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Association between Sleep Duration and the Mini-Mental Score: The Northern Manhattan Study

Alberto R. Ramos, M.D., M.S.P.H.[1]; Chuanhui Dong, Ph.D.[1]; Mitchell S. V. Elkind, M.D., M.S.[2,3]; Bernadette Boden-Albala, M.P.H., Dr.P.H.[4]; Ralph L. Sacco, M.D., M.S.[1,5,6]; Tatjana Rundek, M.D., Ph.D.[1,5]; Clinton B. Wright, M.D., M.S.[1,5,7]

[1]Evelyn F. McKnight Brain Institute, Department of Neurology, Miller School of Medicine University of Miami, Miami, FL; [2]Department of Neurology, College of Physicians and Surgeons, Columbia University, New York, NY; [3]Department of Epidemiology, Mailman School of Public Health, Columbia University, New York, NY; [4]Division of Social Epidemiology, Department of Health Policy, Mount Sinai School of Medicine, New York, NY; [5]Department of Epidemiology and Public Health, University of Miami, Miami, FL; [6]Department of Human Genetics, University of Miami, Miami, FL; [7]Neuroscience Program, University of Miami, Miami, FL

SCIENTIFIC INVESTIGATIONS

Background: Short and long sleep duration are associated with increased mortality and worse global cognitive function, but is unclear if these relations persist after accounting for the risk of sleep disordered breathing (SDB). The aim of our study is determine the association between short and long sleep duration with worse global cognitive function in a racially/ethnically diverse elderly cohort.

Methods: We examined sleep hours and global cognitive function cross-sectionally within the population-based Northern Manhattan Study cohort. We conducted nonparametric and logistic regression to examine associations between continuous, short (< 6 h) and long (≥ 9 h) sleep hours with performance on the Mini Mental State Examination (MMSE).

Results: There were 927 stroke-free participants with data on self-reported sleep hours and MMSE scores (mean age 75 ± 9 years, 61% women, 68% Hispanics). The median (interquartile range) MMSE was 28 (10-30). Sleep hours (centered at 7 h) was associated with worse MMSE (β = -0.01; SE [0.004], p = 0.0113) adjusting for demographics, vascular risk factors, medications, and risk for SDB. Reporting long sleep (≥ 9 h) compared to 6 to 8 h of sleep (reference) was significantly and inversely associated with MMSE (adjusted β = -0.06; SE [0.03], p = 0.012), while reporting short sleep was not significantly associated with MMSE performance. Long sleep duration was also associated with low MMSE score when dichotomized (adjusted OR: 2.4, 95% CI: 1.1-5.0).

Conclusion: In this cross-sectional analysis among an elderly community cohort, long sleep duration was associated with worse MMSE performance.

Keywords: Sleep duration, cognition, short sleep, long sleep, cognitive impairment, mini mental score

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Cognitive impairment and dementia are disabling conditions expected to rise in prevalence with the rapidly aging population.[1,2] The identification of modifiable risk factors for cognitive impairment can provide important prevention strategies with significant public health implications. The impact of inadequate sleep on cognition can be profound. Besides producing sleepiness, which has detrimental effects on mood, job performance, and accident risk, poor sleep is associated with adverse health outcomes.[3,4] This is particularly relevant to the aging population as sleep-wake patterns and sleep quality may change throughout the lifetime, with 50% of the elderly reporting sleep disturbances, and up to one-third reporting either short sleep or long sleep duration.[5,6] Abnormal sleep duration may impair attention/vigilance and cause executive dysfunction,[6,7] but it is unclear if these relationships persist after accounting for the risk of sleep disordered breathing (SDB). SDB is highly prevalent in the elderly, seen in up to 62% of those older than 65 years of age and is associated with poor cognitive function.[8-11] Determining the relationship between sleep duration and cognition could lead to novel strategies to improve health as sleep duration is potentially modifiable.[12]

The aim of this analysis is to evaluate the association between self-reported sleep hours and short and long sleep duration with worse global cognitive function in an elderly racially/ethnically diverse population-based cohort.

BRIEF SUMMARY

Current Knowledge/Study Rationale: Cognitive impairment and dementia are expected to rise with the aging population. There is limited data from elderly race/ethnically diverse cohorts with evaluations of sleep duration and cognitive function. Determining the relation between sleep duration and cognitive function could lead to novel strategies to improve health.

Study Impact: Long sleep duration was associated with worse mini-mental score, a measure of global cognitive function, after adjusting for demographic, vascular risk factors and depressive symptoms. The results of this study suggest that long sleep duration may be an independent predictor of worse cognitive function in the elderly.

METHODS

Study Population

The Northern Manhattan Study (NOMAS) enrolled 3,298 stroke-free participants randomly sampled from the Northern

Manhattan population between 1993 and 2001 using the following criteria: (1) resident of Northern Manhattan ≥ 3 months; (2) from a household with a telephone; (3) age ≥ 40 years at the time of first in-person assessment; and (4) no history of stroke.[13] For the purpose of this analysis, we included participants with self-reported sleep hours obtained during annual telephone follow-up evaluation in 2006 and Mini-Mental Examination (MMSE) scores within one year of reported sleep hours. From the parent cohort, a total of 2,266 subjects were available for follow up in 2006. Of the available sample, a total of 927 participants had reports of sleep hours and MMSE within one year of each assessment. The sample of 927 participants had a similar proportion of women (61%), but a greater proportion of Hispanics (68%) compared to the overall baseline cohort (53%). NOMAS was approved by the Columbia University Medical Center and University of Miami, Miller School of Medicine IRBs, and all participants provided written informed consent.

Cognitive Assessment

Cognitive status was assessed in person by bilingual (English or Spanish) trained research assistants using MMSE.[14] The MMSE is a brief 30-point questionnaire test used to evaluate cognitive function. The MMSE measures various domains of cognitive functioning including memory, orientation to place and time, naming, reading, visuospatial orientation/construction ability, writing, and the ability to follow a 3-stage command. It has good sensitivity (71% to 92%) and specificity (56% to 96%) to screen for cognitive impairment and dementia.[14]

We used the total score of the MMSE as the outcome. Lower educational levels (≤ 8th grade) can adversely affect the MMSE scores. We defined low MMSE scores as a dichotomous outcome by adjusting for age and educational level based on established MMSE cutoffs. A cutoff of MMSE < 24 was used for those with > 8 years education and MMSE < 20 for those with ≤ 8 years of education.[15,16]

Sleep Hours

We collected self-reported sleep duration as an estimate of hours of nightly sleep in the four weeks prior to the annual telephone follow-up interview in 2006, using the following question: "During the past 4 weeks, how many hours, on average, did you sleep each night?" Respondents reported in 30-min increments of each hour.[17] The responses ranged from 3 to 12 h of sleep with a median of 7 hours.

High Risk for Sleep Disordered Breathing

High risk for SDB was estimated by constructing the Berlin questionnaire,[18] based on reports of frequent snoring and daytime sleepiness along with objective information on hypertension and obesity in our sample. Sleep symptoms were derived from a sleep questionnaire during follow-up examination in 2004-2005.[17] The questionnaires were administered in English or Spanish. Habitual snoring was defined as self-report of snoring > 3 times per week, based on prior definitions of habitual snoring.[19] The Epworth Sleepiness Scale was used and adapted for relevance to characteristics of people living in northern Manhattan.[20] Daytime sleepiness was categorized as sum score ≥ 10 based on the established definition for daytime sleepiness from the Epworth Sleepiness Scale.[21] The presence of 2 of the 3 following items was used to classify participants into high risk for SDB: (1) frequent snoring (snoring > 3 times per week), (2) daytime sleepiness (sum score ≥ 10), and (3) presence of hypertension or obesity (BMI > 30 kg/m^2).

Risk Factor Assessments

Data were collected through interviews by trained bilingual research assistants using standardized data collection instruments described elsewhere.[13] Race and ethnicity were defined by self-identification based on questions modeled after the US census. Race/ethnicity were categorized into mutually exclusive groups as non-Hispanic Black, non-Hispanic White, and Hispanic. Depressive symptoms were evaluated with the Center for Epidemiological Studies Depression scale (CES-D). The CES-D is a 20-item scale documenting 4 factors: depressive affect, somatic complaints, positive affect, and interpersonal relations. Scores on the CES-D range from 0 to 60, with higher scores indicating more symptoms of depression.[22] Depressive symptoms were categorized as present if the sum of the scores was ≥ 16 or if the participant was taking an antidepressant medication.[22] Hypertension was defined as a systolic blood pressure > 140 mm Hg or a diastolic blood pressure > 90 mm Hg or a patient's self-report of a history of hypertension or use of antihypertensive medications. Diabetes mellitus was defined as fasting blood glucose ≥ 126 mg/dL or the patient's self-report of diabetes or use of insulin or hypoglycemic medications. Cardiac disease included history of angina, MI, coronary artery disease, atrial fibrillation, congestive heart failure, or valvular heart disease.

We obtained self-reported medication use at baseline. We created a dichotomous variable (yes vs. no) based on the use of the following medications: antidepressant, antiepileptic, pain, and antipsychotic, that could affect sleep duration and or cognitive function.[17]

Statistical Analysis

Results are presented as mean ± standard deviation or median (interquartile range) for continuous variables according to the variable distribution, and proportion for categorical variables. The χ^2 test was used to compare proportions, while ANOVA, or the Kruskal-Wallis test if data were not normally distributed, was used to compare mean or median for continuous variables. We examined self-reported sleep hours in categories of short sleep (< 6 h) and long sleep (≥ 9 h), with 6 to 8.9 h of nightly sleep as the reference.[23] We performed nonparametric regression to test for the association between sleep hours centered at 7 h of sleep continuously and comparing categories of < 6 h and ≥ 9 h versus the reference with the non-normally distributed MMSE using SAS procedure COUNTREG. Sequential models were done to evaluate the unadjusted association between sleep hours (centered at 7 h) and MMSE. We then adjusted for demographic factors: age, sex, education, race/ethnicity, and insurance status (Model 2); alcohol consumption, hypertension, diabetes, depression, medications, and risk for SDB (Model 3). Logistic regression was performed with the categories for low MMSE score as the outcome. As a sensitivity analysis, we also evaluated the relation between sleep hours and memory performance on the MMSE, given that verbal 3-word recall on the MMSE has been reported as an acceptable estimate of episodic memory in epidemiologic studies.[24] Among participants able

Table 1—Demographic and vascular risk factors and cognitive scores across categories of sleep hours

Mean ± SD or N (%) or as indicated	Total (N = 927)	< 6 h (n = 224, 24%)	6-8.9 h (n = 616, 66%)	≥ 9 h (n = 87, 9%)
Demographic				
Age, years	75 ± 9	74 ± 8	74 ± 9	77 ± 9*
Women	567 (61)	144 (64)	371 (60)	52 (60)
≤ 8th grade education	381 (41)	100 (45)	241 (39)	40 (46)
Medicaid or no insurance	302 (33)	68 (30)	199 (32)	35 (40)
Race-Ethnicity				
Non-Hispanic White	127 (14)	25 (11)	93 (15)	9 (11)
Non-Hispanic Black	163 (18)	42 (19)	104 (17)	17 (20)
Hispanic	615 (68)	152 (69)	405 (67)	58 (69)
Risk Factors				
BMI (kg/m^2)	28.2 ± 4.7	28.4 ± 5.0	28.1 ± 4.7	27.5 ± 4.7
Moderate alcohol	363 (39)	69 (31)	259 (42)	35 (41)*
Current smoking	155 (17)	32 (14)	104 (17)	19 (22)
Depression	185 (20)	53 (24)	111 (18)	21 (24)
Hypertension	634 (68)	140 (63)	429 (70)	65 (75)*
Diabetes	131 (14)	34 (15)	75 (12)	22 (25)**
Cardiac disease	163 (18)	37 (17)	105 (17)	21 (24)
Medication£	216 (23)	63 (28)	131 (21)	22 (25)
High risk SDB	245 (26)	54 (24)	166 (27)	25 (29)
Mini-Mental Score (MMSE)				
MMSE, median (interquartile range)	28 (10-30)	28 (3)	28 (4)	26 (6)***
Low MMSE score (Based on age/education cutoff)	93 (10)	18 (8)	59 (10)	16 (18)*

SDB, sleep disordered breathing. *p < 0.05, **p < 0.001, ***p < 0.0001. £Antidepressant, antiepileptic, pain, and/or antipsychotic medication.

Table 2—Association between sleep hours and Mini-Mental Score Examination

	Model 1		Model 2		Model 3	
Sleep hours	**β (SE) per hour**	**p**	**β (SE) per hour**	**P**	**β (SE) per hour**	**p**
Centered at 7 h	-0.01 (0.004)	0.0180	-0.009 (0.004)	0.0393	-0.01 (0.004)	0.0113
Categorical						
< 6 h	0.005 (0.02)	0.74	0.01 (0.02)	0.46	0.01 (0.02)	0.39
6-8.9 h	Reference	–	–	–	–	–
≥ 9 h	-0.07 (0.02)	0.0012	-0.05 (0.02)	0.0187	-0.06 (0.03)	0.0120

Model 1: univariate. Model 2: adjusted for age, sex, race-ethnicity, education, and Medicaid or no insurance status. Model 3: adjusted for covariates in model 2 and reported alcohol consumption, depression, diabetes mellitus, hypertension, high risk for SDB, and medications. SE, standard error.

to register 3 initial words, impaired verbal recall was defined by a score of 0 or 1 obtained on the subsequent 3-word recall task of the MMSE.[24] Additionally, we evaluated the interactions between sleep hours and the covariates. All analyses were performed using SAS software version 9.3 (SAS Institute Inc, Cary, NC).

RESULTS

The mean age was 75 ± 9 years, with 61% women, 68% Hispanics, and 41% with less than a high school education. **Table 1** presents the characteristics of the overall sample and across categories of sleep duration. Self-reports < 6 h were seen in 24% of the sample, and ≥ 9 h were reported by 9% (n = 87). Participants reporting ≥ 9 h of sleep were older, had greater frequencies of hypertension, diabetes, and lower MMSE scores than the reference (p < 0.0001). There was no statistical difference in the frequencies of sex, education, race-ethnicity, Medicaid or no insurance status, BMI, depression, cardiac disease, or risk of SDB among the groups.

The covariates that were associated with the lower MMSE scores were increased age (β = -0.004; p < 0.0001), ≤ 8th grade education (β = -0.14; p < 0.0001), having Medicaid or no insurance (β = -0.14; p < 0.0001), Hispanic race/ethnicity (β = -0.09, p < 0.0001) compared to non-Hispanic white, depression (β = -0.05, p = 0.0029), diabetes (β = -0.05, p = 0.012), hypertension (β = -0.05, p = 0.0007), and medications (β = -0.08, p < 0.0001). Male sex (β = 0.04; p = 0.0065) and moderate alcohol consumption alcohol (β = 0.04, p = 0.009) were positively associated with the MMSE. The BMI (β = 0.0005, p = 0.74), non-Hispanic black (β = 0.006, p = 0.71) compared to non-Hispanic white race/ethnicity, current smoking (β = 0.001, p = 0.94), cardiac disease (β = 0.006, p = 0.71), and risk for SDB (β = -0.02, p = 0.11) were not associated to the MMSE.

Self-reported sleep hours (continuous) was associated with worse MMSE scores in sequential models (**Table 2**). In addition, categorical analysis showed that self-reports of ≥ 9 h (long sleep duration), compared to 6-8.9 h of sleep were associated with worse MMSE scores in fully adjusted models (**Table 2**). When evaluating cognitive scores as a binary outcome, we

Table 3—Odds ratio and 95% confidence interval among categories of sleep duration and low Mini-Mental score (MMSE)*

	Model 1	Model 2	Model 3
< 6 h	0.8 (0.5-1.4)	0.8 (0.4-1.4)	0.8 (0.4-1.6)
≥ 9 h	2.1 (1.2-3.9)	1.9 (1.02-3.7)	2.4 (1.1-5.0)

*Low MMSE: A cutoff of MMSE < 24 was used for > 8 years education and MMSE < 20 for ≤ 8 years of education. Reference: 6-8.9 hours. Model 1: unadjusted. Model 2: adjusted for age, sex, race-ethnicity, education, and Medicaid or no insurance status. Model 3: adjusted for covariates in model 2 and alcohol consumption, depression, diabetes, hypertension, high risk for SDB, and medications.

found that long sleep duration (≥ 9 h) was associated with increased odds of low MMSE scores (**Table 3**). There was no association between sleep hours and delayed verbal memory and no interactions between sleep hours and demographic, vascular risk factors, medications, and risk for SDB.

DISCUSSION

In this cross-sectional study, we found that self-reported long sleep duration was associated with worse global cognitive function in the elderly, racially/ethnically diverse NOMAS sample, after adjusting for vascular risk factors, depression, and risk for SDB.

Self-reported long sleep duration is linked to greater mortality, and an increased risk of stroke and cardiovascular disease.[3,23,25,26] While very few NOMAS participants had dementia and our results most likely reflect subtle cognitive differences, our findings are in agreement with prospective data from an elderly (≥ 65 years) population-based cohort showing a positive association between long sleep duration (≥ 9 h) and incident dementia.[6] A cross-sectional analysis of the Osteoporotic Fractures in Men Study (MrOS) also demonstrated an association between long sleep (≥ 8 h) by actigraphy and worse global cognitive scores.[27] Population-based studies have reported associations between long sleep duration and worse cognitive performance by measures of global cognition (MMSE),[24,28] as well as verbal fluency, delayed recall,[29] and psychomotor speed.[30]

Most studies on sleep hours and cognitive function have examined homogenous populations, with a paucity of data from racially/ethnically diverse communities. Studies comparing self-reported sleep duration in Hispanics and non-Hispanic blacks have provided inconsistent results, suggesting that habitual sleep duration is possibly dependent on factors other than race-ethnicity.[9,31,32] We previously described greater long sleep duration in Hispanics compared to non-Hispanic whites[17] and observed an inverse relation between Hispanic race/ethnicity and MMSE scores. In NOMAS, a greater proportion of Hispanics have less than eight years of formal education as well as Medicaid or no insurance, both surrogate markers of lower SES. Lower SES is associated with a number of comorbidities that could result in long sleep duration.[25,26,33]

Self-reports of long sleep duration have been associated with older age, low socioeconomic status (SES), diabetes, and vascular disease,[25] but we observed an association between long sleep duration and MMSE after controlling for these factors. Additionally, long sleep duration was associated with worse MMSE score after controlling for depressive symptoms, medications (e.g., antidepressants, antiepileptics) and risk for SDB, factors that may worsen cognitive function.[11,34-36] Our findings suggest that in the elderly, long sleep duration (≥ 9 h) could be an independent predictor of worse cognitive function.

It is suggested that the relation between long sleep and adverse health outcomes could be confounded by SDB.[30,37] In our study, there was no difference in risk for SDB among the sleep duration groups, and high risk for SDB did not modify the relation between self-reported long sleep and worse MMSE scores. Our findings are in accordance with an analysis of the Osteoporotic Fractures in Men study[38] that characterized differences in demographic, sleep, and vascular risk factors among elderly participants (mean age 76.4 years) with long sleep duration compared to average sleepers. In this study there were no differences in the apnea-hypopnea index between long sleep compared to 7-8 hours of sleep.[38] In addition, self-reported long sleep duration was positively associated with increased time in bed and sleep time by actigraphy that was not explained by sleep disorders, such as SDB or vascular risk factors.

Our findings could be explained by sleep fragmentation. Fragmented sleep has been linked to long sleep duration.[4] Fragmented sleep measured by actigraphy was associated with worse global cognitive function, independent of sleep duration, in a cross-sectional analysis of the Rush Memory and Aging Project.[39] Sleep fragmentation is directly related to time in bed,[25] and perhaps long sleep duration could be a surrogate or a compensatory response to fragmented sleep in those with worse cognitive function.[29] Sleep-wake disturbances could also exacerbate cognitive dysfunction and cause further sleep disturbances, such as advancement of circadian phase with subsequent prolongation of sleep duration.[24]

In our study, self-reported short sleep duration was not associated with MMSE. Short sleep duration has been associated with worse global cognitive function, memory impairment, and psychomotor speed,[7,30,40,41] but stronger associations have been described for long sleep duration. Our findings are in accordance with population based studies where short sleep duration was not associated, either by self-report[28] or actigraphy,[27] with MMSE. Short sleep duration can cause deficits in attention and vigilance through excessive sleepiness,[42] but the mechanisms by which long sleep duration could affect cognitive function are not fully understood.

Several limitations should be noted. The current study is cross-sectional and does not allow assessment of causality between self-reported sleep hours and cognition. Sleep duration was obtained by subjective reports from a sleep questionnaire. In particular, it could be that those with cognitive impairment tend to report longer sleep duration. Also, we were not able to capture night to night variability of sleep duration, daytime napping, or objective measures of sleep. However, other observational studies of sleep duration and adverse health outcomes are similarly based on subjective reports from sleep questionnaires. Self-reports of long sleep duration might represent a greater sleep time or just more time in bed, which cannot be determined from the current data. There might be unmeasured confounders (e.g., autoimmune disorders) that could cause fatigue and sleepiness and in part explain the results of our study. In spite

of these limitations there are several strengths to our study. We evaluated a relatively large, racially/ethnically diverse, community-based cohort with a high burden of vascular risk factors, risk of SDB, depression, and systematically applied measures of cognition with the MMSE.

In conclusion, we found a cross-sectional association between self-reported long sleep duration, and greater odds of worse cognitive scores that were not explained by high risk of SDB. Prospective studies in racially/ethnically diverse samples are needed to confirm our findings and determine if long sleep duration is in the causal pathway and a harbinger of cognitive decline.

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Address correspondence to: Alberto Ramos, M.D., M.S.P.H., Sleep Medicine Program, Department of Neurology, University of Miami Miller School of Medicine, 1120 NW 14th Street, Suite 1350. Miami, Florida 33136, Tel: (305) 243-1305; Fax: (305) 243-7081

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