

RESEARCH ARTICLE

Sleep duration and brain MRI measures: Results from the SOL-INCA MRI study

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Abstract

INTRODUCTION: Sleep duration has been associated with dementia and stroke. Few studies have evaluated sleep pattern-related outcomes of brain disease in diverse Hispanics/Latinos.

METHODS: The SOL-INCA (Study of Latinos-Investigation of Neurocognitive Aging) magnetic resonance imaging (MRI) study recruited diverse Hispanics/Latinos (35–85 years) who underwent neuroimaging. The main exposure was self-reported sleep duration. Our main outcomes were total and regional brain volumes.

RESULTS: The final analytic sample included $n = 2334$ participants. Increased sleep was associated with smaller brain volume ($\beta_{\text{total_brain}} = -0.05, p < 0.01$) and consistently so in the 50+ subpopulation even after adjusting for mild cognitive impairment status. Sleeping >9 hours was associated with smaller gray ($\beta_{\text{combined_gray}} = -0.17, p < 0.05$) and occipital matter volumes ($\beta_{\text{occipital_gray}} = -0.18, p < 0.05$).

DISCUSSION: We found that longer sleep duration was associated with lower total brain and gray matter volume among diverse Hispanics/Latinos across sex and

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background. These results reinforce the importance of sleep on brain aging in this understudied population.

KEYWORDS

Alzheimer's disease, magnetic resonance imaging, sleep

Highlights

- Longer sleep was linked to smaller total brain and gray matter volumes.
- Longer sleep duration was linked to larger white matter hyperintensities (WMHs) and smaller hippocampal volume in an obstructive sleep apnea (OSA) risk group.
- These associations were consistent across sex and Hispanic/Latino heritage groups.

1 | INTRODUCTION

Sleep patterns can have important public health implications and can lead to increased health care costs. Short (<7 hours) and long sleepers (>8 hours) spend \$1278 and \$2994 (respectively) more on health care compared to average duration sleepers.¹ Sleep patterns vary among demographic groups including by race/ethnicity.² This may partly reflect cultural practices and beliefs around sleep.³ Recent work using self-reported sleep data from the National Health and Interview Survey suggests that Hispanics/Latinos, on average, were more likely to report shorter sleep duration compared to Whites.⁴ Heterogeneities in sleep patterns among Hispanic/Latino subgroups have also been documented in the Hispanic Community Health Study/Study of Latinos (HCHS/SOL). Patel et al.⁵ reported that Hispanics/Latinos of Dominican and Puerto Rican (Caribbean Latinos) heritage reported significantly shorter sleep duration compared to other Hispanic/Latino groups. More recently, Gonzalez et al.⁶ found that Caribbean Latinos are more likely to meet criteria for insomnia relative to other Hispanic/Latinos subgroups. Important demographic modifiers (e.g., sex)⁶ for sleep disorders within Hispanics/Latinos may also exist. For example, Seicean et al.⁷ found that Mexican men had longer sleep compared to women in a young cohort (40 years on average); however, it is unclear whether these sex/gender differences are consistent across other Hispanic/Latino heritages.

Longer sleep duration is associated with increased risk of adverse health outcomes. Compared to average sleep duration (7–9 hours), longer (>9 hour) sleep is associated with increased risk for hypertension, cardiovascular disease, and obesity.^{8,9} Sleep patterns also influence downstream outcomes in older adults including cognition. Both short and long sleep have been linked with worse cognitive performance.¹⁰ Recent meta-analyses have validated these associations.^{11,12} Sleep disruption is a common complaint among persons with dementia.¹³ The Neurological Disorders in Central Spain Studies (NEDICES) found an increased risk of dementia mortality for long sleepers (≥ 9 hours) compared to those sleeping between 6 and 8 hours.¹⁴ Long sleep has also been linked to an increased risk of stroke, a major pathway for disablement and dementia.¹⁵ The pathways in which sleep could lead to dementia have been explored; for example,

Winer et al. found short sleep, but not long sleep, was associated with increased amyloid burden and worse cognitive performance.¹⁶ These findings suggest that sleep duration might be a clinically important metric in at-risk individuals.

We used data from a large and diverse community-based cohort of middle-aged and older Hispanic/Latino adults to fill existing gaps in the literature on sleep and brain health. We did so by (1) examining the associations between sleep duration and total and regional brain volumes, (2) surveying different subpopulations of interest, and (3) examining modifications by sex and Hispanic/Latino background groups. We hypothesized that short (<6 hour) and long (>9 hours) sleep durations (also tested continuously) will be associated with smaller brain volumes (defined below) and larger white matter hyperintensity (WMH) volumes. Furthermore, we proposed that these associations will be more pronounced in Puerto Ricans, Dominicans, and women because they have been reported to experience short sleep duration, fragmented sleep, and insomnia-like symptoms compared to men and other Hispanic/Latino subgroups.⁶

2 | METHODS

2.1 | Data

HCHS/SOL is a community-based prospective cohort study of diverse Hispanic/Latinos. HCHS/SOL used a complex survey design and recruited $n = 16,415$ self-reported Hispanics/Latinos (ages 18–74; 2008–2011) from four major metropolitan cities: San Diego, CA; Chicago, IL; Bronx, NY; Miami, FL. Each center recruited ≈ 4000 community-dwelling diverse Hispanics/Latinos. Sampling design and methods for HCHS/SOL are published elsewhere¹⁷ and can also be found at the HCHS/SOL website <https://sites.csc.unc.edu/hchs>. The SOL-INCA (Study of Latinos-Investigation of Neurocognitive Aging) study, an ancillary study of the HCHS/SOL, aims to assess cognitive performance among participants who were 45–74 years of age at visit 1 ($n = 6377$).

SOL-INCA MRI leverages the HCHS/SOL cohort and neurocognitive data from the SOL-INCA to examine brain health in the Hispanic/Latino

population, which is underserved and faces disparities in vascular risk factors. SOL-INCA MRI seeks to examine brain health and the role of vascular risk factor burden on cerebrovascular pathology and Alzheimer's disease (AD) risk using state of the science magnetic resonance imaging (MRI) techniques. Approximately 2400 participants will be recruited from SOL-INCA with participant selection enriched for individuals with cognitive impairment (González et al. 2019) and the remaining cognitively healthy subjects randomly sampled with sex and field center matching to the participants with cognitive impairment. In addition, ≈400 younger (between 35 and 50 years of age at visit 2) participants will be selected randomly from the parent HCHS/SOL study to achieve a lifespan perspective on Hispanic/Latino brain health. The complete sample of participants who underwent imaging and had MRI data processed successfully include $n = 2667$ participants (weightings male = 43%, female = 56%, ages 35–85 years).

2.2 | MRI data acquisition

Participants were scanned using a GE 3T 750 (three sites) and a Philips 3T Achieve TX (one site). The sequences include: (1) *T1 volume*: ≈1 mm³ resolution; (2) three-dimensional fluid-attenuated inversion recovery (3D FLAIR); (3) *T2*GRE* (gradient echo): 2D, long *T2*GRE*; and (4) *resting-state functional MRI (fMRI)*: 10-min duration, 3 s repetition time (TR). All images are quality checked visually at every step of the segmentation process and minor errors are fixed; failed images are not processed further. Skull stripping, or removal of non-brain tissue, was performed using a convolutional neural network model.¹⁸ Gray matter volumes were calculated using an expectation-maximization (EM) algorithm.¹⁹ We created the initial space of the EM algorithm using the template-space warps of previous segmented images. We then calculated mean and SD of the image intensities, which we used as parameters for the Gaussian model. The Gaussian model is used to generate segmentations, which are then used as input for a Markov Random Field (MRF) model. The MRF model will help us produce further mean and SD values from refined segmentation. This process is repeated until convergence.¹⁹ WMH volume was obtained by performing a modified Bayesian probability structure-based algorithm on FLAIR and 3D T1 images.²⁰ The likelihood values from the Bayesian model are dichotomized (3.5 standard deviation threshold) to create a binary WMH mask. WMH volume is then log-transformed to account for variance. Hippocampal volume was calculated using an atlas-based diffeomorphic approach,²¹ with an additional European Alzheimer's Disease Consortium-Alzheimer's Disease Neuroimaging Initiative (EADC-ADNI) mask to help standardize outputs.²² Finally, we performed intensity-based label refinement to generate final outputs.

2.3 | Cognitive testing

Cognitive testing was administered only to participants middle aged and older (45–74 years) in their preferred language at HCHS/SOL

RESEARCH IN CONTEXT

1. **Systemic review:** Authors surveyed literature regarding sleep duration and brain magnetic resonance imaging (MRI) volumes as well as sleep differences across sex and ethnic groups using traditional sources (e.g., PubMed).
2. **Interpretation:** Most participants (79%) slept between 6 and 9 hours. One hour increase in sleep was associated with smaller total brain volumes. Compared to the 6- to 9-hour group, participants who slept >9 hours had smaller total gray and occipital gray matter volumes. We did not find evidence for non-linear relationships between sleep duration and brain measures. In a high risk obstructive sleep apnea (OSA) group, we found that a 1-hour increase in sleep was associated with larger white matter intensities (WMHs) and smaller hippocampal volume. Results were consistent across sex and background.
3. **Future directions:** Future research should use longitudinal sleep data and consider links between sleep duration and other clinically important brain markers (e.g., amyloid) to further understand how sleep affects risk for Alzheimer's disease and related dementia's.

Visit 1 (older than 50 at MRI). The Neurocognitive Reading Center trained technicians to ensure generalizability of testing across centers. Bilingual/bicultural technicians administered the following cognitive battery at each of the Field Centers: (1) Brief-Spanish English Verbal learning Test (B-SEVLT; Sum = learning, Recall = memory),^{23,24} (2) Word Fluency (WF),²⁵ and (3) Digit Symbol Subtest (DSS; processing speed).²⁶ Global cognitive function at visit 1 was assessed by averaging the four normalized (Z-Score) cognitive measures. Detailed information about cognitive testing and methods at visit 1.²⁷

2.4 | Mild cognitive impairment

Mild cognitive impairment (MCI) was assessed only in individuals over the age of 50 at SOL-INCA MRI (unweighted $n = 255$) using the National Institute on Aging-Alzheimer's Association (NIA-AA) four core criteria: (1) cognitive performance in the mild impairment range for any of the tests ($\bar{x} - 1$ SD) compared to SOL-INCA norms (age, education, sex, and Picture Vocabulary Test score adjusted); (2) Global cognitive decline exceeding -0.55 SD/year between the two visits; (3) any subjective cognitive decline, over the past 10 years, as assessed by the Everyday Cognition-12 rating questionnaire; and (4) no clear evidence of functional impairment using instrumental activities of daily living.²⁸ Individuals scoring less than 2 SD relative to SOL-INCA norms or who met criteria for MCI (criteria 1–3 above) but had evident functional impairment (criteria 4 above) were considered as meeting criteria for suspected severe cognitive impairment.

2.5 | Analytic sample

A total unweighted $n = 2667$ participants had MRI data available. We excluded $n = 333$ participants who did not participate in the baseline sleep module or who had missing data on model covariates for a final analytic sample of $n = 2334$.

2.6 | Exposure

The main exposure was self-reported sleep duration assessed at visit 1. Participants were asked to report, in hours, their usual awake and sleep times during the week and weekend. Participants were not told how many hours of sleep constituted "good" sleep. Daily average sleep duration was obtained by averaging through the typical sleep duration for an entire week (five weekdays and two weekend days). Sleep duration was treated as a categorical and continuous measure. The categorical cutoffs for sleep duration (<6 hours, 6–9 hours, >9 h) were chosen based on previous research on sleep duration and MRI measures,²⁹ and our own HCHS/SOL data using these categories of sleep.³⁰

2.7 | Outcomes

included brain volumes, measured in cm^3 , of the following: total brain, combined gray, frontal gray matter, occipital gray matter, temporal gray matter, parietal gray matter, lateral ventricle, WMHs, and hippocampus. All outcomes were residualized to total cranial volume and standardized. Both WMHs and lateral ventricle were log transformed before residualizing due to non-normal distributions.

2.8 | Covariates

included continuous age at MRI, time between visits (in years), sex (male, female), categorical education (less than high school, high school, more than high school), language preference (Spanish, English), Hispanic/Latino background (Dominican, Puerto Rican, South American, Mexican, Central American, Cuban), continuous body mass index (BMI), continuous depression symptom score (Center for Epidemiologic Depression Scale, CESD³¹), a binary measure for hypertension (no hypertension, hypertension) measured according to National Health and Nutrition Examination Survey (NHANES; systolic or diastolic blood pressure $\geq 140/90$ mm Hg or antihypertensive medication use), testing center (San Diego, Miami, Chicago, Bronx), and continuous respiratory event index (REI; number of apnea and hypopnea events per hour). We also controlled for self-reported sleep quality using a binary measure based on a question probing the respondent's assessment of their "typical night sleep during the past four weeks" (restless, very restless = 0; average quality, sound or restful, and very sound or restful = 1). The sleep quality question was asked before asking about sleep duration to avoid bias in response. Except for age, all covariates were

assessed at visit 1. To assess respiratory events, participants used an ARES unicorder 5.2; B-Alert (Carlsbad, CA) during visit 1.³²

2.9 | Statistical analysis

All analyses were generated using STATA 17 software. First, we generated descriptive statistics of the target population by categorical sleep duration (Table 1). We reported means and SDs for continuous measures and percent and standard errors for categorical measures. To test differences across groups, we performed analysis of variance (ANOVA) and chi-square tests for continuous and categorical (respectively) indicators. Second, we performed linear regressions on each brain outcome using (1) crude; (2) age, sex, education, background; (3) and full covariate adjusted models. We ran each of these models using a categorical (using 6–9 hours as the reference), and continuous measure of sleep (Tables 2–3). For both models, we plotted marginal estimates to ease interpretation of findings (Figures 1, 2). Models not included in the main figures can be found in Figures S1 and S2.

In sensitivity models, we tested the nonlinear relationship between sleep duration and brain volumes by adding a quadratic measure of sleep duration (sleep duration * sleep duration i.e., sleep duration²). Results from these models can be found on Table S1. Next, we reran three separate sets of models, treating sleep duration as a continuous measure, using the following subpopulations: (1) middle-age and older participants (50+ years), (2) young participants (<50 years), and (3) elevated risk obstructive sleep apnea (OSA) participants (REI ≥ 5). In the middle-age and older models (50+ years), we ran three additional models that adjust for (1) baseline global cognition at visit 1 and excluded from analysis those with suspect severe cognitive impairment ($n = 18$), (2) MCI status ($n = 215$) and excluded from analysis those with suspect severe cognitive impairment, and (3) MCI status without excluding the suspect severe cognitive impairment. Beta estimates for these models are included in Tables S2–S4, respectively. In addition, we tested modifications by background and sex in the full sample. Results from ANOVA F -tests for those can be found in Tables S5 and S6, respectively.

3 | RESULTS

3.1 | Descriptive statistics

Descriptive statistics by sleep duration category are reported in Table 1. On average, participants in the <6 hour group ($n = 182$; 6.9%) were less likely to have more than high school education and had the lowest rate of good sleep quality of the three groups. Those in the 6–9 hour group had the highest rates of education, lowest level of depression, the highest proportion of Spanish speakers, performed the highest on the word fluency test, and had the highest rate of sleep quality of the three groups. Those in the >9 hour group skewed male and had education and sleep quality measures between the other two groups.

TABLE 1 Baseline sociodemographic and sleep characteristics of SOL-INCA MRI participants by sleep duration.

	<6 h	6–9 h	>9 h	Total	p-value
Unweighted N	182	1825	327	2,334	
%	6.9	78.5	14.6	100	
Mean ± SD					
Age	64.02 ± 11.06	64.35 ± 11.22	62.48 ± 12.47	64.05 ± 11.44	p = 0.358
BMI	30.05 ± 6.23	29.60 ± 5.14	30.41 ± 5.98	29.75 ± 5.36	p = 0.310
REI	6.95 ± 11.83	7.81 ± 11.35	8.53 ± 16.25	7.86 ± 12.27	p = 0.661
Depression score	8.72 ± 7.20	6.72 ± 5.77	8.24 ± 6.36	7.08 ± 6.00	p = 0.001
Time between visits in years	10.24 ± 1.54	10.32 ± 1.54	10.03 ± 1.40	10.27 ± 1.52	p = 0.102
B-SEVLT Sum	22.70 ± 5.85	23.11 ± 5.55	22.30 ± 5.72	22.98 ± 5.61	p = 0.272
B-SEVLT Recall	8.33 ± 2.92	8.38 ± 2.84	8.22 ± 3.13	8.36 ± 2.89	p = 0.870
WF	18.56 ± 8.58	19.68 ± 7.02	17.82 ± 8.00	19.35 ± 7.30	p = 0.072
DSS	33.71 ± 14.33	35.26 ± 12.79	31.94 ± 12.56	34.70 ± 12.92	p = 0.013
% (SE)					
Language preference					
Spanish	82.46 (3.65)	87.26 (1.30)	79.83 (3.50)	85.84 (1.23)	p = 0.023
English	17.54 (3.65)	12.74 (1.30)	20.17 (3.50)	14.16 (1.23)	
Sex					
Female	49.86 (5.05)	55.76 (2.03)	60.53 (3.89)	56.05 (1.68)	p = 0.279
Male	50.14 (5.05)	44.24 (2.03)	39.47 (3.89)	43.95 (1.68)	
Education					
Less than high school	46.80 (5.35)	34.57 (2.01)	35.65 (3.77)	35.57 (1.76)	p = 0.022
High school or equivalent	21.69 (3.77)	19.65 (1.38)	27.88 (4.50)	20.99 (1.27)	
More than high school	31.51 (4.53)	45.78 (2.03)	36.47 (4.23)	43.44 (1.76)	
Background					
Dominican	9.85 (2.65)	8.57 (1.09)	8.43 (1.99)	8.64 (1.02)	p = 0.204
Central American	13.42 (4.17)	7.10 (0.88)	6.08 (1.83)	7.38 (0.85)	
Cuban	11.62 (3.51)	24.29 (2.88)	23.09 (4.20)	23.24 (2.54)	
Mexican	31.94 (5.13)	35.53 (2.38)	38.30 (4.37)	35.69 (2.15)	
Puerto Rican	22.76 (4.28)	15.84 (1.56)	13.77 (2.44)	16.01 (1.33)	
South American	7.20 (3.45)	5.72 (0.85)	4.19 (1.35)	5.60 (0.74)	
Other	3.22 (2.12)	2.95 (0.63)	6.15 (3.21)	3.44 (0.71)	
Sleep Quality					
Restless/very restless	29.72 (4.72)	18.11 (1.48)	24.86 (3.80)	19.90 (1.41)	p = 0.012
Average/restful/very restful	70.28 (4.72)	81.89 (1.48)	75.14 (3.80)	80.10 (1.41)	
Hypertension					
No hypertension	62.03 (5.27)	58.16 (2.12)	53.66 (4.66)	57.76 (1.81)	p = 0.483
Hypertension	37.97 (5.27)	41.84 (2.12)	46.34 (4.66)	42.24 (1.81)	
Mild Cognitive Impairment (MCI)					
No MCI	84.90 (4.61)	87.10 (1.61)	84.79 (3.48)	86.64 (1.39)	p = 0.150
MCI	15.10 (4.61)	12.90 (1.61)	15.21 (3.48)	13.36 (1.39)	
Suspect severe cognitive impairment	0.24 (0.25)	0.97 (0.34)	3.67 (2.51)	1.27 (0.43)	

Abbreviations: BMI, body mass index; B-SEVLT SEVLT, Brief Spanish English Verbal Learning Test; CESD, Center for Epidemiologic Studies Depression Scale; DSS, Digit Symbol Substitution Test; MCI, mild cognitive impairment; MRI, magnetic resonance imaging; REI, respiratory event index; SD, standard deviation; SE, standard error; WF, word fluency.

TABLE 2 Associations between MRI measures and categorical sleep duration (N = 2334).

	TCB		Combined gray		Frontal gray	
	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]
	M1	M2	M1	M2	M1	M2
6-9 h	Ref	ref	Ref	ref	ref	ref
<6 h	0.05 [-0.11; 0.22]	0.05 [-0.11; 0.21]	-0.18 [-0.38; 0.02]	-0.13 [-0.31; 0.05]	-0.12 [-0.33; 0.09]	-0.07 [-0.26; 0.12]
>9 h	-0.12 [-0.25; 0.02]	-0.08 [-0.21; 0.04]	-0.20* [-0.38; -0.03]	-0.17* [-0.31; -0.02]	-0.15 [-0.33; 0.04]	-0.11 [-0.26; 0.03]
	Occipital gray		Temporal gray		Parietal gray	
	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]
	M1	M2	M1	M2	M1	M2
6-9 h	Ref	ref	Ref	ref	ref	ref
<6 h	-0.25* [-0.47; -0.03]	-0.20 [-0.41; 0.01]	-0.11 [-0.35; 0.08]	-0.10 [-0.29; 0.09]	-0.05 [-0.26; 0.15]	-0.02 [-0.21; 0.16]
>9 h	-0.21** [-0.36; -0.05]	-0.18* [-0.33; -0.04]	-0.11 [-0.29; 0.06]	-0.10 [-0.27; 0.07]	-0.12 [-0.30; 0.06]	-0.09 [-0.25; 0.07]
	Lateral Ventricles		WMH		Hippocampus	
	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]
	M1	M2	M1	M2	M1	M2
6-9 h	Ref	ref	Ref	ref	ref	ref
<6 h	-0.14 [-0.33; 0.05]	-0.16 [-0.35; 0.03]	-0.05 [-0.23; 0.13]	-0.04 [-0.22; 0.14]	0.04 [-0.17; 0.26]	0.05 [-0.17; 0.27]
>9 h	0.10 [-0.04; 0.23]	0.06 [-0.07; 0.18]	0.08 [-0.07; 0.23]	0.05 [-0.09; 0.19]	-0.13 [-0.29; 0.03]	-0.14 [-0.29; 0.02]

Note: M1 model includes time between visits (in years), continuous age, sex (male, female), Hispanic/Latino background (South American, Central American, Mexican, Cuban, Puerto Rican, and Dominican), a categorical measure for testing center (San Diego, Miami, Chicago, Bronx), and continuous respiratory event index.

Abbreviations: CI, confidence interval; MRI, magnetic resonance imaging; TCB, total cranial volume; WMH, white matter hyperintensity.

 $p < 0.05$. $p < 0.05$.
 $p < 0.01$.

TABLE 3 Associations between MRI measures and continuous sleep duration (N = 2334).

	TCB		Combined gray		Frontal gray	
	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]
	M1	M2	M1	M2	M1	M2
Continuous sleep	−0.05** [−0.09; −0.01]	−0.05** [−0.08; −0.01]	−0.02 [−0.07; 0.02]	−0.02 [−0.06; 0.02]	−0.01 [−0.06; 0.03]	−0.01 [−0.05; 0.03]
	Occipital gray		Temporal gray		Parietal gray	
	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]
	M1	M2	M1	M2	M1	M2
Continuous sleep	−0.00 [−0.04; 0.04]	−0.00 [−0.04; 0.04]	−0.03 [−0.08; 0.02]	−0.03 [−0.07; 0.02]	−0.01 [−0.06; 0.04]	−0.01 [−0.06; 0.03]
	Lateral ventricles		WMH		Hippocampus	
	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]	β [95% CI]
	M1	M2	M1	M2	M1	M2
Continuous sleep	0.04 [−0.00; 0.08]	0.03 [−0.01; 0.07]	0.01 [−0.02; 0.05]	0.01 [−0.03; 0.05]	−0.04 [−0.09; 0.00]	−0.05 [−0.09; 0.00]

Note: M1 model includes time between visits (in years), continuous age, sex (male, female), Hispanic/Latino background (South American, Central American, Mexican, Cuban, Puerto Rican, and Dominican), a trichotomous indicator for education (less than high school, high school or equivalent, more than high school). M2 model additionally includes language preference (English, Spanish), continuous measure for body mass index, a dichotomous indicator for sleep quality (Restless/very restless, Average/restful/very restful), continuous depression, a binary measure for hypertension (no hypertension, yes hypertension), a categorical measure for testing center (San Diego, Miami, Chicago, Bronx), and continuous respiratory event index.

Abbreviations: CI, confidence interval; MRI, magnetic resonance imaging; TCB, total cranial volume; WMH, white matter hyperintensity.

** $p < 0.01$.

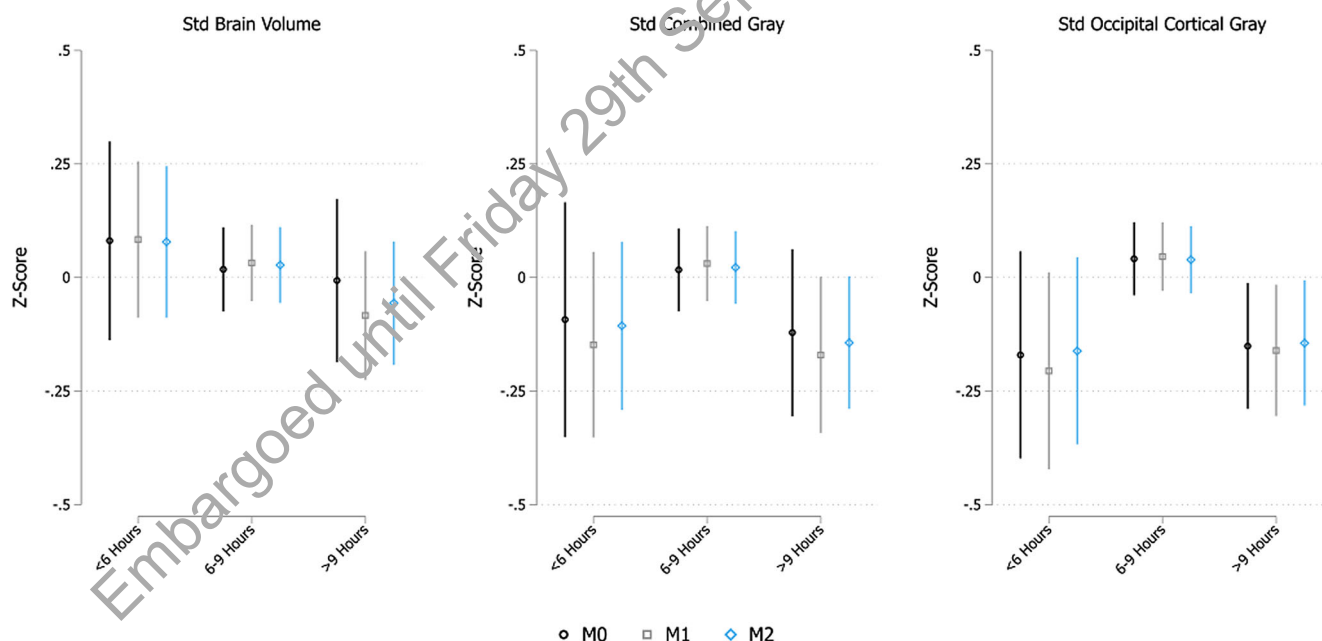


FIGURE 1 Associations between brain measures and categorical sleep duration (N = 2334). Notes: M0 model includes time between visits (in years). M1 model includes time between visits (in years); continuous age, sex (male, female), Hispanic/Latino background (South American, Central American, Mexican, Cuban, Puerto Rican, and Dominican); and trichotomous indicator for education (less than high school, high school or equivalent, more than high school). M2 model additionally includes language preference (English, Spanish), continuous measure for body mass index, a dichotomous indicator for sleep quality (Restless/very restless, Average/restful/very restful), continuous depression, a binary measure for hypertension (no hypertension, yes hypertension), a categorical measure for testing center (San Diego, Miami, Chicago, Bronx), and continuous respiratory event index. Std = standardized; TCB, total cranial volume; WMH, white matter hyperintensity.

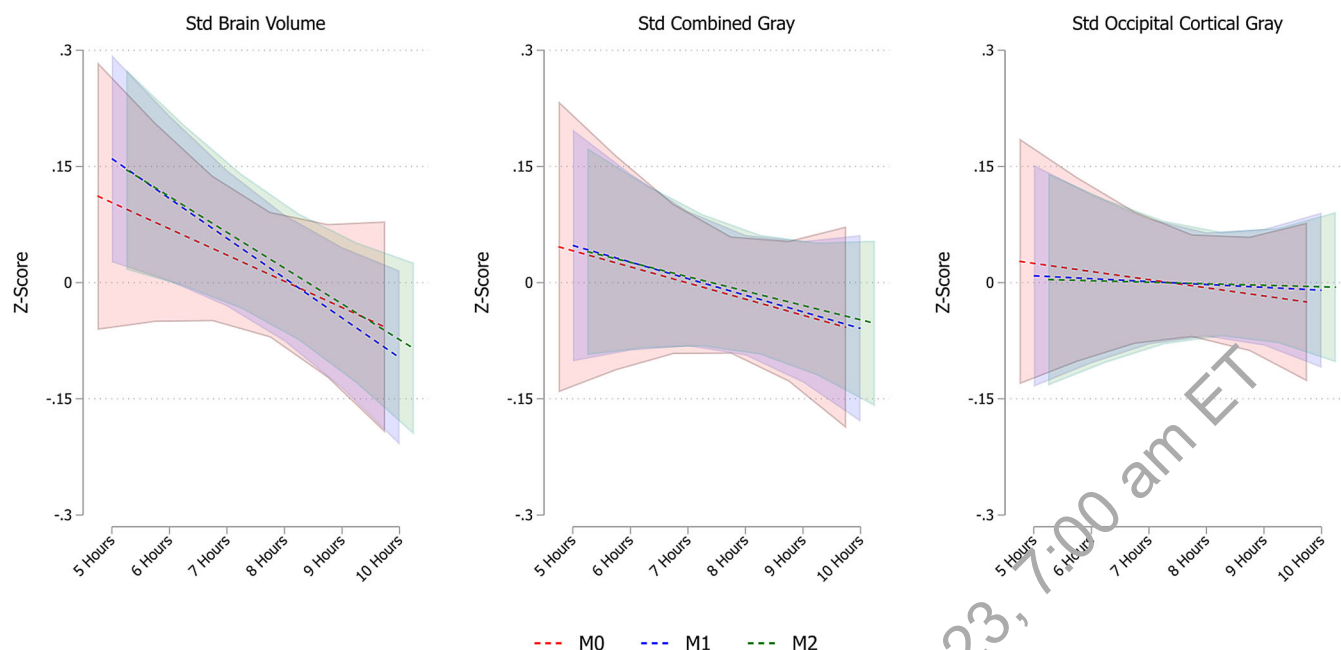


FIGURE 2 Associations between brain measures and continuous sleep duration ($N = 2334$). Notes: M0 model includes time between visits (in years). M1 model includes time between visits (in years); continuous age, sex (male, female), Hispanic/Latino background (South American, Central American, Mexican, Cuban, Puerto Rican, and Dominican), a trichotomous indicator for education (less than high school, high school or equivalent, more than high school). M2 model additionally includes language preference (English, Spanish), continuous measure for body mass index, a dichotomous indicator for sleep quality (Restless/very restless, Average/restful/very restful), continuous depression, a binary measure for hypertension (no hypertension, yes hypertension), a categorical measure for testing center (San Diego, Miami, Chicago, Bronx), and continuous respiratory event index. TCB, total cranial volume; Std, standardized; WMH, white matter hyperintensity.

3.2 | Regression models

Results from categorical and continuous models are found in Tables 2 and 3 and average marginal estimates for the brain outcomes are displayed in Figures 1 and 2. A 1-hour increase in sleep was associated with smaller total brain volumes ($\beta_{\text{total_brain}} = -0.05 [-0.08; -0.01]$, $p < 0.01$) even after full covariate adjustment. In categorical models, those in the >9-hour sleep group had smaller gray matter ($\beta_{\text{combined_gray}} = -0.17 [-0.31; -0.02]$, $p < 0.05$) and occipital gray matter ($\beta_{\text{occipital_gray}} = -0.18 [-0.33; -0.04]$, $p < 0.05$) volumes compared to the 6–9 hour group. In quadratic models (Table S1), we found a linear and a quadratic association between sleep duration and WMHs; however, this association was explained away in the fully adjusted models. Associations with all other considered brain outcomes were not statistically significant.

Results derived from focusing on the middle-age and older (50+ years) cohort were consistent with findings from the overall study (Table S2). These results were robust to adjustment for baseline cognitive function, exclusion of individuals with suspect severe cognitive impairment, as well as correction for MCI status. Total brain associations were not significant in the young (<50 years) cohort after full covariate adjustment, but we found significant decrements in combined gray ($\beta_{\text{combined_gray}} = -0.14 [-0.22; -0.06]$, $p < 0.001$) frontal ($\beta_{\text{frontal_gray}} = -0.15 [-0.24; -0.05]$, $p < 0.01$), and parietal ($\beta_{\text{parietal_gray}} = -0.13 [-0.22; -0.03]$, $p < 0.01$) gray matter volume per hour increase in sleep (Tables S3).

Results from analyses focused on the high-risk OSA ($\text{REI} \geq 5$) group showed that a 1-hours increase in sleep was associated with smaller total brain volume ($\beta_{\text{total_brain}} = -0.06 [-0.10; -0.01]$, $p < 0.05$), larger WMHs ($\beta_{\text{WMH}} = 0.06 [0.01; 0.10]$, $p < 0.05$), and smaller hippocampal volume ($\beta_{\text{hippocampus}} = -0.07 [-0.14; -0.01]$, $p < 0.05$) in fully adjusted models (Tables S4).

3.3 | Modification effects

Modifications by background and sex are included in Tables S5 and S6, respectively. We did not find evidence for significant modifications by either of these groups in the considered models.

4 | DISCUSSION

Among Hispanic/Latino adults, we presented evidence for differential neuroimaging outcomes based on sleep duration. Long sleep duration was linearly associated with smaller total brain matter volumes. When sleep duration was treated categorically to distinguish between short, typical, and long sleepers, we found that long sleep was additionally associated with smaller gray matter and occipital gray matter volumes. Given the lack of evidence for curvilinear associations, we re-fit continuous models for subpopulation analysis. We found that continuous models were consistent for individuals in the middle-age

and older cohort even after adjusting for MCI status. In addition, young participants (<50 years) showed negative associations in gray matter areas including frontal, parietal, and total gray matter. In the high-risk OSA group ($REI \geq 5$), a 1-hour increase in sleep was associated with smaller brain and hippocampal volume, as well as larger WMHs volume. Other work that has examined sleep dysfunction-related markers of brain disease suggest mixed findings. Data from 122 middle age and older (56–86 years) individuals in the Baltimore Longitudinal Study of Aging Neuroimaging Study showed that compared to sleeping 7 hours a day, longer and shorter sleep were both associated with frontotemporal gray matter loss.³³ This is consistent with more recent work demonstrating an inverted-U shape relationship between sleep duration and gray matter volumes, with larger values among those that slept between 6.7 and 7 hours.³⁴ Other studies have found null results. The Atherosclerosis Risk in Communities (ARIC) Study found no association between short sleep duration and brain measures in a middle-aged cohort ($n = 312$).³⁵ Our research expands on previous work by (1) linking subjective sleep duration to brain volumes in a large diverse Latino/Hispanic population and (2) testing for possible sex and background interactions. In previous HCHS/SOL studies, we have found both short and long sleep to be linked with worse cognitive performance in word fluency, memory, and processing speed.³⁶ The Memory and Aging Project linked these to decrements in gray matter volumes.³⁷ Our findings suggest that sleep might follow a “J” shaped relationship with MRI brain volumes. Furthermore, our subpopulation analysis highlights the importance of sleep duration as an important marker for brain health. Young participants (<50 years) were 24–39 years during their sleep visit and presented additional decrements in gray matter volume compared to middle-aged and older participants. One possible explanation for these findings is that long sleep duration is a marker for underlying cardiovascular or psychological conditions, and these could have stronger effects in younger adults. For example, one study found that affective disorders in young participants is associated with decreased frontal gray matter.³⁸ Another possible contributor is drinking, which has also been linked to decreased total gray and frontal gray matter in young adults.³⁹ Finally, our results in the high-risk OSA group ($REI \geq 5$) complement other studies that have found sleep apnea to be linked with increased WMHs volume⁴⁰ and changes in hippocampal volumes.⁴¹ These findings suggest that, regardless of the number of respiratory events, sleep duration could be an important factor for understanding brain health in this at-risk population.

Subjective sleep duration could be a surrogate for slow wave sleep. Slow wave sleep, also known as N3 sleep, is believed to serve many important functions during sleep,⁴² and reduced N3 sleep has been associated with smaller brain and larger WMH volumes.⁴³ Short sleep has also been linked to increased amyloid beta (A β) deposition,⁴⁴ which can lead to smaller gray matter volumes.⁴⁵ Sleep duration could interact with brain volumes through cerebrovascular and cardiovascular pathways. A meta-analysis found self-reported sleep to have a J-shaped relationship with mortality and incident cerebrovascular disease (CVD): those who slept less or more than 7 to 8 hours had increased risk of CVD and mortality, and these effects were stronger for those with longer sleep.⁴⁶ Lifestyle factors, such as reduced physical activity,⁴⁷ could partly explain the association between long sleep

duration and smaller brain volumes.⁴⁸ Increased WMHs in the high-risk OSA population could suggest sleep disruptions may be linked to cardiovascular or cerebrovascular pathways. Indeed, WMHs have been found to be potentially crucial biomarkers for CVD.⁴⁹ Because we did not assess the temporality of CVD and sleep, it is possible that short or long sleep are health sequelae of CVD (or other factors) and thus serve more as a marker of other complications rather than a risk factor for brain atrophy.

Despite notable brain differences between men and women, we did not find evidence for sex modification in sleep duration. Men, on average, have larger brain and cranial volume as well as white and gray matter volumes.⁵⁰ Recently, Stickel et al.⁵¹ reported that although Hispanic/Latino men on average have bigger brain volumes at younger ages, this relationship, relative to Hispanic/Latina women, shifts in older age. Sex differences, although not found in the limited context of this study, deserve further focus. Kim et al., for example, found evidence of curvilinearity in parietal, frontal, and total gray matter for men but not women.³⁴ Previous SOL-INCA MRI found that men, on average, have larger WMH volumes compared to women.⁵² Although not statistically significant, the interaction model with WMHs had a low p value ($p < 0.10$). We also did not find Hispanic/Latino background modifications. DeCarli et al.⁵³ reported that Dominicans and Puerto Ricans had the largest residualized gray matter volumes, whereas Cuban, South Americans, and Central Americans had the smallest. Our results suggest that despite these differences, the associations between sleep duration and brain MRI volumes are not modified by Hispanic/Latino groups.

4.1 | Strengths and limitations

This study has many strengths. SOL-INCA MRI is the largest and most representative sample of diverse Hispanics/Latinos with neuroimaging markers of brain health. This has allowed us to validate our findings in clinical meaningful subpopulations. Our study also benefits from multimodal measures, thus allowing us to adjust for important confounders such as REI, depression, baseline global cognition, and cardiovascular disease. This study also has several limitations. Sleep duration was measured subjectively. Subjective methods to measure sleep duration are correlated moderately with actigraphy-based methods,⁵⁴ although they tend to overreport total sleep.⁵⁵ Despite using self-reported measures, we used well-validated questionnaires.⁵ Future research should validate these results with actigraphy-based methods, or with measures of sleep architecture. We have used probability weights so our results can be generalized to the target population. Finally, MRI data acquisition was performed using multiple scanners, but we accounted for this by adjusting for testing center in models.

5 | CONCLUSIONS

Sleep duration was an important independent indicator for understanding brain health and managing healthy brain aging across sex

and background groups. These effects could have significant clinical implications and help guide future guidelines and treatment.

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CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interests to report. Author disclosures are available in the [supporting information](#).

CONSENT STATEMENT

Participants provided written informed consent, and institutional review boards approved the study at each of the coordinating centers.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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