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Original Contribution

Determinants of Change in Objectively Assessed Sleep Duration Among Older Men

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We examined potential risk factors for changes in objectively assessed sleep duration within a large sample of community-dwelling older men. Participants (n = 1,055; mean baseline age = 74.6 (standard deviation (SD), 4.7) years) had repeated ActiGraph assessments (ActiGraph LLC, Pensacola, Florida) taken at the baseline (2003–2005) and follow-up (2009–2012) waves of the Outcomes of Sleep Disorders in Older Men Study (an ancillary study to the Osteoporotic Fractures in Men (MrOS) Study conducted in 6 US communities). Among men with a baseline nighttime sleep duration of 5–8 hours, we assessed the odds of becoming a short-duration (<5 hours) or long-duration (>8 hours) sleeper at follow-up. The odds of becoming a short-duration sleeper were higher among men with peripheral vascular disease (adjusted odds ratio (aOR) = 6.54, 95% confidence interval (CI): 2.30, 18.55) and ≥1 impairment in Instrumental Activities of Daily Living (IADL) (aOR = 2.57, 95% CI: 0.97, 6.78). The odds of becoming a long-duration sleeper were higher among those with greater baseline age (per SD increment, aOR = 1.49, 95% CI: 1.12, 2.00), depression symptoms (aOR = 3.13, 95% CI: 1.05, 9.36), and worse global cognitive performance (per SD increment of Modified Mini-Mental State Examination score, aOR = 0.74, 95% CI: 0.58, 0.94). Peripheral vascular disease and IADL impairment, but not chronological age, may be involved in the etiology of short sleep duration in older men. The risk factors for long-duration sleep suggest that deteriorating brain health predicts elongated sleep duration in older men.

actigraphy; aging; chronic disease; longitudinal studies; sleep; sleep duration; sleep measures

Abbreviations: IADL, Instrumental Activities of Daily Living; MrOS, Osteoporotic Fractures in Men; PASE, Physical Activity Scale for the Elderly; SD, standard deviation; TNF-α, tumor necrosis factor α.

Changes in sleep duration may be both a cause and a consequence of specific chronic diseases among older adults. Evidence regarding the consequences of short and long sleep duration on cardiovascular disease, diabetes, and mortality has risen to the level of several meta-analyses (1–5). To date, however, very few epidemiologic studies have examined the specific determinants of changes in sleep duration among older adults (6–8).

Current literature (9, 10), including a systematic review of available longitudinal evidence (11), suggests that specific disease processes, rather than chronological aging itself, predict changes in sleep with aging. Establishing the determinants of objectively measured changes in sleep duration is particularly important for informing evidence-based recommendations regarding the amount of sleep needed across the life span, specifically by understanding what sleep-duration changes experienced by older adults could be signaling (i.e., a normative aging process vs. a pathogenic process). However, prior epidemiologic investigations of change in sleep duration among older adults have been limited by reliance on subjective (i.e., self-reported) measures (6–8), which may inaccurately reflect sleep duration due to systematic bias (12). Therefore, while changes in sleep duration may reflect or occur due to particular preexisting disease processes, longitudinal research

utilizing objective measures of sleep duration are needed to establish the specific health factors which predict changes in sleep duration with aging.

We performed a longitudinal study evaluating changes in objectively measured sleep duration over a period of 6 years in a large sample of community-dwelling men. Further, we tested whether the changes observed were predictable based on chronological age or antecedent heath factors. We selected a comprehensive set of physical (7, 13–20) and mental health (7, 13–16, 18–22) factors previously associated with sleep in aging. Sleep changes predict (23) and co-occur with (24) cognitive impairment among older adults; therefore, we also tested whether baseline cognitive function predicts sleep duration changes. Finally, prior literature has demonstrated that high levels of proinflammatory markers like interleukin-6 are associated with future self-reported long sleep duration (6) and that other cytokines, such as tumor necrosis factor α (TNF-α), have known roles in sleep regulation (25–27); we therefore also examined whether a panel of these and other cytokines were associated with changes in sleep duration.

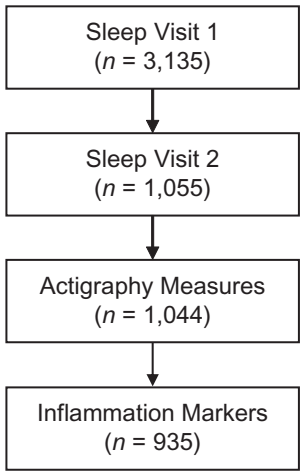

Figure 1. Selection of the analytical sample from the Outcomes of Sleep Disorders in Older Men Study, an ancillary study of the Osteoporotic Fractures in Men (MrOS) Study. The sleep study was conducted in 6 US communities between 2003 and 2012. Sleep visit 1 took place in 2003–2005, and sleep visit 2 took place in 2009–2012.

METHODS

Participants

The Outcomes of Sleep Disorders in Older Men Study (the MrOS Sleep Study) was conducted at 6 US sites (Birmingham, Alabama; Minneapolis, Minnesota; Palo Alto, California; the Monongahela Valley, near Pittsburgh, Pennsylvania; Portland, Oregon; and San Diego, California) in 2003–2005 as an ancillary study to the Osteoporotic Fractures in Men (MrOS) Study. The baseline study visit (sleep visit 1; $n = 3,135$) took place at that time. More information on the MrOS parent study (28, 29) and details of the inclusion/exclusion criteria for sleep visit 1 (30) have been published previously. From 2009 to 2012, participants were recontacted and invited to participate in sleep visit 2 (target $n = 1,000$), and a total of 1,055 participants returned for this second sleep visit (Figure 1). Of sleep visit 2 participants, usable actigraphy data were collected from 1,044. Of the men with ActiGraph (ActiGraph LLC, Pensacola, Florida) measurements at both sleep visits, 935 had fasting serum collected as part of the ancillary cytokine study conducted at sleep visit 1. The study protocol was approved by the institutional review boards at each site, and written informed consent was obtained from all participants.

Sleep assessments

At sleep visit 1, participants were asked to wear the ActiGraph Octagonal Sleep Watch (SleepWatch-O; Ambulatory Monitoring, Inc., Ardsley, New York) on the nondominant wrist for a minimum of 5 consecutive 24-hour periods, except when bathing or engaging in water sports. However, this model of the device was unavailable when sleep visit 2 was initiated; therefore, the Actiwatch 2 (Respironics, Inc., Bend, Oregon) was utilized. A comparability study determined that, when similar option settings were used to define sleep latency and sleep offset (sleep visit 1: proportional integration mode data from SleepWatch-O; sleep visit 2: data from the low sensitivity threshold of Actiwatch 2), data from the two models of ActiGraphs used at sleep visit 1 and sleep visit 2 were comparable for measurement of total sleep time (31).

Participants were asked to keep a sleep log, including times of sleep onset and offset, which was used to edit ActiGraph data. Sleep onset was defined as the point at which the participant achieved sleep for 20 continuous minutes after getting into bed, and total sleep duration was calculated using an automated algorithm as described previously (32). Nighttime sleep duration at baseline was defined as number of hours per night spent sleeping in bed after "lights off" and was categorized as <5 hours (short), 5–8 hours (typical), or >8 hours (long). To examine incident cases of short (<5 hours) and long (>8 hours) sleep duration, analyses of change in sleep duration were conducted only among participants who had 5–8 hours' sleep per night at baseline. These cutpoints were selected to enable analysis of changes from normative sleep to both shortened and prolonged sleep while capturing clinically significant changes in sleep duration.

Predictors of change in sleep duration

Demographic and lifestyle factors. Participants completed questionnaires that included collection of information on demographic factors, education, alcohol use (number of drinks consumed per week), smoking history, and physical activity (the Physical Activity Scale for the Elderly (PASE) (33)).

Mental and physical health. Geriatric Depression Scale-15, a validated short form of the Geriatric Depression Scale (34–36) for screening for major depressive disorder among older persons (37), was administered, and a standard cutpoint of ≥6 was used to define clinically significant depression symptoms; this cutoff yields a sensitivity of 90.9% and a specificity of 64.5% in comparison with a *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*, diagnosis of major depression (36). The Goldberg Anxiety Scale with a cutoff of ≥5 was used to determine the presence of clinically

significant anxiety symptoms. This was originally validated with a sensitivity and specificity of 86% and 91%, respectively (38). Cognitive function was measured using a global test of cognitive function (Teng and Chui's (39) Modified Mini-Mental State Examination), as well as the Digit Vigilance Test (40) and Trail Making Test Part B (41).

Information on self-reported health status (excellent/good vs. fair/poor/very poor) was obtained. Usual gait speed was assessed using a 6-m walk. The presence of impairment in Instrumental Activities of Daily Living (IADL) was determined by asking participants whether they had difficulty with the following activities: heavy housework, preparing one's own meals, shopping for groceries or clothing, walking 2–3 blocks, and climbing 10 stairs (42, 43). Self-reported history of the following medical diagnoses was also obtained: rheumatoid arthritis, osteoarthritis, cataracts, stroke, Parkinson disease, diabetes mellitus, chronic obstructive pulmonary disease, hypertension, peripheral vascular disease, angina, and myocardial infarction. Participants were asked to bring all current medications used within the last 30 days with them to sleep visit 1; ingredients were coded to active ingredients using a coding dictionary (44). Medications considered in the analyses included antidepressants, benzodiazepines, and other (nonbenzodiazepine) sedatives/hypnotics.

Inflammatory markers. Serum was collected during morning clinic visits after an overnight fast. A natural log transformation was applied to normalize their data distributions, which were initially skewed. C-reactive protein was measured using the enzyme-linked immunosorbent assay kit from American Laboratory Products Company (Salem, New Hampshire). Interleukin-6, TNF-α, and interferon-γ were assayed using the Human ProInflammatory I 4-Plex Ultra-Sensitive Kit by Meso Scale Discovery (catalog no. K15009C-4; Meso Scale Diagnostics LLC, Rockville, Maryland). TNF-α soluble receptor II was measured with an enzyme-linked immunosorbent assay from R&D Systems (catalog no. DRT200; R&D Systems, Inc., Minneapolis, Minnesota). Interassay coefficients of variation for these markers have been published previously (45).

Statistical analysis

All continuous predictor variables were standardized except those categorized to represent clinically significant categories as above. Multinominal logistic regression models were used to examine associations between all baseline predictors and short (<5 hours) and long (>8 hours) sleep duration at follow-up (compared with participants remaining in the 5- to 8-hour sleep duration range). To further ensure that any associations detected were independent of baseline variability in sleep duration, we adjusted all model results for baseline sleep duration expressed continuously. We first examined age- and site-adjusted associations between all predictors and the odds of change in sleep duration using separate multinominal logistic regression models. We then created a multivariable-adjusted model by entering only predictor variables that met the standard of crude significance at $P < 0.10$ with a Benjamin-Hochberg (46) false-discovery rate of $q < 0.10$ (to protect against false-positive findings due to the large number of predictor variables examined). To examine whether other variables that did not meet this stringent criterion represented potential independent contributors to variability in sleep-duration changes, we performed a sensitivity analysis adding all bivariate associations meeting the $P < 0.10$ standard to the multivariable model described above.

RESULTS

Baseline demographic and health characteristics by baseline sleep duration are presented in Web Table 1 (available at http://aje.oxfordjournals.org/) for descriptive purposes. The average time from sleep visit 1 to sleep visit 2 (among all 1,044 men) was 6.5 years. Among men with a baseline sleep duration of 5–8 hours ($n = 865$), 89% remained 5- to 8-hour sleepers at follow-up, 3.6% ($n = 31$) became short (<5 hours) sleepers, and 7.4% ($n = 64$) became long (>8 hours) sleepers. The mean and median changes in sleep duration among those becoming short sleepers (mean = −82.7 (standard deviation (SD), 36.3) minutes; median, −76 minutes) and long sleepers (mean = 78.8 (SD, 42.3) minutes; median, 69 minutes) represented substantial/clinically significant changes, while the vast majority of men remained 5- to 8-hour sleepers and had an average change in sleep duration of 1.98 (SD, 46.0) minutes at follow-up (median, 1 minute).

Demographic/lifestyle predictors of change in sleep duration

In base models, age was associated with higher odds of becoming a long-duration sleeper but not a short-duration sleeper (odds ratios are shown in Table 1). No other demographic/lifestyle factor met the criteria for being a significant predictor of change in sleep duration, although greater physical activity (measured with the PASE) was associated with reduced odds of becoming a short sleeper.

Physical and mental health predictors of change in sleep duration

Men with clinically significant depressive symptoms (measured with Geriatric Depression Scale-15) at baseline had higher crude odds of becoming a long sleeper, while better global cognitive performance (higher scores on the Modified Mini-Mental State Examination) and faster gait speed were associated with lower odds of becoming a long sleeper (Table 2). In these base models, the presence of any IADL impairment and peripheral vascular disease were associated with higher odds of becoming a short sleeper. Several other associations met the traditional standard of significance but not the multiple-comparison-corrected significance level: Faster gait speed was associated with lower odds of becoming a short sleeper, having "good/fair" perceived health was associated with lower odds of becoming a long sleeper, and both stroke and Parkinson disease were associated with greater odds of becoming a long sleeper. No other mental or physical health variables were significantly associated with change in sleep duration.

Table 1. Demographic and Lifestyle Predictors of Change in Sleep Duration Among Older[a] Men ($n = 1,055$) in the Outcomes of Sleep Disorders in Older Men Study (Sleep Visit 1; 2003–2005)[b]

Characteristic	Total Sleep Time							
	Short (<5 Hours) ($n = 31$)				Long (>8 Hours) ($n = 64$)			
	OR	95% CI	*P* Value	*q* Value	OR	95% CI	*P* Value	*q* Value
Age, years (per SD increment)	0.93	0.63, 1.38	0.7187	0.875	1.63	1.25, 1.13	0.0003	0.0102
White race/ethnicity	1.40	0.43, 4.53	0.5791	0.875	0.50	0.22, 1.13	0.097	0.2888
College/graduate education (per SD increment)	0.92	0.31, 2.70	0.8796	0.9475	0.52	0.25, 1.07	0.0746	0.2818
Alcohol intake, drinks/week								
0	1	Referent			1	Referent		
1–13	0.68	0.17, 2.80	0.5969	0.875	1.17	0.36, 3.80	0.7896	0.839
≥14	0.40	0.10, 1.54	0.1816	0.5287	0.70	0.23, 2.14	0.5332	0.6666
Smoking status (past/current vs. never)	1.35	0.63, 2.89	0.4463	0.875	1.58	0.89, 2.81	0.1189	0.2888
PASE score (per SD increment)	0.63	0.43, 0.94	0.0244	0.2237	0.91	0.67, 1.24	0.5478	0.6666

Abbreviations: CI, confidence interval; OR, odds ratio; PASE, Physical Activity Scale for the Elderly; SD, standard deviation.
[a] Mean age at baseline = 74.6 (standard deviation, 4.7) years.
[b] Results from crude models are shown (adjusted for age, site, and baseline sleep duration in minutes). The reference category was maintaining a sleep duration of 5–8 hours per night at sleep visit 2 (2009–2012).

Table 2. Physical and Mental Health Predictors of Change in Sleep Duration Between Sleep Visit 1 (2003–2005) and Sleep Visit 2 (2009–2012), Outcomes of Sleep Disorders in Older Men Study[a]

Characteristic	Total Sleep Time							
	Short (<5 Hours) ($n = 31$)				Long (>8 Hours) ($n = 64$)			
	OR	95% CI	*P* Value	*q* Value	OR	95% CI	*P* Value	*q* Value
Geriatric Depression Scale score ≥6	2.53	0.67, 9.55	0.1698	0.5287	4.10	1.52, 11.02	0.0052	0.0536
Self-perceived good/excellent health	0.41	0.16, 1.04	0.0616	0.3491	0.41	0.18, 0.93	0.0324	0.1836
Goldberg Anxiety Scale score ≥5	2.22	0.77, 6.42	0.1400	0.5287	2.13	0.85, 5.35	0.1088	0.2888
Use of antidepressant medication	2.35	0.73, 7.54	0.1498	0.5287	1.68	0.63, 4.46	0.2983	0.5338
3MS score (per SD increment)	1.09	0.71, 1.67	0.6868	0.875	0.69	0.55, 0.86	0.001	0.0170
≥1 IADL impairments	3.78	1.59, 9.00	0.0026	0.0442	1.81	0.86, 3.78	0.1169	0.2888
Leg pain	1.75	0.78, 3.91	0.1765	0.5287	1.49	0.78, 2.84	0.2313	0.4626
Rheumatoid arthritis	1.32	0.30, 5.94	0.7148	0.875	1.34	0.51, 3.53	0.549	0.6666
Osteoarthritis	0.97	0.40, 2.35	0.9485	0.9485	1.37	0.72, 2.60	0.3361	0.5442
Cataracts	1.20	0.55, 2.62	0.6471	0.875	0.72	0.40, 1.30	0.2776	0.5244
Stroke	2.14	0.25, 18.3	0.4866	0.875	3.78	1.22, 11.7	0.0216	0.1469
Parkinson disease	6.20	0.34, 112.8	0.2177	0.5287	10.7	1.12, 99.9	0.0397	0.1928
Diabetes	1.23	0.40, 3.73	0.7206	0.875	1.91	0.88, 4.13	0.1013	0.2888
Chronic obstructive pulmonary disease	1.11	0.14, 9.01	0.9196	0.9475	1.71	0.46, 6.35	0.4196	0.5944
Hypertension	1.62	0.77, 3.42	0.2041	0.5287	1.15	0.66, 2.00	0.6149	0.6969
Peripheral vascular disease	7.22	2.59, 20.1	0.0002	0.0068	1.81	0.74, 4.40	0.192	0.408
Angina	1.33	0.44, 4.01	0.6172	0.8750	1.25	0.56, 2.76	0.5883	0.6897
Myocardial infarction	1.38	0.49, 3.84	0.5413	0.8750	1.13	0.54, 2.39	0.7447	0.8168
Use of benzodiazepine	0.88	0.11, 6.99	0.9020	0.9475	0.34	0.04, 2.81	0.317	0.5389
Digital Vigilance Test score (per SD increment)	1.13	0.78, 1.64	0.5068	0.8750	1.12	0.86, 1.47	0.3917	0.5821
Trail Making Test Part B score (per SD increment)	1.24	0.89, 1.74	0.2016	0.5287	1.12	0.86, 1.46	0.3938	0.5821
Gait speed (per SD increment)	0.64	0.43, 0.96	0.0329	0.2237	0.63	0.46, 0.88	0.0063	0.0536

Abbreviations: CI, confidence interval; IADL, Instrumental Activities of Daily Living; 3MS, Modified Mini-Mental State Examination; OR, odds ratio; SD, standard deviation.
[a] Results from crude models are shown (adjusted for age, site, and baseline sleep duration in minutes). The reference category was maintaining a sleep duration of 5–8 hours per night at sleep visit 2.

Inflammatory marker predictors of change in sleep duration

None of the inflammatory markers examined were significantly associated with change in sleep duration by multiple-comparison-corrected criteria (Table 3). In the base models, however, higher levels of interleukin-6 were associated with greater odds of becoming a short sleeper, and higher levels of TNF-α were associated with greater odds of becoming a long sleeper ($P = 0.06$).

Multivariable model

The base associations described above meeting criteria for multiple-comparison-adjusted statistical significance were entered into a multivariable model. The magnitude and significance of these associations were not substantively altered, except for the association of walking speed with future long sleep duration, which was completely attenuated by multivariable adjustments (Table 4).

Adding to the multivariable model other associations that achieved bivariate $P < 0.10$ (no false discovery rate criteria) altered some but not all of these associations. The associations of age and global cognitive function with increased odds of becoming a longer sleeper were not substantively altered (Web Table 2). However, in this model, the association of depression with future long sleep was attenuated and failed to retain statistical significance (adjusted odds ratio = 1.97, 95% confidence interval: 0.57, 6.82). None of the additional covariates were significantly associated with becoming a long sleeper. The associations of IADL impairment and peripheral vascular disease with becoming a short sleeper were not substantively altered in the expanded final model, and the only additional factor to achieve a statistically significant association with becoming a short sleeper was physical activity level (higher PASE scores were associated with a lower likelihood of becoming a short sleeper; adjusted odds ratio = 0.54, 95% confidence interval: 0.33, 0.89).

DISCUSSION

Over more than 6 years of follow-up, among men with 5–8 hours' sleep per night at baseline, we found, using objective measures of sleep duration, that 3.6% of the participants developed short sleep duration and 7.4% developed long sleep duration. The mean continuous level of change among men with incident changes in sleep duration indicates that, on average, becoming a short sleeper and becoming a long sleeper alike represented substantial change.

To our knowledge, our study is the first to identify potential risk factors for the development of objectively assessed changes in sleep duration in a community-based sample of older adults. Our findings suggest that aging itself does not cause reductions in the duration of nighttime sleep and that peripheral vascular disease, IADL impairment, reduced physical activity, and inflammation may contribute to the etiology of short sleep in older men. Peripheral vascular disease was the strongest predictor of the likelihood of becoming a short sleeper, although age itself was not; in base models, the presence of IADL impairment, lower levels of physical activity, and greater levels of interleukin-6 also significantly predicted a greater likelihood of becoming a short sleeper. Future research should target these factors to further elucidate the specific mechanism(s) underlying the risk of shortening sleep in this population. The association of peripheral vascular disease with future short sleep was particularly strong (i.e., in terms of effect size); therefore, this predictor may be of clinical use for the early identification of men at risk of short sleep duration and related consequences (e.g., effects on metabolic/cardiovascular health).

Age and worse cognitive performance were both independently associated with becoming a long-duration sleeper. These findings are consistent with chronological aging's being related to elongation of nighttime sleep; alternatively, however, unmeasured disease/biological process may account for the detected association between age and long sleep duration. Interestingly, all 3 of the predictors we observed (age, depression, and cognition) to increase the likelihood of becoming a long sleeper have been previously associated with worse brain

Table 3. Inflammatory Marker Predictors of Change in Sleep Duration Between Sleep Visit 1 (2003–2005) and Sleep Visit 2 (2009–2012), Outcomes of Sleep Disorders in Older Men Study[a]

Characteristic	Total Sleep Time							
	Short (<5 Hours) ($n = 27$)				Long (>8 Hours) ($n = 58$)			
	OR	95% CI	*P* Value	*q* Value	OR	95% CI	*P* Value	*q* Value
TNF-α, pg/mL[b]	1.06	0.73, 1.54	0.7524	0.8821	1.35	0.99, 1.85	0.0621	0.2639
IL-6, pg/mL[b]	1.43	1.04, 1.99	0.0301	0.2237	1.22	0.91, 1.63	0.1903	0.4080
CRP, μg/mL[b]	0.81	0.55, 1.21	0.3028	0.6863	0.90	0.66, 1.23	0.5028	0.6666
IFN-γ, pg/mL[b]	0.97	0.63, 1.48	0.8788	0.9475	0.99	0.74, 1.34	0.9810	0.9810
TNF-α-sRII, pg/mL	1.19	0.83, 1.70	0.3419	0.7265	1.01	0.75, 1.36	0.9493	0.9781

Abbreviations: CI, confidence interval; CRP, C-reactive protein; IFN-γ, interferon γ; IL-6, interleukin-6; OR, odds ratio; TNF-α, tumor necrosis factor α; TNF-α-sRII, tumor necrosis factor α soluble receptor 2.

[a] Results from crude models are shown (adjusted for age, site, and baseline sleep duration in minutes). All ORs show the odds of a change in sleep duration per standard-deviation increment in the cytokine levels. The reference category was maintaining a sleep duration of 5–8 hours per night at sleep visit 2.

[b] Log-transformed.

Table 4. Results From the Final Multivariable-Adjusted Model of Change in Sleep Duration Between Sleep Visit 1 (2003–2005) and Sleep Visit 2 (2009–2012), Outcomes of Sleep Disorders in Older Men Study[a]

Characteristic	Total Sleep Time					
	Short (<5 Hours) ($n = 31$)			Long (>8 Hours) ($n = 62$)		
	OR	95% CI	*P* Value	OR	95% CI	*P* Value
Age, years (per SD increment)	0.80	0.53, 1.22	0.3019	1.49	1.12, 2.00	0.0071
Geriatric Depression Scale score ≥6	1.62	0.38, 6.86	0.5123	3.13	1.05, 9.36	0.0411
3MS score (per SD increment)	1.29	0.80, 2.08	0.2973	0.74	0.58, 0.94	0.0135
≥1 IADL impairments	2.57	0.97, 6.78	0.0577	1.24	0.54, 2.83	0.6090
Peripheral vascular disease	6.54	2.30, 18.6	0.0004	1.57	0.64, 3.86	0.3220
Gait speed (per SD increment)	0.68	0.43, 1.07	0.0956	0.85	0.60, 1.21	0.3641

Abbreviations: CI, confidence interval; IADL, Instrumental Activities of Daily Living; 3MS, Modified Mini-Mental State Examination; OR, odds ratio; SD, standard deviation.

[a] Results were also adjusted for site and baseline sleep duration in minutes. The reference category was maintaining a sleep duration of 5–8 hours per night at sleep visit 2.

structural health (47–51). Therefore, these predictors of future long sleep may be markers of worse brain structural health, although future research is needed to establish whether and what type of brain structural pathology might cause older persons' sleep to become prolonged.

We also found that although gait speed predicted future long sleep duration in the bivariate model, this association was completely attenuated by adjustments in the first multivariable model. This suggests that the association between gait speed and the development of long sleep duration can be explained by age, depression, or cognitive performance (i.e., factors included in the multivariable model in Table 4 that are markers of brain health). Consistent with this interpretation, a previous study found that overt cerebrovascular disease (i.e., stroke) was related to the future development of long sleep duration (8), and our crude analysis replicated this association. In addition, the association between clinically significant depression symptoms with future long sleep duration was attenuated to nonsignificance in the final, inclusive multivariable model. This suggests that depression may mark an increased likelihood of becoming a future long sleeper, and that the underlying pathogenic processes by which depression and the other physical health factors considered (e.g., stroke or specific cytokine levels) affect sleep duration may overlap. Finally, in base models, we found that TNF-α levels predicted higher odds of becoming a long-duration sleeper, potentially by directly increasing sleep propensity (25), by altering sleep architecture towards deeper sleep (26), or via an association with brain structural damage (52). Future research is needed to establish whether these risk markers cause prolonged sleep—for example, due to a decreased ability of the brain to efficiently produce restorative sleep or to dissipate enhanced homeostatic drive. Future research is also needed to establish whether, in this context, prolonged sleep duration serves a therapeutic or toxic process.

What we did *not* observe is also worth noting. Diabetes and cardiovascular disease, which have been recognized as potential consequences of both short and long sleep duration (1, 4, 5), were not related to changes in sleep duration. These observations may help to exclude the potential role of reverse causality by preexisting metabolic/cardiovascular factors' causing changes in sleep duration. However, this conclusion must be tempered by the fact that we did not include a comprehensive assessment of subclinical measures of metabolic/cardiovascular disease that may contribute to changes in sleep duration.

Several additional limitations should be noted. We corrected for multiple comparisons to minimize false-positive findings, but there may be a risk for false-negative findings, given that our sample and number of events were relatively small. Our analysis did not include some potentially important determinants of future sleep, potentially including other biological (e.g., cerebrovascular disease) or psychosocial (e.g., sleeping environment) factors; furthermore, we focused on baseline predictors of change in sleep, although changes occurring between the study visits (e.g., changes in medication use) may also explain changes in sleep duration. In addition, sleep health is multidimensional (53), and future research is needed to address the determinants of changes in other aspects of sleep (e.g., sleep fragmentation or napping) and how changes in different aspects of sleep interrelate (e.g., how sleep electrophysiology or quality changes among men who became long sleepers). Finally, our sample was restricted to older men who were mostly white and who survived through and were willing to partake in multiple objective sleep assessments; therefore, these participants may have been healthier than the general population, and our findings may have underestimated the true rates of changes in sleep duration among older adults.

Despite these limitations, and acknowledging that these findings need to be replicated other samples, our study had several strengths surpassing the available literature. Using repeated sleep measures enabled us to stratify analyses to participants without a short or long sleep duration at baseline and to adjust for levels of baseline sleep parameters; these methodological advantages over past studies (e.g., the work of Dowd et al. (6)) ensured that the associations between baseline predictors and sleep-duration changes were independent of initial sleep duration. Importantly, we also used objective measures of sleep, which adds greater assessment reliability to current

epidemiologic literature on sleep changes in aging, which had previously been limited to self-reported sleep duration. Finally, we performed a comprehensive assessment of lifestyle/clinical factors and adjusted for multiple comparisons to reduce the chances of false-positive findings.

In conclusion, rather than chronological aging itself affecting sleep, aging-related increases in the prevalence of identified diseases that predict changes in sleep duration may explain why sleep duration changes during aging. We found that the predictors of shortening and elongating sleep in aging men differed markedly, and our observations suggest potential mechanisms by which sleep duration changes in these directions. In future research, investigators should target the identified factors to further elucidate the pathophysiology and pathogenesis of changes in sleep duration. Peripheral vascular disease may be the most robust risk factors for future short sleep duration. Long sleep duration may result from aging-related disease processes affecting central nervous system integrity. Future intervention studies are needed to test whether modifying these risk factors reduces changes in sleep duration (and related consequences), and in trials targeting these risk factors for other purposes, researchers should consider monitoring sleep as a secondary outcome.

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