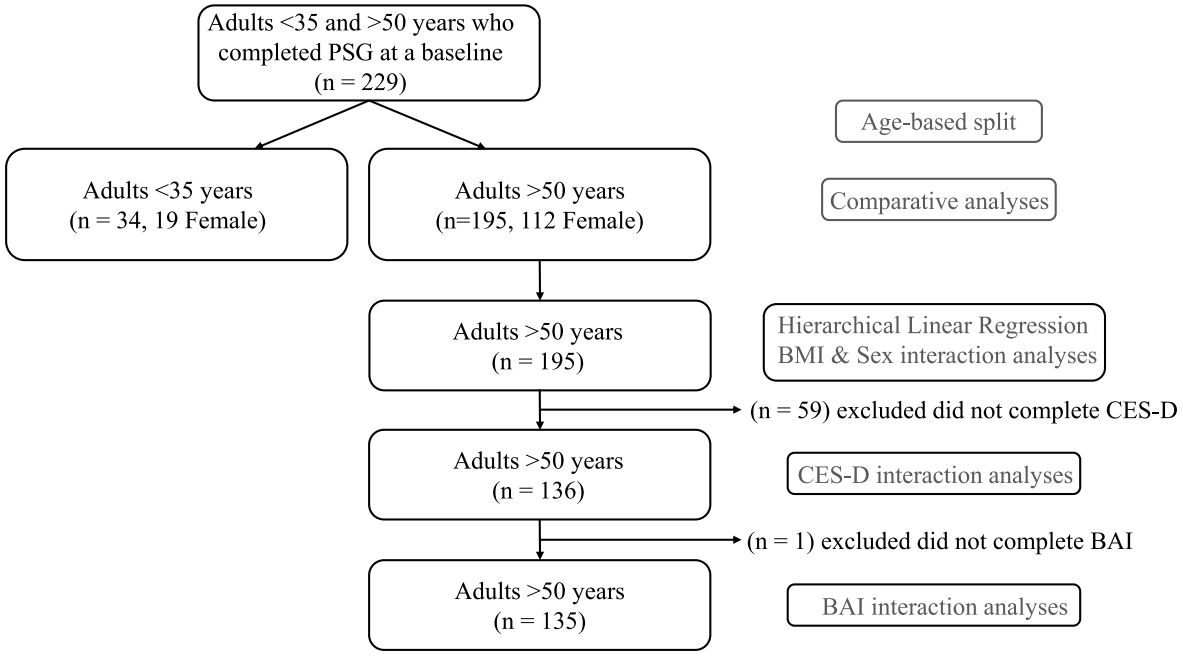

Fig. 1. Sample sizes and their allocation for each analysis. Black boxes contain information on sample type and size, as well as reason for exclusion. Grey boxes and text denote the specific analyses conducted with each corresponding sample.
Abbreviations: PSG, polysomnography; BMI, Body Mass Index; CES-D, Centres for Epidemiological Studies Depression Scale; BAI, Beck Anxiety Inventory.

3.2. Comparison of sleep architecture between younger and middle-aged to older adults

Fig. 2 shows the relationship between ORP in combined NREM sleep and in TRT vs. time in SWS. Note that N3 time can be very long (>80 min) when sleep depth in all non-REM sleep, or in total recording time, is very light (high values) or very deep, and vice versa. As shown in Table 2 and reported previously [1–4] middle-aged to older adults spent less percentage of TST in the traditional sleep stage 3 (i.e., slow-wave sleep) but more percentage of TST in stage 1, stage 2 and awake ($p < .05$). Middle-aged to older adults had higher average ORP within stage 1, stage 2, stage 3, REM, NREM, TRT, and Wake ($p < .05$). Middle-aged to older adults spent less time in stage 3 sleep ($p < .05$), and no differences were observed between time spent in stage 1, 2 or REM ($p > .05$).

Differences in ORP deciles between younger and middle-aged to older groups are summarized in Table 2 and represented in Fig. 3. The older group spent less percentage of TRT in deciles 1, 2, 3, 4 and more percentage of TRT in deciles 6, 7, 8, 9 and 10 ($p < .05$).

3.3. Regression analyses

Table S3 summarizes the linear regression analyses between age and sleep outcomes in middle-aged to older adults. Age was negatively associated with deciles 1 (β = −.142), 2 (β = −.196), 3 (β = −.122), 4 (β = −.119), 5 (β = −.098) and positively associated with deciles 7 (β = .123), 8 (β = .178), 9 (β = .131), and 10 (β = .229). No associations were found between age and decile 6.

Table S4 summarizes the results of the hierarchical regression analyses. Adding AHI and sex to the regression models statistically improved the prediction of sleep outcomes beyond age alone. Significant model improvements were observed for individual deciles 1, 2, 5, 6, and 7. Improvements were also evident in predicting traditional sleep stages, including stage 1, 2, and 3. No statistical improvements were observed for prediction of deciles 3, 4, 8, 9, 10, awake or REM. AHI emerged as a significant predictor for many sleep outcomes. Sex, however, was a significant predictor only for decile 1, 7, stage 1, and stage 3 (Table S4).

3.4. BMI interactions on age-related changes in sleep

Table S5 presents the results of the regression model used to assess the potential interaction between age and BMI on sleep architecture. The interaction was significantly and positively associated with decile 1 (β = .027, Fig. 5) and decile 2 (β = .042, Fig. 5) and negatively associated with decile 5 (β = −.024).AHI was a significant predictor of deciles 2, 6, stage 1 and stage 2.

3.5. Sex interactions on age-related changes in sleep

Table S6 presents the results of the regression model used to assess the interaction between age and sex on sleep architecture. No significant interactions were observed for ORP-based architecture or traditional sleep stages. AHI was a significant predictor of deciles 2, 4, 5, 6 and stages 1 and 2. ($p < .05$).

3.6. Anxiety interactions on age-related changes in sleep

Table S7 summarizes the regression model used to assess the influence of self-reported anxiety severity. No significant interactions were found between age and the BAI. AHI was a significant predictor of deciles 1, 2, 4, 5, 6 and stages 1, 2, and 3 ($p < .05$).

3.7. Depression interactions on age-related changes in sleep

Table S8 presents the results of the regression model assessing the interaction between CES-D and age on sleep architecture. The interaction was not significantly associated with any sleep outcome. However, AHI was a significant predictor of deciles 1, 2, 4, 5, 6 and stages 1, 2 and 3 ($p < .05$).

4. Discussion

The current study examined age-related differences in sleep architecture and tested whether age interacts with BMI, sex, self-reported anxiety and depression in predicting sleep outcomes. We observed age-related differences in both traditional and ORP-based sleep architecture. Compared to younger adults, middle-aged to older adults spent a greater percentage of time in lighter sleep and less in deep sleep. Middle-aged to older adults also present with higher ORP within a given sleep stage than younger adults. We report that time in Stage 3 largely unexplained average ORP in NREM and in TRT, indicating that time in Stage 3 or SWS does not adequately explain sleep depth in NREM sleep or TRT. BMI appeared to affect the relationship between age and sleep architecture. Specifically, higher BMI in middle-aged to older adults was associated with a smaller age-related decline in deciles 1 and 2 and smaller age-related increases in decile 5. No interaction was observed between age and sex in predicting sleep architecture. Similarly, no interactions were found between age and either anxiety or depression in relation to sleep outcomes. AHI was included as a covariate in all interaction models due to the well-established relationship between sleep-disordered breathing and sleep disruption [35]. As expected, AHI was significantly associated with the majority of sleep architecture variables across all interaction analyses, indicating its influence on

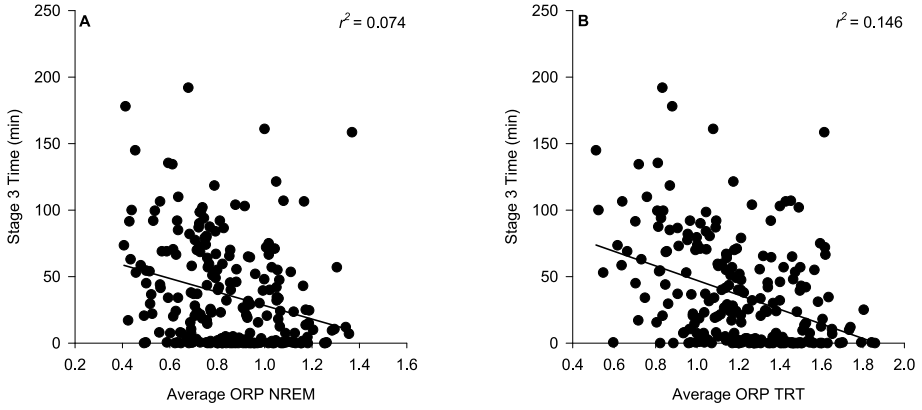

Fig. 2. (A) Association between time spent in stage 3 and average ORP in NREM sleep of the entire sample (n = 229). **(B)** Association between time spent in stage 3 and average ORP for the total sleep recording of the entire sample (n = 229). Both figures display negative associations.
Abbreviations: ORP; odds-ratio product, SWS; slow wave sleep, NREM; non-rapid eye movement, TRT; total recording time.

Table 2
Younger vs. middle-aged to older adult sleep characteristics.

	Age			
	Younger (<35 years)	Middle-aged to older (>50 years)	p-value	η^2
Average ORP				
TRT ORP	0.91 ± 0.25	1.25 ± 0.26	<.001	.186
NREM ORP	0.76 ± 0.42	0.90 ± 0.40	<.001	.074
Stage 1 ORP	1.22 ± 0.78	1.28 ± 0.66	.015	.026
Stage 2 ORP	0.72 ± 0.19	0.83 ± 0.22	.009	.029
Stage 3 ORP	0.38 ± 0.13	0.48 ± 0.22	.012	.042
REM ORP	1.07 ± 0.56	1.42 ± 0.49	<.001	.182
Wake ORP	2.07 ± 0.13	2.14 ± .042	.002	.042
Traditional stages				
Awake Stage (%TRT)	11.7 ± 7.7	24.4 ± 11.2	<.001	.151
Stage 1 (%TST)	10.6 ± 5.0	15.1 ± 8.4	.003	.038
Stage 2 (%TST)	54.4 ± 7.3	58.7 ± 10.9	.030	.021
Stage 3 (%TST)	17.5 ± 10.1	8.8 ± 10.7	<.001	.078
REM (%TST)	17.5 ± 8.0	17.4 ± 7.6	.990	.000
Stage 1 (min)	44.2 ± 24.7	51.8 ± 28.8	.148	.009
Stage 2 (min)	219.3 ± 49.9	203.8 ± 55.6	.129	.010
Stage 3 (min)	68.2 ± 38.7	30.6 ± 37.1	<.001	.114
REM (min)	71.5 ± 37.4	61.8 ± 31.9	.109	.001
Individual Deciles				
Decile 1 (%TRT)	7.7 ± 6.3	3.9 ± 5.3	<.001	.058
Decile 2 (%TRT)	19.1 ± 7.8	12.6 ± 8.7	<.001	.069
Decile 3 (%TRT)	21.4 ± 5.7	13.5 ± 5.0	<.001	.231
Decile 4 (%TRT)	17.2 ± 5.7	12.3 ± 4.3	<.001	.128
Decile 5 (%TRT)	11.0 ± 5.0	11.4 ± 4.2	.652	.001
Decile 6 (%TRT)	6.9 ± 4.7	10.4 ± 3.7	<.001	.096
Decile 7 (%TRT)	4.7 ± 2.7	8.7 ± 3.8	<.001	.131
Decile 8 (%TRT)	3.5 ± 2.0	7.4 ± 4.2	<.001	.109
Decile 9 (%TRT)	4.3 ± 3.2	7.0 ± 3.7	<.001	.068
Decile 10 (%TRT)	4.0 ± 3.3	12.7 ± 9.0	<.001	.117

Notes. Younger sample (n = 34), middle-aged to older sample (n = 195). ORP; odds-ratio product, REM; rapid eye movement stage, NREM; non-rapid eye movement stage, %TRT; percentage of total recording time, %TST; percentage of total sleep time. Data are presented as mean ± SD, results are from a one-way ANOVA. Eta-squared (η^2) = small (0.01), moderate (0.06), or large (>0.14) effect size.

age-related sleep changes.

Our study has demonstrated again [8] that time in stage 3 is not an appropriate reflection of sleep depth across the night Fig. 2. Thus, the use of N3 time alone would not have identified the decrease in sleep depth in stages 1, 2, or REM (Table 2) or the increased vigilance in stage wake (greater time in decile 10, Table 2). The significant decrease in deep sleep deciles with a paradoxical increase in time in full wakefulness are the expected changes associated with a decrease in sleep need or drive [16]. Had the poorer traditional sleep quality been due to comorbidities that promote poor sleep, time in decile 10 would have decreased, rather than increased. This lends additional support to the earlier conclusion [10] that age-related changes in traditional sleep architecture, including delayed sleep onset, deceased total sleep time, lower arousal threshold, decreased time in SWS, sleep fragmentation, increased wake time, and lower delta power [1–4] are primarily related to lower sleep need and lighter sleep.

With respect to our data, younger adults spent more time in deciles associated with deep sleep, while middle-aged to older adults spent a greater proportion in deciles associated with lighter sleep. Middle-aged to older adults spent more percentage of time in stage 1 and 2 sleep, more percentage of time awake, and less percentage of time in stage 3 sleep compared to younger adults. These findings, taken together, support previously reported shifts in sleep patterns associated with aging [3, 18]. Our results align with existing literature which reports that as age increases, the proportion of sleep time shifts from deeper sleep stages to lighter sleep stages, except we now report these changes with ORP. We also report higher ORP values within sleep stages in middle-aged to older adults compared to younger adults, despite no statistically significant differences in time spent in stage 1, 2 or REM we observed lighter sleep within these stages in middle-aged to older adults.

Our study found a statistically significant interaction with age and BMI in relation to deep sleep deciles 1 and 2, and additionally with decile 5. Prior research suggests that both age and BMI independently contribute to reduction in sleep depth, particularly with respect to slow-wave sleep, where increased age or BMI is associated with decreased time in stage 3 [18,24]. The previous work reporting associations between BMI and sleep architecture has most often assessed sleep using traditional sleep stages, which has limitations in the ability to assess sleep depth [8]. Younes et al. [10] observed no differences between ORP deciles among individuals with higher BMI (26–30 kg/m^2 and >30 kg/m^2) compared to those with lower BMI (18–26 kg/m^2). Our study, however, observed a positive association between the interaction of BMI and age on deep sleep deciles 1 and 2. It is important to emphasize that we are not suggesting that obesity can have a positive effect on sleep architecture. Rather, we cautiously note a potential interaction that warrants further investigation. BMI, while widely used, is a limited proxy measure of body composition and may not accurately reflect individual variability in muscle or fat mass [36]. The observed interaction may partially be explained by the limitations of BMI as a body composition measure. Higher BMI in our sample may reflect greater relative muscle mass or better muscle quality, which has been associated with greater sleep quality [37]. Our results may also be confounded by the sedentary inclusion criteria, which itself is a risk factor for prefrailty and frailty [38]. Individuals with lower BMI may have reduced muscle mass and be vulnerable to pre-frailty, which can negatively impact sleep [38, 39]. When our sample was divided into BMI groups represented in Fig. 5, we observed a larger proportion of women in the low BMI group (<22.7 kg/m^2, 81 %) compared to the high BMI group (>32.5 kg/m^2; 52 %). Given that all women in the study were post-menopausal, and that sleep complaints are common in the postmenopausal population, the observed BMI interaction may be influenced by differences in sex distribution across BMI groups [40]. Future studies could make use of higher resolution measures of body composition to address the limitations of our study.

We observed no significant interaction of sex and age in relation to sleep parameters. This finding differs from other ORP-based work [10]. Younes et al. [10] reported that women spent more time in deep sleep and less time in transitional sleep and full wakefulness compared to men; however, their study only examined differences by sex, without modeling interactions with age. Similar findings have been reported using traditional sleep architecture, where women spend more time in stage 3–4 and less time in stage 1 and 2 compared to men across age quartiles [18]. Our findings suggest no evidence that the age-related changes in sleep differ by sex. Similar findings are reported in a meta-analysis examining traditional sleep architectures by Boulos et al., [4], which found no significant interactions of sex and age for changes in stage 1, stage 2, or stage 3 sleep.

There were no interactions present between age and either anxiety or depression in predicting sleep architecture, suggesting that age-related changes in sleep occur independently of the severity or frequency of self-reported mental health symptoms. Previous research has linked depression with reductions in slow-wave sleep, and anxiety to both reduced slow-wave sleep and increased stage 1 sleep [25,26]. Our findings suggest that severity of anxiety or depression does not alter age-related changes in sleep architecture. However, it is important to note that these instruments (CES-D and BAI) are not diagnostic and rely on self-reported symptom frequency and severity. It is possible that in a larger sample, including individuals with clinically diagnosed anxiety or depression, interactions between age and mental health could emerge. Furthermore, this was not a clinically depressed or anxious population, and the restricted range of symptom scores may have limited our ability to detect any effects.

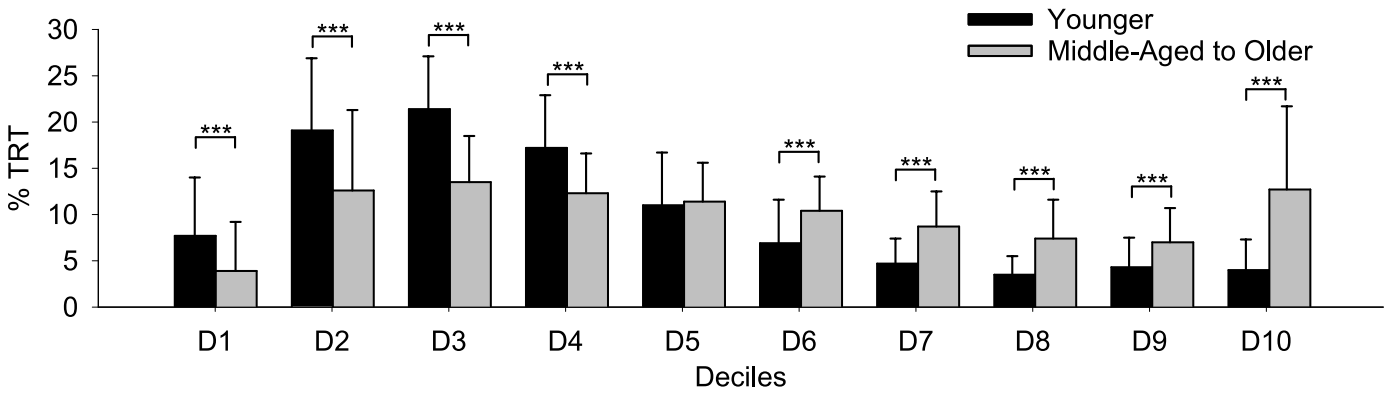

Fig. 3. Comparisons of deciles between younger (n = 34) and middle-aged to older adults (n = 195). The x-axis shows the ten deciles, and the y-axis shows percent recording time. Younger adults are spending significantly more time in deciles 1 through 4 and less time in deciles 6 through 10.
Abbreviations: %TRT, percent total recording time, D, decile. Asterisks indicate significant results from Tukey-Kramer post hoc test. *p < .05. **p < .01. ***p < .001. Bars represent means and standard deviations. Black bars represent younger adults and grey bars represent middle-aged to older adults.

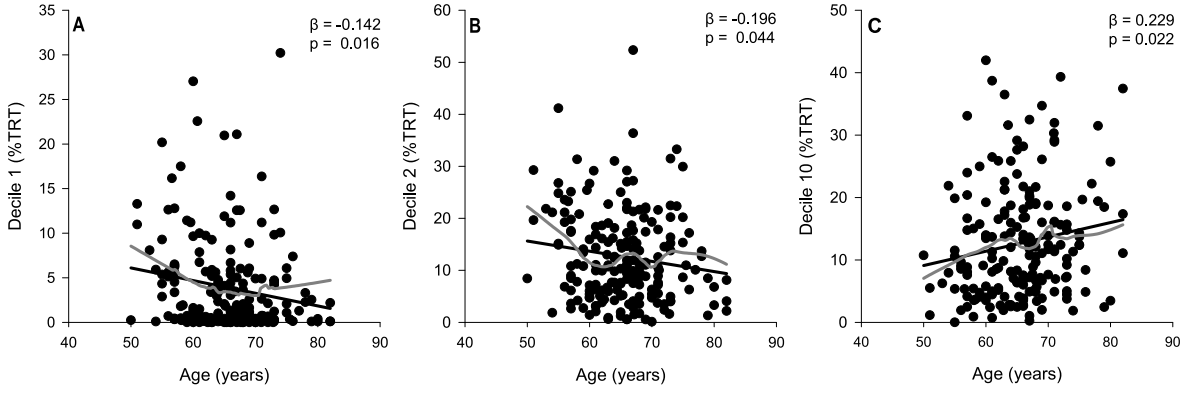

Fig. 4. A–C Association between age and deciles in middle-aged to older adults (n = 195). In each plot the solid black line represents the linear regression slope, while the solid grey line represents a locally estimated scatterplot smoothing (LOESS) curve. **(A)** The graph shows a negative association between decile 1 and age in middle-aged to older adults. The x-axis shows age in years, and the y-axis signifies decile 1 in percent recording time. **(B)** The graph shows a negative association between decile 2 and age in middle-aged to older adults. The x-axis shows age in years, and the y-axis signifies decile 2 in percent recording time. **(C)** The graph shows a positive association of decile 10 (full wakefulness) with age, in middle-aged to older adults. The x-axis shows age in years, and the y-axis signifies full wakefulness in percent recording time.
Abbreviations: β, unstandardized beta; p, statistical significance, %TRT, percent total recording time.

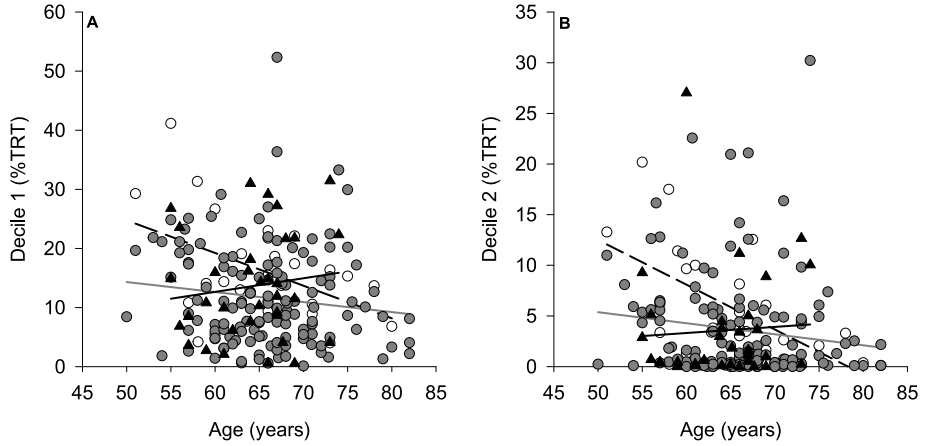

Fig. 5. A–B Associations between age and sleep architecture outcomes, stratified by BMI mean ± 1 SD (27.6 ± 4.9), based on the age × BIM interaction analyses (n = 195). In each plot black triangles and the solid black regression line represent BMI at +1 SD, filled grey circles and solid grey regression line represent mean BMI, and unfilled black circles and dashed black regression line represent BMI -1 SD **(A)** Displays the association of decile 1, showing a positive slope at high BMI (+1 SD), and negative slopes at mean and low BMI (−1 SD). **(B)** presents the association of decile 2, showing a positive slope at high BMI (+1 SD), and negative slopes at mean and low BMI (−1 SD).
Abbreviations: BMI, body mass index; %TRT, percentage of total recording.

Understanding how these factors influence sleep architecture in middle-aged to older adults could inform the development of targeted strategies to promote health and well-being in aging populations. Future research should employ ORP-based analyses to enhance our understanding of sleep depth changes across the lifespan. Longitudinal assessments may enhance insight into the mechanisms that drive changes in sleep architecture. Additionally, future studies should examine the relationship between body composition and sleep architecture using higher resolution measures of body composition.

Several limitations should be noted. First, the study did not account for variations in sleep hygiene such as bedtime routines or exposure to screen time, which could influence sleep quality [41]. Second, the use of different PSG systems, specifically the Embletta MPR and Cerebra Prodigy 2, may introduce a minor, though likely insignificant source of variability in data collection and scoring. However, our findings using two different acquisition systems (Embletta and Prodigy 2) are virtually identical to what was reported in a large community study using a totally different acquisition system (Compumedic) [10]. Third, we used self-reported measures of depression and anxiety. While these instruments have demonstrated validity and reliability, they remain susceptible to subjective biases, which could impact the reliability of our findings. Fourth, the three cohorts had different inclusion criteria, resulting in different baseline characteristics. Fifth, only the first night of PSG was recorded. This is a limitation due to the well documented first night effect (FNE). However, there is evidence to suggest that both stage 2 and stage 3 sleep are less affected by the FNE, and our interest was on sleep depth, suggesting that any potential effect of the FNE would be minimal [42]. Moreover, the use of portable, at-home PSG self-instrumented by participants likely reduced the effect of a novel environment and observer effect typically implicated in the FNE [43]. Additionally, the cross-sectional design introduces the possibility of selection bias. The current sample likely represents a predominantly Caucasian, healthier, and socioeconomically advantaged segment of the population. Participants in both BIM-I and BIM-II cohorts self-selected into an exercise intervention study, suggesting a higher level of health consciousness. These characteristics may limit the generalizability to more diverse aging populations. The younger cohort could not be included in the interaction analysis due to the absence of mental health questionnaire data, which limits our ability to fully explore the interactions. Finally, the cross-sectional design does not allow for causal inferences to be made regarding the reported age-related declines in deep sleep, increases in awake time or the interactions between age and BMI on these age-related changes.

5. Conclusion

This study reports that aging is associated with changes in ORP sleep architecture, reproducing findings from the Sleep Heart Health Study cohort, now demonstrated as a continuous rather than categorical association [10]. Middle-aged to older adults spend more percentage time in lighter stages of sleep and less time in deep stages of sleep compared to younger adults. Despite no differences in time spent in some traditional sleep stages we observed greater ORP within these stages in middle-aged to older adults, suggesting they exhibit lighter sleep within traditional sleep stages. We display that time in stage 3 or SWS does not adequately explain sleep depth, evidenced by the only minor association between time in stage 3 and average NREM ORP or TRT ORP. Additionally, the interaction of age and BMI was positively associated with time spent in deep sleep deciles 1 and 2 and negatively with decile 5, suggesting that BMI may influence age-related changes in ORP architecture. These findings highlight the importance of evaluating objective sleep architecture in aging populations.

CRediT authorship contribution statement

Shane Magnison-Benoit: Writing – review & editing, Writing – original draft, Visualization, Validation, Project administration, Methodology, Investigation, Formal analysis, Conceptualization. **Connor Snow:** Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation, Formal analysis. **Matiram Pun:** Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation, Formal analysis, Conceptualization. **Harshita Gauba:** Writing – original draft, Methodology, Investigation, Formal analysis, Conceptualization. **Sophie Berghmans:** Writing – review & editing, Writing – original draft, Investigation. **Aimee Clarke:** Writing – review & editing, Writing – original draft, Investigation. **Bradley Hansen:** Project administration, Investigation. **Alison M.H. Donald:** Writing – review & editing, Writing – original draft, Investigation. **Beth Gerardy:** Software, Investigation, Formal analysis, Data curation. **David B. Hogan:** Writing – review & editing, Writing – original draft, Funding acquisition, Conceptualization. **Michael D. Hill:** Writing – review & editing, Methodology, Funding acquisition, Conceptualization. **Jessalyn Holodinsky:** Validation, Methodology, Conceptualization. **Willis H. Tsai:** Writing – review & editing, Methodology, Conceptualization. **R. Stewart Longman:** Writing – review & editing, Methodology, Conceptualization. **Jean M. Rawling:** Writing – review & editing, Funding acquisition, Conceptualization. **Magdy Younes:** Writing – review & editing, Validation, Methodology, Conceptualization. **Marc J. Poulin:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Project administration, Methodology, Funding acquisition, Conceptualization.

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Declaration of competing interest

The author is an Editorial Board Member/Editor-in-Chief/Associate Editor/Guest Editor for this journal and was not involved in the editorial review or the decision to publish this article.

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2025.106629.

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