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Original Article

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Age and objectively measured sleep: investigating associations and interactions by sex and race in middle-aged and older adults

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Abstract

Study Objectives: Few studies of middle-aged and older adults have examined the association between age and sleep using objective sleep measures. We examined these associations in adults aged ≥40 years using wrist actigraphy, and investigated whether these associations differed by sex and race.

Methods: Participants were 468 cognitively normal adults aged ≥40 years enrolled in the Baltimore Longitudinal Study of Aging who completed wrist actigraphy. We used Generalized Least Squares Models to examine the associations of age with actigraphic sleep parameters, including total sleep time (TST), sleep efficiency, sleep onset latency, and wake after sleep onset (WASO). We conducted interaction and stratification analyses to test whether cross-sectional age-sleep associations were modified by sex and race.

Results: In analyses adjusting for sex, body mass index, and individual medical conditions, older age was associated with longer TST from ages 40–70 that plateaued after age 70. Older age also was associated with lower sleep efficiency, longer sleep onset latency, and greater WASO. In men only, after age 70, older age was associated with shorter TST, lower sleep efficiency, longer onset latency, and greater WASO. However, we did not observe any significant interactions of race with age.

Conclusions: Older age was associated with longer TST from ages 40 to 70 and with poorer sleep quality after age 40, and these relationships might vary by sex. Future studies with larger sample sizes are needed to investigate mechanisms that may account for sex differences in the observed age-sleep associations.

Key words: aging; sleep; actigraphy; middle-aged adults; older adults

Statement of Significance

Although differences in sleep between age groups have been identified previously, few studies have investigated associations between age and objectively measured sleep in people aged 40 years and older after adjusting for common aging-related comorbidities, or explored whether these associations differ by sex or race. We found that older age was associated with longer total sleep time (TST) from ages 40 to 70, but this plateaued after age 70. Among adults aged >70 years, older age was associated with shorter TST, lower sleep efficiency, longer onset latency, and greater WASO in men, but not women. Results further elucidate how the aging process affects sleep in middle-aged and older adults.

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Sleep is a fundamental determinant of health [1]. Consistent and sufficient sleep is associated with maintained cognitive function, reduced risk of cardiovascular disease (CVD), and removal of DNA damage and reactive oxygen species [2–5]. Sleep patterns change across the lifespan [6] due to brain maturation that occurs from childhood through adulthood [7], changes in life circumstances (e.g. employment, occupation, retirement, and bereavement) [8–10], onset and course of medical and psychiatric conditions [11, 12], and as a consequence of the biological aging process [13].

Compared with young adults, middle-aged and older people commonly have worse objectively measured sleep quality and shorter sleep duration [7]. These deficiencies in sleep have also been associated with negative health outcomes, including poorer cognitive performance [14] and a higher risk of cardiovascular and metabolic disease [15, 16]. Although a recently published meta-analysis investigated links of age with actigraphic sleep [17], few epidemiologic studies have investigated the associations of objectively measured sleep parameters in persons aged 40 years and older, and findings from existing studies have been inconsistent. For example, a cross-sectional study of 504 adults ages 40–87 found older age was associated with higher actigraphic sleep efficiency, but not with sleep onset latency [18]. In contrast, another cross-sectional study found older age was associated with lower actigraphic sleep efficiency and longer sleep onset latency in adults ages 36–91 [19]. In addition, Redline and colleagues reported that older age was associated with lower polysomnography-assessed sleep efficiency in 2685 adults aged 37–92 [20]. These inconsistent age-sleep associations could be due to differences in study design, sample size, and participant characteristics [17]. Importantly, Evans et al. reported that many studies of age and sleep have overlooked the potential confounding effects of physical or psychiatric comorbidities, such as depression, obesity, and respiratory diseases on age-sleep associations [17]. It is well established that these factors can affect sleep. Therefore, not adjusting for these variables may lead to confounded estimates of age-sleep associations [5, 21–23]. Thus, it remains unclear how age is associated with actigraphic sleep in middle-aged and older adults, after accounting for these comorbidities.

In addition to mental and physical health conditions, moderators, such as sex and race that may affect changes in sleep over time, have been understudied in sleep and aging research [17]. For example, women are more likely to experience insomnia and sleep disruptions throughout their lifespan than men [24]. In addition, members of minoritized racial and ethnic groups are more likely to have sleep disturbances and on average lower sleep quality than white individuals in the United States [25–27]. It is unclear, however, whether sex and race modify associations between age and sleep.

In the current study, we addressed these literature gaps by examining cross-sectional associations between age and actigraphy-assessed sleep measures after adjusting for common aging-related comorbidities. In addition, we explored whether these associations differ by sex and race. We hypothesized that: (1) older age will be associated with shorter total sleep time (TST), lower sleep efficiency, longer sleep onset latency, and greater wake after sleep onset (WASO) and (2) age-sleep associations will be modified by sex and race.

Methods

Participants

We studied 468 participants in the Baltimore Longitudinal Study of Aging (BLSA), an observational study that originally aimed to investigate the effect of aging on physical and cognitive health outcomes [28]. To be eligible for enrollment in BLSA, participants are required to be free of medical conditions (except for well-controlled hypertension and a history of skin cancer), cognitive or mobility impairments, and disabilities during enrollment. They could not be taking medications, such as corticosteroids, immunosuppressants, antibiotics, or H2 blockers. However, these criteria do not apply to follow-up assessments after the initial enrollment. We included participants aged ≥40 years who completed wrist actigraphy at least once and were free of dementia or mild cognitive impairment at our study baseline, defined here as the first visit at which participants had valid actigraphy data. Participants aged <60 have study visits every 4 years, those aged 60–79 have visits every 2 years, and participants aged >80 have annual visits. Informed consent was obtained from each participant. The study protocol was approved by the Institutional Review Board at the National Institutes of Health Intramural Research Program.

Measures

Wrist actigraphy data collection began in the BLSA in 2012. At each study visit, participants were asked to wear the Actiwatch-2 wrist actigraph (Philips-Respironics, Bend, OR) on their nondominant wrists for seven 24-hour periods. Both movement in activity counts and ambient light levels were recorded by the actigraph. Participants were asked to press an event-marker button on the device each time they intended to go to sleep (i.e. at "lights out") and when they got up to start the day. While wearing the actigraph, participants were also asked to complete sleep diaries in the morning and at bedtime, to record sleep-related information, including times they began trying to go to sleep at night and got up for the day (consistent with event-marker presses), any actigraph removal, and the timing of any naps. Using Actiware v. 6.0.9 software (Philips-Respironics), two trained research staff members used event markers, sleep diary, and ambient light data to independently identify the period during which participants were in bed with the intention of sleeping. We used a validated algorithm to derive sleep parameters [29]: TST (total minutes spent asleep while in bed); sleep efficiency (% of the time in bed spent asleep); sleep onset latency (minutes from "lights out" until sleep onset); and WASO (minutes spent awake after sleep onset). We computed the mean of each parameter across nights of valid actigraphy data for each participant.

Demographic characteristics and comorbidities

Self-reported demographic characteristics were obtained, including sex, age, and race. Participants' height and weight were measured, and body mass index (BMI) was calculated. Due to the unbalanced race distribution (Table 1), we dichotomized race into white and minoritized (comprised of black, Asian, and other nonwhite) participants. During each visit, participants were asked whether they had ever been diagnosed with respiratory diseases (i.e. asthma and bronchitis), diabetes, liver and kidney diseases (i.e. cirrhosis, hepatitis, and kidney diseases), any cancers, and CVD (i.e. hypertension [CVD risk factor], coronary artery disease, heart attack, congestive heart failure, angina, stroke, transient ischemic, having any vascular procedures, peripheral artery disease, and peripheral artery disease). Participants' depressive symptomatology was measured using the Center for Epidemiologic Studies Depression Scale (CES-D), with the sleep item removed. In addition, participants reported the use of sleeping pills/medications in the past month (options: never used, </

Table 1. Participant Characteristics

	Value
Number of participants, *N*	468
Total number of visits with actigraphy data, *N*	636
Distribution of participants by number of visits with actigraphy data, N (%)	
One visit	345 (73.7%)
Two visits	91 (19.4%)
Three visits	22 (4.7%)
Four visits	7 (1.4%)
Five visits	3 (0.6%)
Sex, N (%)	
Men	223 (47.6%)
Women	245 (52.4%)
Race, N (%)	
Minoritized	137 (29.3%)
Black	106 (22.6%)
Asian	24 (5.1%)
Other nonwhite	4 (0.9%)
Unknown	3 (0.6%)
White	331 (70.7%)
Sleep medication usage, *N* (%)	69 (14.7%)
Baseline age, mean (SD) [range] (year)	70.8 (12) [40 – 96]
Baseline BMI, mean (SD) [range] (Kg/m^2)	27.0 (4.4) [17.1 – 42.9]
Baseline years of education mean (SD) [range] (year)	17.8 (2.4) [9 – 25]
Baseline CES-D, mean (SD) [range] Baseline comorbidity status, *N* (%)	3.9 (4.4) [0 – 25]
CVD	276 (59.0)
Respiratory disease	73 (15.6)
Diabetes	79 (16.9)
Liver and kidney disease	59 (12.6)
Cancer	147 (31.4)

CVD, cardiovascular disease; CES-D, Center for Epidemiologic Studies Depression Scale; minoritized group comprised of black (*N* = 106), Asian (*N* = 23), and other nonwhite (*N* = 8).

week, 1–2/week, 3–4/week, or ≥5/week). We dichotomized these responses into the use of any sleep medication versus no sleep medication use.

Statistical analysis

First, generalized least squares (GLS) models were used to assess the cross-sectional association of age with sleep parameters. Across models, age was the primary predictor and sleep parameters were the outcomes. Given the association between age and the sleep parameters could be nonlinear, age was modeled as a restricted cubic spline with three knots. We controlled for fixed-effect covariates that have previously shown to be associated with sleep, namely race and sex, as well as time-varying covariates such as BMI, CES-D, and disease status (i.e. CVD, respiratory disease, diabetes, liver and kidney disease, and cancer, separately), which we binarized. To adjust for the correlations of data across time within individuals, we used a correlation structure with age as the time covariate and participant identifier as the grouping factor. Both the number of knots and correlation structure were chosen based on better model performance, indicated by a lower Akaike Information Criterion. After the best-fitting model was selected, we used a corCAR1, which represents an autocorrelation structure of order 1 with a continuous time covariate, with three knots as the correlation structure.

Second, in the best-fitting GLS models identified, we tested whether the cross-sectional age-sleep associations examined in the first set of models were modified by sex and race by adding an interaction term between age and sex or an interaction term between age and race. To further investigate how sex and race modified age-sleep associations, we used the GLS models to reexamine age-sleep relationships after stratifying by sex (men vs women) and race (white adults vs minoritized adults) following the same covariate structure as described above.

We performed sensitivity analyses that excluded observations from participants who reported using sleep medications in the past month prior to completing actigraphy or who had questionable actigraphy data. We also conducted sensitivity analyses that further adjusted for either self-reported physical activity, years of education, cognitive performance, or number of hours worked per week in models examining the association between age and TST. We reexamined the association between age and TST after stratifying participants by whether they reported working 0 versus >0 hours for pay at the visit(s) at which they completed actigraphy, following the same covariate structure as described in the main model. All statistical analyses were conducted using *nlme*, *rms*, and *parameters* packages in R software (version 4.0.2). A *p*-value cutoff of < .05 was used to determine statistical significance. We provide detailed descriptions of equations used for the statistical models in Supplementary Information.

Results

Participant characteristics

Of the 468 participants, 223 (47.6%) were men and 331 (70.7%) were white (Table 1). In addition, 345 (73.7%) participants were assessed once, and 123 participants had repeated measurements (2–5 measurements per participant). The 123 participants with follow-up data (mean (SD) [range] baseline age = 74 (9.7) [42–93] years) were followed for an average (SD) of 2.4 (0.7) years. At our study baseline, 276 (59.0%) participants had CVD, 73 (15.6%) had respiratory disease, 79 (16.9%) had diabetes, 59 (12.6%) had liver or kidney disease, and 147 (31.4%) had a history of cancer. The baseline average age, BMI, and CES-D were 70.8 years, 27.0 kg/m^2, and 3.9, respectively. Across all visits, the mean (SD) TST was 401 (61) minutes, sleep efficiency was 84% (7.0), sleep onset latency was 13 (12) minutes, and WASO was 47 (22) minutes (Table 2). Participant characteristics stratified by sex and race are shown in Supplementary Tables S1 and S2. Liver and kidney disease and cancer were significantly more common in men compared to women, and CVD and diabetes were more common among minoritized participants compared to white participants.

Cross-sectional associations between age and sleep parameters

TST steadily increased from ages 40–70, and plateaued at ages >70 (Figure 1A). However, we found quasilinear associations of age with sleep efficiency, sleep onset latency, and WASO, such that older age was associated with lower sleep efficiency, longer

sleep onset latency, and greater WASO (Figures 1, B, C, and D). The fully-adjusted model results showing the cross-sectional associations of age and the covariates with the four sleep parameters are shown in Table 3, and the standardized effect sizes of each variable in these models (refitted from models with original data after z-scoring) are shown in Supplementary Table S3.

We found a significant and positive cross-sectional association between age and TST. There was a main effect of sex such that men had a shorter TST, lower sleep efficiency, longer sleep onset latency, and greater WASO compared to women. In addition, compared to white participants, minoritized participants had shorter TST and lower sleep efficiency.

Table 2. Actigraphic Sleep Parameters Across All Visits

	Mean (SD)	Median (IQR)	Range
TST (minutes)	401 (61)	406 (82)	161–602
Sleep efficiency (%)	84 (7.0)	85 (7.7)	50–97
Sleep onset latency (minutes)	13 (12)	9.2 (10)	0.14–101
WASO (minutes)	47 (22)	42 (26)	8.4–146

TST, average total sleep time; WASO, wake after sleep onset; Measurements were collected from 468 participants, resulting in a total of 636 observations over time.

Sex and race moderation and stratified analyses

Interactions of age with sex and race are shown in Table 4. Race did not statistically significantly modify the cross-sectional association of age with any of the sleep parameters. However, we found that sex significantly modified the cross-sectional association of age with sleep efficiency and WASO. To further explore the sex-by-age interaction effects, we performed sex-stratified analyses.

The associations between age and the sleep parameters stratified by sex are shown in Figure 2, and the detailed model outcomes of the results are listed in Supplementary Table S4. We found a significant linear effect of age on TST only in women (Supplementary Table S4). The cross-sectional association between age and sleep parameters differed by sex. In men aged >70, but not in women aged >70, older age was associated with shorter TST (Figures 2, A and E), lower sleep efficiency (Figures

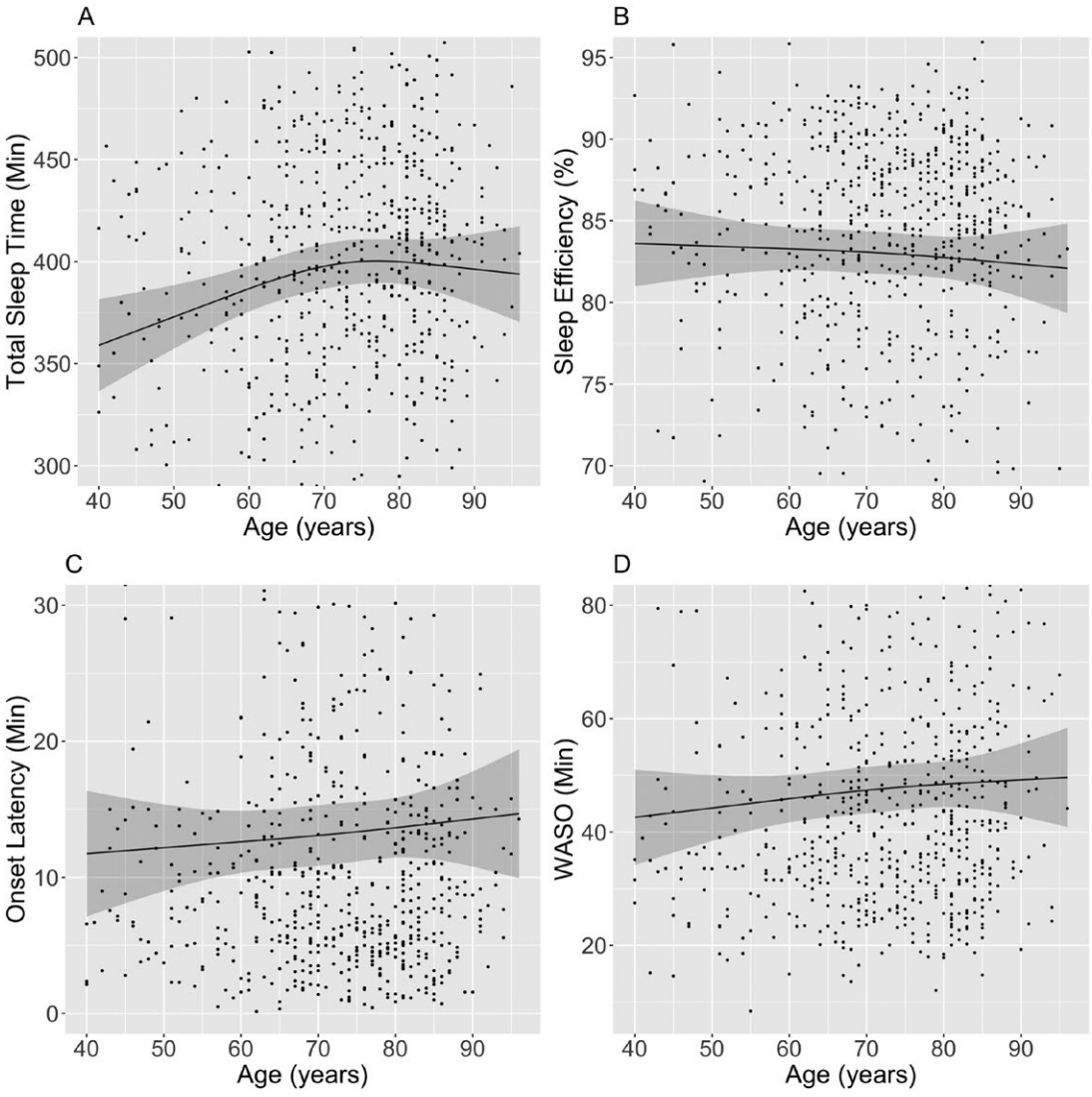

Figure 1. Cross-sectional associations between age and sleep parameters.

Table 3. Cross-Sectional Associations of Participant Characteristics With Sleep Parameters (B [95% CI])

	TST	Sleep efficiency	Onset latency	WASO
Age (linear effect)	**1.4 (0.5, 2.3)****	−0.02 (−0.1, 0.1)	0.04 (−0.1, 0.2)	0.2 (−0.2, 0.5)
Age (nonlinear effect)	**−1.0 (−2.0, 0.05)**^	−0.01 (−0.1, 0.1)	0.01 (−0.1, 0.2)	−0.05 (−0.4, 0.3)
Sex (ref. women)	**−28 (−39, −18)*****	**−2.7 (−3.9, −1.5)*****	**1.9 (−0.3, 4.1)**^	**4.8 (0.9, 8.7)***
BMI	**−1.4 (−2.6, −0.2)***	**−0.1 (−0.3, 0.003)**^	**0.4 (0.1, 0.6)****	−0.1 (−0.5, 0.4)
Race (ref. white)	**−24 (−36, −12)*****	**−2.4 (−3.8, −1.0)** **	1.5 (−0.9, 3.9)	0.5 (−4.0, 5.0)
CVD	−3.0 (−14, 8.4)	**−1.2 (−2.5, 0.1)**^	0.9 (−1.5, 3.2)	**4.9 (0.6, 9.2)***
Liver and kidney disease	−6.6 (−22, 8.7)	−1.3 (−3.1, 0.5)	**3.5 (0.4, 6.6)***	1.8 (−3.9, 7.5)
Cancer	8.6 (−2.8, 20)	0.8 (−0.6, 2.1)	−1.1 (−3.5, 1.3)	−1.3 (−5.4, 2.8)
Respiratory disease	−5.8 (−20, 8.3)	−0.9 (−2.5, 0.8)	1.9 (−1.0, 4.8)	1.9 (−3.4, 7.2)
Diabetes	−2.8 (−17, 11)	0.4 (−1.2, 2.0)	**−3.6 (−6.3, −0.9)***	−1.4 (−6.5, 3.7)
CES-D	−0.5 (−1.5, 0.5)	−0.1 (−0.2, 0.1)	0.2 (−0.1, 0.4)	−0.04 (−0.4, 0.3)

^$p < .1$, *$p < .05$; **$<p < .01$; ***$p < .001$; bold values, $p < .1$; BMI, body mass index; CI, confidence interval; CVD, cardiovascular disease; CES-D, Center for Epidemiologic Studies Depression Scale.

Table 4. Interaction *p*-Values for Age*Sex and Age*Race

	TST	Sleep efficiency	Onset latency	WASO
Age (linear effect)*sex + age (nonlinear effect)*sex	0.74	**0.005****	0.25	**0.02***
Age (linear effect)*race + age (nonlinear effect)*race	0.76	0.75	0.96	0.71

*$p < .05$; **$<p < .01$; bold values, $p < .1$; *p*-value obtained from the Wald Chi-Square test.

2, B and F), longer onset latency (Figures 2, C and G), and greater WASO (Figures 2, D and H). In addition, the slopes of the associations of age with sleep efficiency, onset latency, and WASO were steeper in men at ages >70 compared to those at ages <70 (Figures 2F, G, and H).

The cross-sectional associations between age and sleep parameters stratified by race are shown in Figure 3, and the detailed model outcomes are listed in Supplementary Table S5. A significant linear association of age with TST was observed in White but not minoritized participants (Supplementary Table S5). In terms of nonlinear relationships, older age was associated with steadily longer TST in minoritized participants (Figure 3A), whereas a positive association was found only among white participants below age 70 (Figure 3E). Similarly, older age was associated with greater WASO in white participants (Figure 3H), while a similar trend was observed only in minoritized participants below age 70 (Figure 3D). In addition, we found that older age was cross-sectionally associated with steadily lower sleep efficiency (Figure 3F) and longer sleep onset latency (Figure 3G) in white participants, while a somewhat flat association was observed among minoritized participants (Figures 3B and C).

Sensitivity analysis

As a sensitivity analysis, we excluded 85 observations from 69 participants who reported using sleep medications (>4 times/week) in the past month prior to the actigraphy assessment. As shown in Supplementary Figure S1 and Table S6, there were no meaningful differences in the findings from the main analyses, in terms of statistical significance. The age-sleep associations seen in the main analyses remained relatively unchanged in a subdataset, excluding 4 observations from three participants who had questionable actigraphy data. We only found that the nonlinear effect of age on TST was changed from nonsignificant to significant (Supplementary Figure S2 and Table S7). As shown in Supplementary Table S8, adjusting for either self-reported physical activity, years of education, or number of hours worked per week did not substantially alter the age-TST association in terms of effect sizes and statistical significance. Accounting for cognitive performance did not noticeably alter the age-TST association in terms of effect size, but the association fell below the level of significance (*p*-value = .15). In participants who did not work for pay, we found that older age was still associated with longer TST from age 40 through the 70s (Supplementary Figure S3A), although the effect was smaller compared to the main results. On the other hand, in participants who did work for pay, we found a steeper increasing trend in TST from age 40 to the 60s (Supplementary Figure S3B).

Discussion

In the present study, we examined associations of age with objective sleep parameters, measured by actigraphy, in middle-aged and older adults. In contrast to most prior studies, we adjusted for the possible association of race, sex, BMI, and common aging-related comorbidities, and examined whether age-sleep associations were modified by sex and race. We found significant and nonlinear associations of age with TST, such that older age was associated with longer TST from ages 40–70 and plateaued after age 70, after adjusting for common aging-related comorbidities. We also found that, among adults aged >70 years, older age was associated with shorter TST, lower sleep efficiency, longer sleep onset latency, and greater WASO in men, but not women.

Our findings on the association between age and TST in all participants are partially consistent with prior research. A previous cross-sectional study reported a positive association between age and actigraphic sleep in 300 adults in the United States aged 20–71 [30]. Similarly, a cross-sectional study of 24 350 adults aged 55–70 in Germany reported a positive association between age and self-reported sleep, and the authors hypothesized this association was due to participants transitioning to retirement or part-time work [31]. In contrast to our findings, Evans et al. found nonsignificant positive cross-sectional associations of age with actigraphy-assessed TST in a group of 14 participants aged

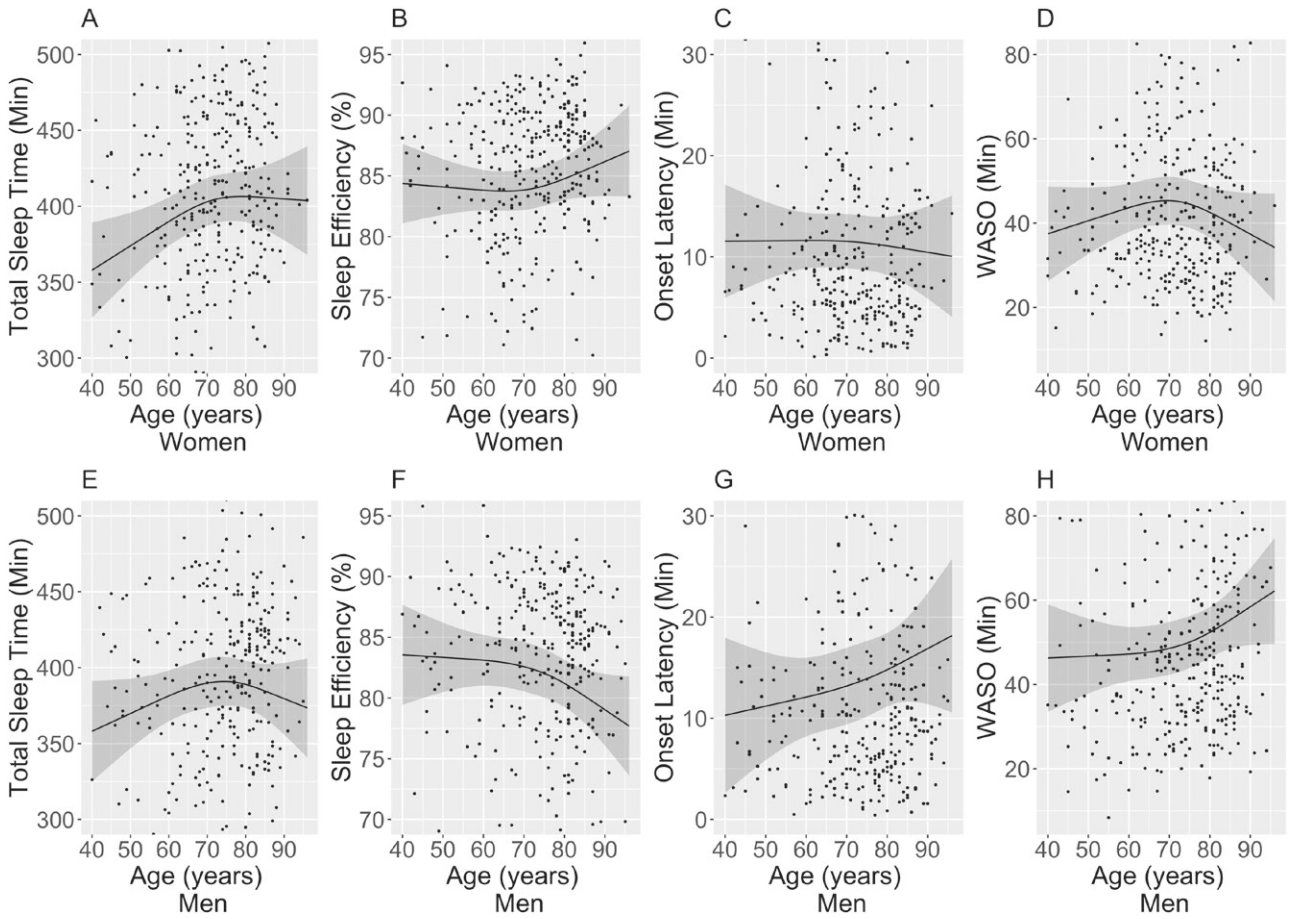

Figure 2. Cross-sectional associations between age and sleep parameters stratified by sex (women vs Men).

71–91 [32], in contrast with our findings that TST plateaued at ages >70. However, we acknowledge that the current findings contradict the majority of existing literature, which reports that older age is associated with shorter TST. For example, one study found a negative association of age with TST in people of a similar age range [33]. This discrepancy may be due to differences in participant characteristics (e.g. demographics, comorbidities, and working status). BLSA participants tend to be highly educated (mean of 17.8 years in this sample) and are required to be free of most comorbidities on enrollment, although they commonly develop them subsequently. In addition, working status may have played a role, supported by our findings that there was a flatter increasing trend in TST from ages 40 to 70s, among participants who did not work for pay, compared to the results observed in all participants or participants who worked for pay (Supplementary Figure S3A). In addition, the current results should be interpreted with caution given the interindividual variability we observed.

Although we found a positive association of age with TST from ages 40–70, overall sleep quality appeared to be poorer as people transitioned from middle age to older adulthood. To the best of our knowledge, this is the first study to report among middle-aged and older adults that age was cross-sectionally associated with longer sleep onset latency, greater WASO, and lower sleep efficiency after adjusting for demographics and comorbidities as potential confounders. Our findings are supported by previous findings that, among adults aged 21 to 91, those aged 80–91 had the lowest actigraphic sleep efficiency, early middle-aged people aged 36–44 had the highest actigraphic sleep efficiency, and those aged 61–79 had moderate levels of sleep efficiency [19]; however, that study did not adjust for participants' demographics or medical conditions. Our results are also in line with a previous meta-analysis, which reported that age is associated with longer sleep onset latency, greater WASO, and lower sleep efficiency assessed by polysomnography or actigraphy [7]. The age-sleep associations observed in the current study may be due to: (1) age-related changes in sleep-related hormones (e.g. growth hormone, sex hormones, and melatonin) that affect sleep quality [6] and (2) possible unmeasured aging-related characteristics (e.g. comorbidities or major life events, such as retirement and death of a family member) that may adversely affect sleep [8, 34, 35]. Future research is needed on the underlying mechanisms through which age may affect sleep quality across the lifespan.

Although the aging process is associated with declines in sleep quality and quantity, not all older adults experience notable sleep disruptions [36]. For example, we found that older age was associated with longer TST, lower sleep efficiency, and greater WASO in both men and women aged <70, but in adults older than 70, older age was only associated with shorter TST, lower sleep efficiency, longer sleep onset latency, and greater WASO among men. In addition, we found that the slope of the associations of age with sleep efficiency and WASO were steeper at ages >70 compared to those at ages <70 in men, but not in women. If our findings are replicated, greater attention to these aspects of sleep quality may be needed among men ages 70 and older. Although no studies to our knowledge have investigated sex differences in the associations between age and actigraphic sleep parameters, a cross-sectional study of 2500 adults aged 37–92 used PSG (the

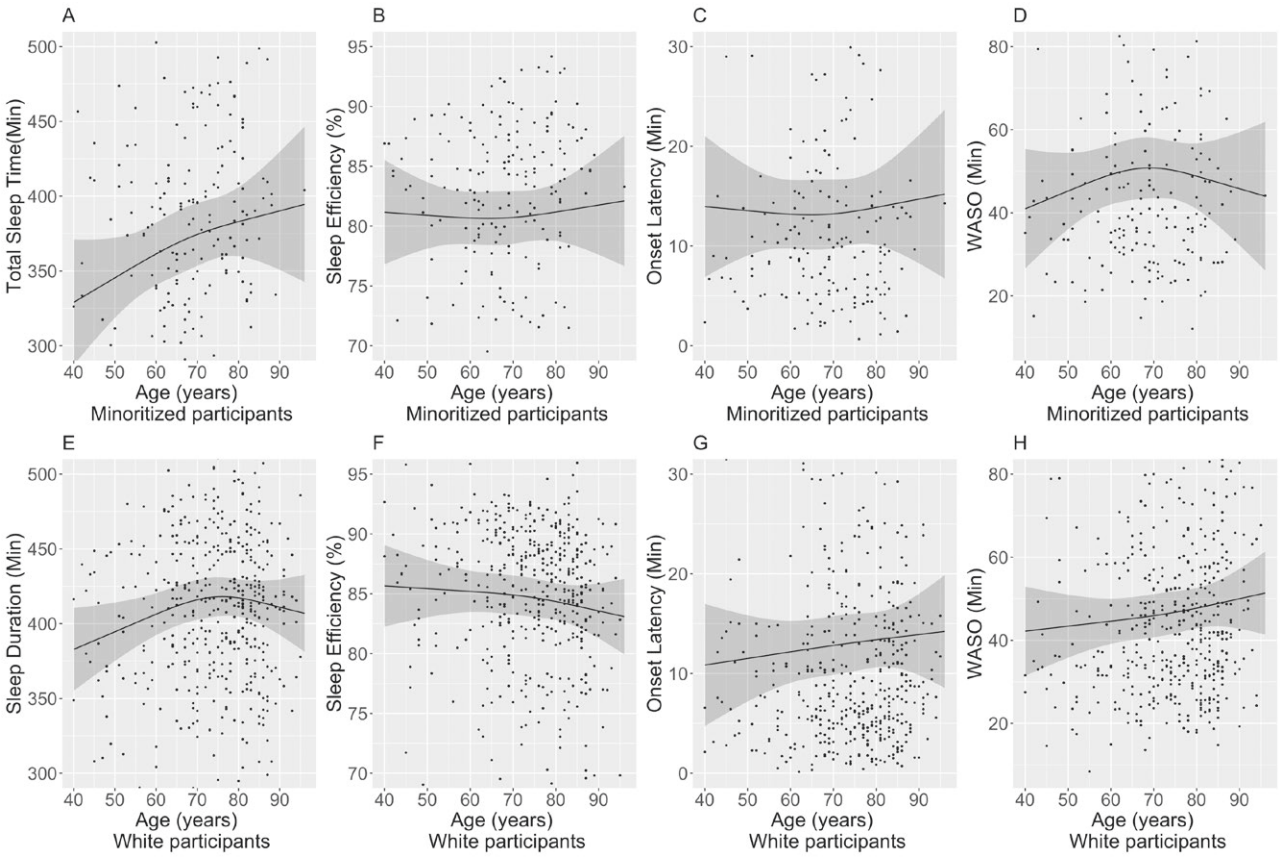

Figure 3. Cross-sectional associations between age and sleep parameters stratified by race (minoritized adults vs white adults).

gold-standard objective sleep measure), and observed sex differences in slow-wave sleep as a function of age [20]. On the other hand, the current findings differ from those of a previous study, in which older age was associated with a more rapid decrease in self-reported TST in women than in men [37]. The sex differences in age-sleep associations may be driven by factors related to biological aging [38, 39]. For example, testosterone in men and estrogen in women are closely linked to nocturnal sleep [40]. In men, testosterone levels progressively and gradually decline with aging [41], and lower testosterone levels have been associated with greater WASO and lower sleep efficiency [42]. The sex differences in the age-sleep associations could be attributed to other participant characteristics that differed between men and women (e.g. race and comorbidities). Future studies are needed to fully understand sex-specific differences in sleep during the aging process.

To the best of our knowledge, the present study is the first to examine the moderating role of race on age-sleep associations. Although age X race interaction terms were nonsignificant, we performed race-stratified analyses to further explore the potential moderating effect of race. We found that older age was associated with lower sleep efficiency and greater WASO only in minoritized participants aged <70 and with longer sleep onset latency only in minoritized participants aged>70. These race differences could be partially due to other participant characteristics that differed between white and minoritized participants (e.g. aging-related comorbidities). Nonetheless, results should be interpreted with caution given the limited sample of minoritized participants. Future studies with larger sample sizes are needed to rigorously examine race-related differences in age-sleep associations, which may shed light on sleep health disparities.

This study has several strengths. These include: (1) the use of wrist actigraphy to assess sleep, (2) adjustment for numerous comorbidities that may affect age-sleep associations, and (3) examination of moderation of the age-sleep associations by sex and race. There are also some limitations, however. First, the current findings may not be generalizable to the broader population of middle-aged and older adults, given that the BLSA participants tend to be healthier on average than the general population. Second, only 29.3% of participants were from minoritized populations, which decreases our ability to accurately estimate how race modifies the age-sleep associations. Third, the associations between age and sleep parameters could be confounded by unmeasured subclinical disease. Fourth, some of the sleep parameters (i.e. TST and sleep efficiency) were derived from an algorithm based on a limited sample with an age range that did not cover the older individuals represented in the current study [29]. More studies are needed to establish the validity of using actigraphy to examine the age-sleep associations, especially in older adults. Finally, it is possible that some of the variables we adjusted for to avoid confounding may actually be on the causal pathway between age and the sleep parameters, leading to an over-adjustment.

Conclusion

This study of age and sleep in a community-based cohort of older adults found that, after controlling for common aging-related comorbidities, age is associated with sleep time (longer TST from ages 40–70 and plateaued after age 70) and quality (longer sleep onset latency, greater WASO, and lower sleep efficiency) in people

aged 40 years and older. In addition, the cross-sectional relationships of age with sleep efficiency and WASO may be modified by sex, and efforts should be made to improve sleep quality, especially in men over the age of 70. Future studies are needed to investigate mechanisms that may account for the plateau in TST we observed after age 70 and the sex differences in observed age-sleep associations.

Supplementary Material

Supplementary material is available at SLEEP Advances online.

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Disclosure Statement

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Author Contributions

Linchen He (Conceptualization [equal], Data curation [equal], Funding acquisition [equal], Investigation [lead], Methodology [equal], Software [lead], Supervision [equal], Validation [lead], Visualization [lead], Writing—original draft [lead], Writing—review & editing [lead]), Jill Rabinowitz (Conceptualization [equal], Data curation [equal], Investigation [equal], Methodology [equal], Visualization [equal], Writing—original draft [equal], Writing—review & editing [equal]), Yang An (Methodology [equal], Visualization [equal], Writing—review & editing [equal]), Chandra Jackson (Methodology [equal], Writing—review & editing [equal]), Ryan Hellinger (Investigation [equal]), Sarah Wanigatunga (Investigation [equal], Project administration [equal]), Jennifer Schrack (Investigation [equal], Writing—review & editing [equal]), Luigi Ferrucci (Writing—review & editing [equal]), Eleanor Simonsick (Writing—review & editing [equal]), Kirsten Koehler (Writing—review & editing [equal]), and Adam Spira (Conceptualization [lead], Methodology [equal], Resources [lead], Supervision [lead], Visualization [equal], Writing—original draft [equal], Writing—review & editing [equal])

Data Availability

The data for this study are available upon request from the BLSA website (https://www.blsa.nih.gov). All requests undergo a review process performed by the BLSA Data Sharing Proposal Review Committee. Requests to access the datasets should be directed to https://www.blsa.nih.gov.

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