Behavioral Correlates of Sleep-Disordered Breathing in Older Men

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Study Objectives: To examine the association between sleep-disordered breathing (SDB) and subjective measures of daytime sleepiness, sleep quality, and sleep-related quality of life in a large cohort of community-dwelling older men and to determine whether any association remained after adjustment for sleep duration.

Design: Cross-sectional. The functional outcome measures of interest were daytime sleepiness (Epworth Sleepiness Scale, ESS), sleep-related symptoms (Pittsburgh Sleep Quality Index, PSQI), and sleep-related quality of life (Functional Outcomes of Sleep Questionnaire, FOSQ). Analysis of variance and adjusted regression analyses examined the association between these outcome measures and SDB severity and actigraphy-determined total sleep time (TST). We then explored whether associations with SDB were confounded by sleep duration by adjusting models for TST.

Setting: Community-based sample in home and research clinic settings.

Participants: Two-thousand eight-hundred forty-nine older men from the multicenter Osteoporotic Fractures in Men Study that began in 2000. All participants underwent in-home polysomnography for 1 night and wrist actigraphy for a minimum of 5 consecutive nights.

Interventions: N/A.

Measurements and Results: Participants were aged 76.4 ± 5.5 years and had an apnea-hypopnea index (AHI) of 17.0 ± 15.0. AHI and TST were weakly correlated. ESS scores individually were modestly associated with AHI and TST, but the association with AHI was attenuated by adjustment for TST. PSQI and FOSQ scores were largely not associated with measures of SDB severity but were modestly associated with TST.

Conclusions: Daytime sleepiness, nighttime sleep disturbances, and sleep-related quality of life were modestly associated with TST. After adjustment for TST, there was no independent association with SDB severity. These results underscore the potential differences in SDB functional outcomes in older versus young and middle-aged adults.

Keywords: sleep-disordered breathing, obstructive sleep apnea, older adults, sleepiness, quality of life

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THE PREVALENCE OF SLEEP-DISORDERED BREATHING (SDB) IN OLDER ADULTS IS HIGHER THAN IN MIDDLE-AGED ADULTS. ESTIMATES OF SDB IN MIDDLE-aged adults are generally less than 10%,[1] whereas side-by-side comparison studies confirm a higher prevalence of SDB in older adults compared to the middle-aged.[2-6] In a population of adults aged 65 to 95 years, Ancoli-Israel et al. reported a SDB prevalence (apnea-hypopnea index, or AHI, ≥ 20) of 39% in women and 51% in men.[7] In an older sample of community-dwelling women, Kezirian et al estimated a prevalence of SDB (AHI ≥ 15) of 24%.[8] In a community-dwelling sample of older men aged 65 and older, Mehra et al reported an SDB prevalence (AHI ≥ 15) of 26%.[9]

In spite of the higher prevalence of SDB among older adults, the functional consequences have not been clearly established. In young and middle-aged adults, SDB has been associated with daytime somnolence, sleep-related symptoms, and decrements in sleep-related quality of life.[10] Large population-based studies of older adults initially showed an association between SDB severity and daytime sleepiness.[7, 11-13] However, age-related comorbidities and medication use are associated with sleep disruption,[14-16] and sleep disruption may itself contribute to daytime symptoms. In older women, associations between SDB and both self-reported daytime sleepiness and sleep-related quality of life have been largely explained by short sleep duration.[8] It is therefore important to consider the independent associations of SDB and sleep duration with functional consequences in studies of older adults.

The objectives of this study were (1) to examine the association between SDB severity and daytime sleepiness, sleep-related symptoms, and sleep-related quality of life in a large cohort of primarily community-dwelling older men and (2) to determine whether any associations were influenced by adjustment for sleep duration (specifically total sleep time [TST], determined by actigraphy). Based on data from young and middle-aged adults and our previous research in older women, we hypothesized that SDB severity would be associated with daytime sleepiness, sleep disturbances, and poorer sleep-related quality of life but that these associations would be attenuated by adjustment for TST.

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METHODS

Population

Participants were from the Osteoporotic Fractures in Men (MrOS) Study, a multicenter prospective cohort study of 5995 primarily white men aged 65 years or older that began in 2000. Details of the MrOS Study have been described previously.[17,18]

From 2003 to 2005, a sample (n = 3135) of older men from the parent MrOS Study, including all study sites, underwent in-home polysomnography and wrist actigraphy in the MrOS Sleep Study.[9] Men were screened for use of mechanical devices during sleep, including pressure mask for sleep apnea (continuous positive airway pressure or bilevel positive airway pressure), mouthpiece for snoring or sleep apnea, or oxygen therapy. In general, those who reported nightly use of any of these devices were excluded from the MrOS Sleep Study; however, the study sample includes 49 men who reported use of 1 of these devices. Seventeen men were able to forego use of their sleep devices during the night of the in-home polysomnography study. The 2849 men with acceptable polysomnography and actigraphy data who had no current treatment for SDB comprised the sample population. All study participants were evaluated with a clinic interview, anthropometric measurements, performance measures, and actigraphy. Men who reported regular use of mechanical devices during sleep including positive airway pressure therapy, open tracheostomy tube, oral appliances for SDB, or oxygen therapy were excluded from this analysis (n = 150). This study was approved by the institutional review board of each involved institution.

Polysomnography

In-home polysomnography data were collected using the Compumedics Safiro Unit (Melbourne, AU). The recording montage consisted of C3/A2 and C4/A1 electroencephalograms, bilateral electrooculograms, a bipolar submental electromyogram, thoracic and abdominal respiratory inductance plethysmography, airflow (using nasal-oral thermocouple and nasal pressure cannula), finger pulse oximetry, electrocardiogram, body position (mercury switch sensor), and bilateral leg movements (piezoelectric sensors). Sleep data were scored centrally by certified scorers blinded to other data. Sleep stages and arousals were scored using standard criteria.[19] Apneas were defined as a complete or almost complete cessation of airflow (by thermocouple) associated with at least a 3% oxygen desaturation, and hypopneas were identified as a clearly discernible (at least 30%) reduction in respiratory sensor channels associated with at least a 3% oxygen desaturation. Apneas associated with no evidence of effort on both thoracic and abdominal channels were considered to be "central" and otherwise as "obstructive." Arousals were defined as an abrupt shift in electroencephalographic frequency of 3 seconds or more and requiring an increase in chin electromyographic activity if occurring during rapid eye movement sleep.[20]

Calculated variables used as indexes of SDB severity were AHI (apneas plus hypopneas per hour of sleep), hypopnea index (HI, number of hypopneas per hour of sleep), obstructive apnea index (OAI, number of obstructive apneas per hour of sleep), central apnea index (CAI, number of central apneas per hour of sleep), arousal index (ArI, number of arousals per hour of sleep), and the percentage of sleep time with oxygen saturation below 90% (Sao_2<90%). AHI was considered a continuous variable as well as a categorical variable based on tertiles. The Sao_2<90% was markedly skewed and therefore was considered as a binary exposure based (< 2% vs ≥ 2% time spent at < 90% saturation). Wakefulness after sleep onset was defined as the minutes awake during the sleep period after sleep onset (the first 2 continuous minutes scored as sleep). All other polysomnography variables were evaluated as continuous variables.

Polysomnography data quality was excellent, with a failure rate of less than 4% and more than 70% of studies graded as being of excellent or outstanding quality. Quality codes for signals and studies were graded using previously described approaches, which included coding the duration of artifact-free data per channel and overall study quality (reflecting the combination of grades for each channel).[21]

Actigraphy

Actigraphy was used to record wrist activity from which sleep/wake was calculated. Actigraphy allows recordings for multiple nights, which reduces the night-to-night variability and captures daytime napping behavior.[22] Wrist actigraphy (Sleep-Watch-O, Ambulatory Monitoring, Inc., Ardsley, NY) data were collected for a minimum of 4 consecutive 24-hour periods, ie, 120 hours (mean recording time, 113.6 ± 19.2 hours; mean number of nights, 5.2 ± 0.9). Data collected in the proportional integration mode were used for this analysis because this mode has shown the highest correlation with polysomnography-determined sleep time in a population of older women.[23] TST was calculated as the mean night sleep time (defined as sleep time within the reported period spent in bed) averaged over all nights. Napping time was calculated as the mean daytime inactivity (defined as inactivity time from the time out of bed in the morning to the time to bed at night) averaged over all nights. The time periods the participants wore the actigraphs were divided into "up" intervals, which are defined as the time the participant reported being out of bed, and "down" intervals, which are defined as the time period the participant was in bed. The "up" intervals must have been bounded by 2 "down" intervals to be used in the summaries. Actigraphy monitoring included 1 night that overlapped polysomnography in 909 participants. In these studies performed concurrently, the correlation for TST was $r = 0.61$ ($P < 0.0001$).

Functional Outcome Measures

Daytime sleepiness was quantified using the Epworth Sleepiness Scale (ESS). The ESS measures sleep propensity on a 0- to 3-point scale in 8 standardized daily situations. Possible scores range from 0 to 24, and higher scores reflect greater sleepiness.[24] ESS score was considered as a continuous variable. Since the ESS includes a question related to driving, which may not be as applicable to all older adults, the ESS was considered with and without inclusion of this question; the modified ESS score has a possible score range from 0 to 21.

Subjective sleep symptoms, disturbances, and patterns were assessed with the Pittsburgh Sleep Quality Index (PSQI), an instrument validated for use in older populations.[25,26] The PSQI

measures a broad range of symptoms of sleep disturbances over a 1-month period. Responses to 19 questions are categorized into 7 component scores (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction); these are then combined to create 1 global score.[27] The total global score ranges from 0 to 21, and greater scores indicate higher levels of sleep symptoms. PSQI score was considered as a continuous variable.

Sleep-related quality of life was evaluated with the Functional Outcome of Sleep Questionnaire (FOSQ) a 30-item instrument that measures the effect of excessive daytime sleepiness on activities of daily living[28] that has been validated in older audults.[29] Mean-weighted item scores are used to generate 5 subscales (activity level, vigilance, intimacy and sexual relationships, general productivity, and social outcome) that together produce a composite score. The total score ranges from 5 to 20, and lower scores indicate greater dysfunction. The FOSQ score was analyzed as a continuous variable.

Covariates

Study participants completed anthropometric evaluation and questionnaire assessment of medical history and brief physical examination at each study visit. For this analysis, the following variables were included: age, race, body mass index (BMI; kg/m^2), Geriatric Depression Scale (GDS) score (range 0-15),[30] Goldberg Anxiety Scale score (range 0-9),[31] and self-reported health status. Self-reported health status was scaled as excellent, good, fair, poor, or very poor; the responses were treated as an ordinal variable, with the responses converted to a whole number from 1 to 4 after collapsing the categories poor and very poor (excellent, 1; poor/very poor, 4) in regression analysis.

Statistical Analysis

Statistical analyses were performed using SAS (SAS Institute, Cary, NC) software. Generalizability of the results from this analysis cohort of 2849 to the remainder of the MrOS cohort who were alive at the time of the sleep visit (n = 2802) was examined by calculating mean values (with standard deviations where applicable) for age, race, BMI, GDS score, Goldberg Anxiety Scale score, self-reported health status, TST, and the functional measures (ESS, PSQI, and FOSQ scores). Student t-tests (continuous variables), Wilcoxon rank sum tests (continuous variables with skewed distribution), and χ^2 tests (categorical variables) were used to compare the values across groups. Similar calculations were then performed after dividing the analysis cohort into AHI tertiles, using analysis of variance for normally distributed data and Kruskal-Wallis tests for skewed continuous data for comparison across groups.

To test for an association between sleep time and SDB severity, the Pearson correlation coefficient (assuming a normal distribution of the variables) and Spearman rank correlation coefficient (accounting for nonnormal distribution) were calculated for TST and each measure of SDB severity. Short sleep time was defined by a TST less than 360 minutes, and the prevalence of short sleep time across AHI groups defined by commonly used cutpoints was compared using a χ^2 test for trend. To assess for an association between sleep fragmentation and SDB severity, the Spearman rank correlation coefficient was calculated for wakefulness after sleep onset and TST.

One-way analysis of variance was used to consider the variation in outcome measures among groups separately defined either by SDB severity (using AHI tertiles) or by TST (using tertiles) without adjustment for other variables.

Multiple linear regression analysis was performed to determine the differences in ESS, PSQI, and FOSQ scores associated with measures of SDB severity. Coefficient estimates are reported with 95% confidence intervals. All regression models were adjusted for age, race, BMI, GDS score, Goldberg Anxiety Scale score, and self-reported health status. To determine the influence of TST on the observed associations between SDB severity and the outcome measures, each model was tested separately with and without adjustment for TST. Data are reported for actigraphy-determined TST, but regression models were evaluated with substitution of polysomnography-determined TST and additional adjustment for wakefulness after sleep onset. P values less than 0.05 were considered statistically significant in all analyses.

Power analysis revealed that this sample had an 80% power to detect a mean difference of 0.48 in ESS score, 0.43 in PSQI score, and 0.20 in FOSQ score between the lowest and highest AHI tertiles.

RESULTS

Description of the Sample

The MrOS Sleep study enrolled 3135 participants, of which 2849 had acceptable polysomnography and actigraphy data with no current treatment for SDB. When compared with those remaining men who could have potentially participated in the sleep visit or did not meet our sample criteria at the sleep visit, the sample was slightly younger, had a lower proportion of African Americans, and had better self-reported health status. Compared with those men who did participant in the sleep visit but did not meet our criteria, our sample had lower GDS scores and slightly more TST (Table 1). There were no significant differences between those with and without sleep recordings in any other variables, including TST (derived from actigraphy) and ESS, PSQI, or FOSQ scores.

The distribution of SDB exposures is shown in Table 2. The distribution of the AHI was less than 5 (21.3%), 5 to less than 15 (35.4%), 15-30 (26.0%), and greater than 30 (17.3%). The range for AHI was 0 to 104.4, with the 25th percentile of 5.9 and 75th percentile of 23.9. One thousand eighty-three participants (38.0%) demonstrated at least 2% of sleep time with an Sao_2<90%.

Table 3 presents the covariates and TST for the entire study population and by AHI tertiles. Individuals with higher AHI levels were slightly older, had greater BMI and poorer self-reported health status, had higher GDS score, and had lower levels of average TST by actigraphy than men with lower AHI levels.

The Spearman rank correlation coefficient for AHI and TST was -0.15 (P < 0.0001), suggesting a weak correlation whereby higher AHI was associated with lower TST. There was also a weak association between AHI and napping time (Spearman rank correlation coefficient 0.06, P = 0.002). The Sao_2<90% was also weakly associated with napping time (data not shown).

Table 1—Comparison Between Analysis Cohort and Those Remaining MrOS Participants Who Were Alive at the Time of the Sleep Visit

Characteristic[a]	Analysis cohort n = 2849	Remaining MrOS cohort alive at time of sleep visit n = 2802	P Value
Age, y	73.0 ± 5.5	73.8 ± 6.0	< 0.0001
African American, no. (%)	95 (3.3)	130 (4.6)	0.01
BMI, kg/m^2	27.4 ± 3.7	27.4 ± 4.0	0.38
GDS score	1.8 ± 2.1	2.2 ± 2.6	0.02
GAS, score	1.0 ± 1.9	1.1 ± 1.9	0.33
Self-reported health status, no. (%)			
Excellent	1065 (37.4)	876 (31.3)	< 0.0001
Good	1464 (51.4)	1500 (53.5)	
Fair	293 (10.3)	374 (13.4)	
Poor or very poor	26 (0.9)	52 (1.9)	
ESS score	6.2 ± 3.7	6.2 ± 3.7	0.76
PSQI score	5.6 ± 3.3	5.6 ± 3.3	0.98
FOSQ score	18.7 ± 1.5	18.5 ± 2.0	0.67
Actigraphic TST, min	385.3 ± 73.4	371.0 ± 83.4	0.02

[a]**Note**: Data for age, body mass index (BMI), and self-reported health were taken from the baseline visit, approximately 3 years prior to the sleep visit. The remaining variables are only gathered at the sleep visit, so the summaries are on the 286 participants who were at the sleep visit but do not meet criteria for the analysis subset. For continuous variables, means are shown with standard deviation in parentheses.
P Values reflect the appropriate test of significance (Student t-test for continuous variables, Wilcoxon Rank Sum test for continuous variables with skewed distribution, and χ^2 test for categorical variables) to determine whether there is statistically significant variation among the apnea-hypopnea index tertiles.
Abbreviations: MrOS refers to the Outcomes of Sleep Disorders in Older Men study; GDS, Geriatric Depression Scale; GAS, Goldberg Anxiety Scale; ESS, Epworth Sleepiness Scale; PSQI, Pittsburgh Sleep Quality Index; FOSQ, Functional Outcomes of Sleep Questionnaire.

Table 2—Distribution of Sleep-Disordered Breathing Indexes

	Mean ± SD	Median	Interquartile Range
Apnea-hypopnea index	17.0 ± 15.0	12.5	5.9-23.8
Hypopnea index	9.6 ± 9.8	6.5	2.9-12.7
Obstructive apnea index	6.1 ± 9.3	2.5	0.4-7.9
Central apnea index	1.4 ± 3.9	0.2	0.0-1.0
Arousal index	23.6 ± 11.7	21.2	15.3-29.4
Sao_2 < 90%, % of total sleep time	4.2 ± 9.6	1.0	0.0-3.7

Thirty-one percent (889/2849) of older men had short sleep time (TST < 360 minutes), and the prevalence of short sleep time increased with AHI: 23% (140/608) for AHI less than 5; 29% (291/1007) for AHI 5 to less than 15; 35% (259/741) for AHI 15 to 30; and 40% (199/493) for AHI greater than 30 (χ^2-test for trend P < 0.0001). Wakefulness after sleep onset was associated with AHI (Spearman rank correlation coefficient 0.16, P < 0.0001). The mean difference in TST between actigraphy and polysomnography was 29.40 minutes, with actigraphy TST (that includes daytime napping) higher for all AHI tertile groups. The correlation between actigraphy and polysomnography TST was 0.61 for the entire cohort (0.66 for the lowest AHI tertile, 0.60 for the middle tertile, and 0.58 for the highest AHI tertile).

SDB, Sleep Duration, and Daytime Sleepiness

Thirteen percent (365/2849) of participants had an ESS of at least 10, a level commonly considered to be excessive sleepiness. In univariate analyses, a higher ESS score was associated with both higher AHI and lower TST when considered in isolation using 1-way analysis of variance (Tables 4 and 5). The difference in ESS mean scores between the highest and lowest tertiles of AHI and TST were 0.5 and 1.5, respectively. Similar results were obtained for the modified ESS score that did not include a driving question.

Multiple regression analysis (Table 6) showed a modest association between ESS and AHI, adjusting for age, race, BMI, GDS score, Goldberg Anxiety Scale score, and self-reported health status; an increase in AHI of 1 standard deviation (approximately 15 units) was associated with an ESS score 0.18 points higher, a difference that was statistically significant but small in absolute magnitude. After adjustment for TST, this relationship was attenuated; the point estimate decreased by 48% in magnitude and was no longer statistically significant. Similar relationships with ESS were observed for CAI and OAI, although the association between ESS and the CAI was not attenuated by adjustment for TST (data not shown).

In contrast, the unadjusted association between ESS score and TST was slightly stronger (as seen in Table 5) and persisted following adjustment for AHI (Table 6; Model 2); a 1–standard-deviation increase in TST (approximately 73 minutes) was associated with an ESS score 0.69 points lower. Similar associations between ESS and TST were seen in models including alternative indexes of SDB. The ESS score was not associated with the ArI or the dichotomous variable describing oxygen desaturation. Similar results were also obtained with the modified ESS score. Adjustment for napping time, including the addition of napping time to TST, had no substantive effect on these findings. Substitution of polysomnography-determined TST for

Table 3—Subject Characteristics by Level of AHI

	Total (N = 2849)	AHI Tertiles < 7.90 (n = 948)	7.90-< 18.89 (n = 949)	≥ 18.89 (n = 952)	P Value
Age, y	76.4 ± 5.5	76.2 ± 5.6	76.1 ± 5.5	76.8 ± 5.4	0.02
African American, no. (%)	95 (3.3)	27 (2.9)	38 (4.0)	30 (3.2)	0.35
BMI, kg/m^2	27.2 ± 3.8	26.0 ± 3.4	27.4 ± 3.6	28.2 ± 4.0	<0.0001
GDS score	1.8 ± 2.1	1.7 ± 2.1	1.6 ± 2.0	1.9 ± 2.2	0.002
GAS score	1.0 ± 1.9	1.0 ± 1.9	1.0 ± 1.9	1.0 ± 1.9	0.30
Self-reported health status, no. (%)					
Excellent	955 (33.5)	349 (36.8)	340 (35.8)	266 (28.0)	0.0001
Good	1521 (53.4)	490 (51.7)	484 (51.0)	547 (57.5)	
Fair	341 (12.0)	100 (10.6)	109 (11.5)	132 (13.9)	
Poor or very poor	31 (1.1)	9 (1.0)	16 (1.7)	6 (0.6)	
TST, min	385.0 ± 73.8	397.0 ± 65.8	388.4 ± 68.9	369.7 ± 83.0	<0.0001

Note: For continuous variables, means are shown with standard deviation in parentheses. P Values reflect the appropriate test of significance (1-way analysis of variance for continuous variables, Kruskal-Wallis test for continuous variables with skewed distribution, and χ^2 test for categorical variables) to determine whether there is statistically significant variation among the apnea-hypopnea index (AHI) tertiles. BMI refers to body mass index; GDS, Geriatric Depression Scale; GAS, Goldberg Anxiety Scale; TST, total sleep time.

Table 4—Variation in Sleepiness and Functional Outcome Measures by AHI

	AHI Tertiles < 7.90	7.90 - < 18.89	≥ 18.89	P Value
ESS	5.9 (3.6)	6.0 (3.6)	6.4 (3.8)	0.005
PSQI	5.6 (3.3)	5.6 (3.2)	5.7 (3.3)	0.53
FOSQ	18.8 (1.4)	18.7 (1.6)	18.6 (1.6)	0.08

Data are shown as mean (standard deviation) for each variable within tertiles. P Values reflect 1-way analysis of variance testing to determine whether there is statistically significant variation among the apnea-hypopnea index (AHI) tertiles. ESS refers to the Epworth Sleepiness Scale; PSQI, Pittsburgh Sleep Quality Index; FOSQ, Functional Outcomes of Sleep Questionnaire.

Table 5—Variation in Sleepiness and Functional Outcome Measures by TST Tertiles

	< 6.1 h	6.1 - < 7.0 h	≥ 7.0 h	P Value
ESS	6.9 (3.8)	6.1 (3.6)	5.4 (3.5)	< 0.0001
PSQI	6.0 (3.4)	5.4 (3.2)	5.5 (3.3)	0.0001
FOSQ	18.5 (1.6)	18.7 (1.5)	18.8 (1.5)	0.0002

Data are shown as mean (standard deviation) for each variable within tertiles. P Values reflect 1-way analysis of variance testing to determine whether there is statistically significant variation among the total sleep time (TST) tertiles. ESS refers to the Epworth Sleepiness Scale; PSQI, Pittsburgh Sleep Quality Index; FOSQ, Functional Outcomes of Sleep Questionnaire.

actigraphy-determined TST also had no substantive effect on these findings. Results were also largely unchanged after adjustment for wakefulness after sleep onset, which itself was associated with an increase in ESS score of 0.15 (95% confidence interval 0.02, 0.29) for an increase of 66 minutes when added to Model 2 of Table 6.

SDB, Sleep Duration, and Sleep-Related Symptoms

PSQI score was not associated with AHI but had a univariate association with TST (Tables 4 and 5). In adjusted analyses, PSQI score was not associated with AHI or other measures of SDB severity, but there was a modest association with TST (Table 7). Adjustment for napping time, including the addition of napping time to TST, had no meaningful effect on these findings. Substitution of polysomnography-determined TST for actigraphy-determined TST also had no substantive effect on these findings. Results were also largely unchanged after adjustment for wakefulness after sleep onset, which itself was associated with an increase in PSQI score of 0.27 (95% confidence interval 0.16, 0.38) for an increase of 66 minutes when added to Model 2 of Table 7.

SDB, Sleep Duration, and Sleep-Related Quality of Life

FOSQ score demonstrated a weak bivariate association with TST but not AHI (Tables 4 and 5). After adjusting for age, race, BMI, GDS score, Goldberg Anxiety Scale score, and self-reported health status in Model 1, FOSQ score was not associated with AHI. This lack of association persisted after adjustment for TST, which was itself associated with FOSQ score (Table 8). There were no associations between FOSQ score and other measures of SDB severity (data not shown). Adjustment for napping time, including the addition of napping time to TST, had no meaningful effect on these findings (data not shown). Substitution of polysomnography-determined TST for actigraphy-determined TST also had no substantive effect on these findings. Results were also largely unchanged after adjustment for wakefulness after sleep onset, which itself was not associated with FOSQ score.

DISCUSSION

In this population of older men, there was a weak but statistically significant association between TST and various measures of SDB severity, including the AHI. SDB severity was associ-

Table 6—Linear Regression Analysis of the ESS Score with AHI and TST in 2849 Men

	Unit	Difference in ESS Score[a]	
		Model 1	Model 2
AHI	15.0	0.18 (0.04, 0.32)	0.09 (-0.04, 0.23)
TST, min	73.4		-0.69 (-0.82, -0.55)
Age, y	5	-0.03 (-0.15, 0.10)	-0.02 (-0.14, 0.10)
African American race		1.04 (0.30, 1.78)	0.85 (0.12, 1.58)
BMI	3.8	0.15 (0.01, 0.29)	0.04 (-0.10, 0.18)
GDS score	1	0.22 (0.14, 0.29)	0.22 (0.15, 0.30)
GAS score	1	0.08 (-0.002, 0.16)	0.07 (-0.01, 0.15)
Self-reported health status		0.30 (0.08, 0.51)	0.27 (0.06, 0.48)
Adjusted R-squared		0.041	0.074

[a]Model 1 is a multivariate linear regression analysis of the association between Epworth Sleepiness Scale (ESS) score (dependent variable) and apnea-hypopnea index (AHI) (independent variable), adjusting for age, race, body mass index (BMI), Geriatric Depression Scale (GDS) score, Goldberg Anxiety Scale (GAS) score, and self-reported health status. Model 2 adds total sleep time (TST) as an independent variable to Model 1 to adjust for potential confounding of the relationship between ESS and AHI. Regression results shown represent coefficient point estimates with 95% confidence intervals.

Table 7—Linear Regression Analysis of the PSQI Score With AHI and TST in 2849 Men

	Unit	Difference in PSQI Score[a]	
		Model 1	Model 2
AHI	15.0	-0.02 (-0.13, 0.09)	-0.05 (-0.16, 0.06)
TST, min	73.4		-0.22 (-0.33, -0.11)
Age, y	5	0.05 (-0.05, 0.15)	0.05 (-0.05, 0.15)
African-American race		0.50 (-0.10, 1.10)	0.43 (-0.16, 1.03)
BMI	3.8	0.06 (-0.06, 0.17)	0.03 (-0.09, 0.14)
GDS score	1	0.22 (0.16, 0.28)	0.22 (0.16, 0.28)
GAS score	1	0.51 (0.44, 0.57)	0.51 (0.44, 0.57)
Self-reported health status		0.74 (0.56, 0.91)	0.73 (0.56, 0.90)
Adjusted R-squared		0.211	0.215

[a]Model 1 is a multivariate linear regression analysis of the association between Pittsburgh Sleep Quality Index (PSQI) score (dependent variable) and apnea-hypopnea index (AHI) (independent variable), adjusting for age, race, body mass index, Geriatric Depression Scale (GDS) score, Goldberg Anxiety Scale (GAS) score, and self-reported health status. Model 2 adds total sleep time (TST) as an independent variable to Model 1 to adjust for potential confounding of the relationship between PSQI and apnea-hypopnea index (AHI). Regression results shown represent coefficient point estimates with 95% confidence intervals.

ated with sleep duration, whether due to sleep fragmentation or other mechanisms, and sleep duration was associated with most outcome measures. Thus, TST appeared to operate as a confounder in the association between functional measures and SDB severity. Although most research has considered SDB and sleep duration as distinct constructs and accordingly has incorporated only 1 measure in statistical models, our data underscore the importance of considering sleep duration in assessments of effects potentially attributable to SDB. Since the correlation between sleep duration and AHI is only weak, it is also likely that the 2 variables reflect distinct phenomena.

Daytime sleepiness, as measured by the ESS, was modestly associated with AHI; however, this association was confounded by TST, which was shorter in individuals with more severe SDB. Overall, the relationship between daytime sleepiness and TST was stronger than the relationship between daytime sleepiness and SDB severity, but the clinical relevance of less than a 1-point difference in ESS score for a 73-minute increase in TST is likely small.

Previous large, population-based studies have shown a relationship between SDB severity and daytime sleepiness. In 1824 middle-aged and older adults from the Sleep Heart Health Study, Gottlieb et al reported a linear relationship between SDB severity and daytime somnolence, measured by the ESS score.[32] A second study of 5777 older adults similarly demonstrated an association between ESS and both snoring frequency and AHI.[12] In a cohort of 718 elderly Japanese American men in the Honolulu-Asia Aging Study of Sleep Apnea, a higher fraction of men with an AHI greater than 30 reported excessive daytime somnolence (ESS > 10) than did those with a lower AHI.[11] Ancoli-Israel also reported an association between SDB severity and symptoms of sleepiness—such as falling asleep reading while not in bed and falling asleep while in conversation—in a study of 427 community-dwelling elderly subjects.[7] Finally, in a study of 4578 noninstitutionalized older adults from the Cardiovascular Health Study, signs and symptoms of SDB (such as loud snoring, awakening with dyspnea or snorting, or frequent awakenings) were associated with ESS scores.[13] None of these studies included adjustment for sleep duration. Kezirian et al[8] were the first to include adjustment for sleep duration in a population of community-dwelling older women, and the results were markedly similar to this study's: a modest association between SDB severity and ESS score was attenuated by adjustment for TST.[8]

The finding in this study, that SDB severity, by and large, was not independently associated with ESS score in older men, conflicts with earlier the results of studies from other groups. There are a few possible explanations. First, this study only focused on older men, whereas previous analyses often studied both men and women. Because the age-associated changes in sleep-wake patterns appear to be more pronounced in men than in women,[14] these may have a more pronounced effect on associations between functional measures and SDB severity when examined in older men alone. In addition, women with sleep disorders report a higher frequency of symptoms than do men. However, it is notable that our previous study of older women that included sleep duration had similar findings.

The PSQI, which has been validated in the elderly, was also not associated with SDB severity but had a modest association with TST. No previous studies have considered the association between PSQI scores and SDB severity in older men, although Kezirian et al reported similar results in older women.[8] The FOSQ has also been validated in the elderly.

Although the FOSQ scores were modestly associated with AHI in unadjusted analysis in this study, this association was attenuated after adjustment for sleep duration (which had a stronger association with FOSQ score than with AHI). These findings contrast

Table 8—Linear Regression Analysis of the FOSQ Score With AHI and TST in 2849 Men

	Unit	Difference in FOSQ Score[a]	
		Model 1	Model 2
AHI	15.0	-0.05 (-0.10, 0.01)	-0.03 (-0.08, 0.02)
TST, min	73.4		0.12 (0.07, 0.17)
Age, y	5	-0.05 (-0.10, 0.00)	-0.05 (-0.10, 0.00)
African-American race		-0.13 (-0.41, 0.15)	-0.09 (-0.37, 0.19)
BMI	3.8	0.02 (-0.04, 0.07)	0.04 (-0.02, 0.09)
GDS score	1	-0.23 (-0.26, -0.20)	-0.23 (-0.26, -0.20)
GAS score	1	-0.12 (-0.15, -0.09)	-0.12 (-0.15, -0.09)
Self-reported health status		-0.17 (-0.25, -0.09)	-0.16 (-0.24, -0.08)
Adjusted R-squared		0.208	0.214

[a]Model 1 is a multivariate linear regression analysis of the association between Functional Outcomes of Sleep Questionnaire (FOSQ) score (dependent variable) and apnea-hypopnea index (AHI) (independent variable), adjusting for age, race, body mass index (BMI), Geriatric Depression Scale (GDS) score, Goldberg Anxiety Scale (GAS) score, and self-reported health status. Model 2 adds total sleep time (TST) as an independent variable to Model 1 to adjust for potential confounding of the relationship between FOSQ and AHI. Regression results shown represent coefficient point estimates with 95% confidence intervals.

with those of Kezirian et al, who showed no association between FOSQ scores and SDB severity or TST in older women.[8]

These findings for PSQI and FOSQ scores are not entirely surprising because functional consequences of SDB (including FOSQ scores) have shown no consistent cross-sectional association with SDB severity, as measured by a frequency count of nocturnal respiratory disturbances, in young and middle-aged adults.[33,34] Sleep disturbances and sleep-related quality of life could be expected to have a stronger association with TST than with SDB severity, especially because sleep duration is 1 component of the PSQI. In addition, weaker associations may be observed in community samples and cohorts of older adults due to a survival bias. Advanced elderly may also experience competitive risk factors, contributing to variation in PSQI and FOSQ scores that are not reflected by measures of SDB severity or TST.

Another explanation is that any observed association between daytime functional impairment and SDB severity may, in fact, be mediated by TST. We believe that the inclusion of an objective measure of sleep time may explain some of the differences between our findings and those of previous studies that were unable to adjust for sleep duration.

A limitation of this study, which has limited prior research as well, is that the ESS was developed in predominantly middle-aged samples and has not been validated specifically in older populations, in whom there may be less sensitivity and specificity. Acknowledging that the ESS contains a driving question and because older individuals often do not drive, we also explored the utility of a modified ESS score derived by omitting the driving question but did not show any change in the association with AHI and TST. Another limitation is that the ESS was the only measure of sleepiness included in this study population.

This population demonstrated, on average, mild to moderate SDB, with a mean AHI of 17.0 ± 15.0, and a different sample population with greater SDB severity may experience greater functional consequences. Regression analyses were designed to minimize this potential bias, but replication of these results in a different sample may prove useful.

Taken together with our previous research,[8] these results suggest 1 of 2 possibilities: that these measures (ESS, PSQI, and FOSQ scores) are not sufficiently sensitive or specific to detect the functional consequences of SDB in older men, or SDB in older community-dwelling men may not have the same associated functional consequences seen in young and middle-aged adults or in older adults referred for clinical evaluation. Objective measures of daytime sleepiness, such as the Maintenance of Wakefulness Test or Multiple Sleep Latency Test, may have greater validity for detecting daytime sleepiness, and future investigations may choose to include these in spite of the significant logistical challenges of administering the tests to a population of older adults.

Although sleep disorders are common among the elderly, the impact of sleep disturbances—and the interaction of multiple sleep disturbances—remains to be elucidated. To our knowledge, this is the first study to evaluate the extent to which any associations were confounded by short sleep duration in a population-based sample of older men. Given the high prevalence of SDB in older men in this study, the results also highlight the importance of understanding the morbidity of SDB in this population. This study supports the notion that the functional consequences of SDB in older men may differ from those in younger populations and/or may need to be measured with instruments designed specifically for them.[35-38]

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Portions of this work were performed at each author's institution.

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DISCLOSURE STATEMENT

This was not an industry supported study. Dr. Kezirian has consulted for Apneon and Pavad Medical and is on the advisory board of Apnex Medical. Dr. Ancoli-Israel has received research support from Sepracor, Takeda, and Litebook; is on the advisory board of Acadia, Arena, Cephalon, Ferring Pharmaceuticals, Merck, Neurocrine Biosciences, Neurogen, Sanofi-Aventis, Sepracor, Somaxon, and Takeda; and has had the use of discounted equipment from Litebook and Respironics. Dr. Redline has had the use of equipment from Respironics for an NIH-funded clinical trial. Dr. Goldberg has received research support from Carbylan Medical and has consulted for Aspire Medical. Dr. Spira has worked as a clinical editor for International Journal of Sleep and Wakefulness which receives industry support. The other authors have indicated no financial conflicts of interest.

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