



Does poor sleep impair cognition during aging? Longitudinal associations between changes in sleep duration and cognitive performance among older Mexican adults

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ABSTRACT

Alterations in sleep patterns are common among older adults; further, short and long sleep durations have been linked with impaired cognitive performance in older individuals. Yet most research examining these relationships has been cross-sectional, limited to high-income nations, and has failed to consider how changes in sleep duration may impact cognitive decline. The present longitudinal study uses nationally-representative data to test whether changes in sleep length among “healthy” baseline sleepers are associated with reduced cognitive function in older Mexican adults (≥ 50 years old) at follow-up.

Data were drawn from the first and second waves of the World Health Organization’s Study on global AGEing and adult health. Self-report data captured sleep duration over two nights, and five cognitive tests (immediate and delayed verbal recall, forward and backward digit span, and verbal fluency) were used to measure various cognitive domains and create a composite z-score of cognitive performance. Linear regressions were performed to assess associations between sleep length changes and cognitive decline, controlling for relevant lifestyle and health factors.

Increased sleep durations at follow-up among individuals who reported intermediate sleep durations (6–9 h/night) at baseline were significantly associated with greater rates of decline in overall cognitive function. Longer sleepers also trended toward greater rates of decline for attention/working memory and executive function. This study suggests that long sleep durations are a risk factor for certain types of impaired cognition among older adults living in a middle-income country. These findings are clinically important given the growing rates of dementia and aging populations globally.

1. Introduction

Detrimental changes in sleep duration often occur as individuals age (Bombois, Derambure, Pasquier, & Monaca, 2010; Descamps & Cespuglio, 2010), with older adults reporting higher rates of sleep disorders associated with disrupted and fragmented sleep patterns. These age-related sleep alterations are hypothesized to increase risk of cognitive decline in older adults, and both short and long sleep duration have been linked with impaired cognitive function (Chen et al., 2016;

Crenshaw & Edinger, 1999; Devore, Grodstein, & Schernhammer, 2016; Faubel et al., 2009; Lo, Groeger, Cheng, Dijk, & Chee, 2016). Sleep deprivation dulls the senses, slows the reflexes, and impedes ability to recall information (Crenshaw & Edinger, 1999; Devore et al., 2016; Lo et al., 2016). Conversely, long sleep duration is thought to reflect poor sleep quality, physiological dysregulation, or disturbed sleep patterns – perhaps due to underlying illness – which may impair cognitive performance (Devore et al., 2016; Faubel et al., 2009; Lo et al., 2016; Tsapanou et al., 2017).

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In addition, chronic problems related to sleep latency (time to sleep onset) and maintenance are frequently associated with adverse consequences, including drowsiness and reduced performance on cognitive tests (Nebes, Buysse, Halligan, Houck, & Monk, 2009). Recurrent sleep problems which detrimentally affect sleep maintenance and duration can therefore impair daily functioning, increase risk of accidents, and negatively impact the physical and psychosocial status of older individuals. These patterns have been documented cross-culturally. For example, previous work conducted as part of the World Health Organization's Study on global AGEing and adult health (SAGE) has demonstrated significant cross-sectional relationships between sleep duration and cognitive performance (Gildner, Liebert, Kowal, Chatterji, & Snodgrass, 2014). This study found that individuals with intermediate sleep durations (6–9 h/night) exhibited significantly higher cognitive scores than individuals with short (< 6 h/night) or long (> 9 h/night) sleep durations. This suggests that healthy sleep patterns are linked with improved cognition; however, this work was cross-sectional in nature.

Longitudinal work is needed to clarify how changes in sleep patterns might impact cognitive performance, especially in lower-income nations where comparatively little research on these topics have been conducted. Relatively few studies have tested how changes in sleep duration over time in association with cognitive function (Devore et al., 2016), although these few studies indicate that both decreased (Devore et al., 2014; Ferrie et al., 2011; Virta et al., 2013) and increased (Devore et al., 2014; Ferrie et al., 2011; Loerbroks, Debling, Amelang, & Stürmer, 2010; Virta et al., 2013) sleep durations over time are related to poor cognitive function at follow-up. Even less work has been done examining how changes in sleep duration are associated with differences in cognition between multiple time points; this research has produced non-significant or mixed results (Devore et al., 2014).

Additional work is therefore needed to clarify whether detrimental changes in sleep duration are linked with reduced cognitive function, as has been hypothesized. This information is critical since the ability to minimize age-related cognitive deficits are dependent on the identification of key modifiable factors. These clinical interventions have important implications toward addressing the global burden of dementia, which is projected to more than triple by the year 2050 (WHO, 2012), placing an increased burden on family and caregivers. This is especially true in middle-income countries like Mexico, which often lack the healthcare and social welfare institutions required to support a growing population of older adults (Aguila, Diaz, Fu, Kapteyn, & Pierson, 2012). Thus, improving behavioral and lifestyle factors (e.g., sleep patterns) during aging may represent an important strategy for maintaining cognitive function at older ages (Haimov & Shatil, 2013; Keage et al., 2012).

The present study examines links between changes in sleep duration and cognitive test performance in a middle-income country using longitudinal data drawn from SAGE (Kowal et al., 2012). Nationally representative data from Mexico were utilized to construct a more comprehensive picture of whether changes in sleep duration from baseline measures substantially impact cognitive performance among older adults at follow-up. The following hypothesis was tested: among older adults with intermediate baseline sleep durations (6–9 h/night), substantial changes in sleep duration at follow-up will be associated with reduced cognitive performance.

2. Materials and methods

2.1. Study design and participants

A nationally-representative sample of older Mexican adults (≥ 50 years old) was collected using a stratified multistage cluster sample design (Kowal et al., 2012; Naidoo, 2012). In-person interviews were used to collect baseline household and individual level data between 2009/10 (Wave 1), and follow-up data collected in 2014 (Wave 2).

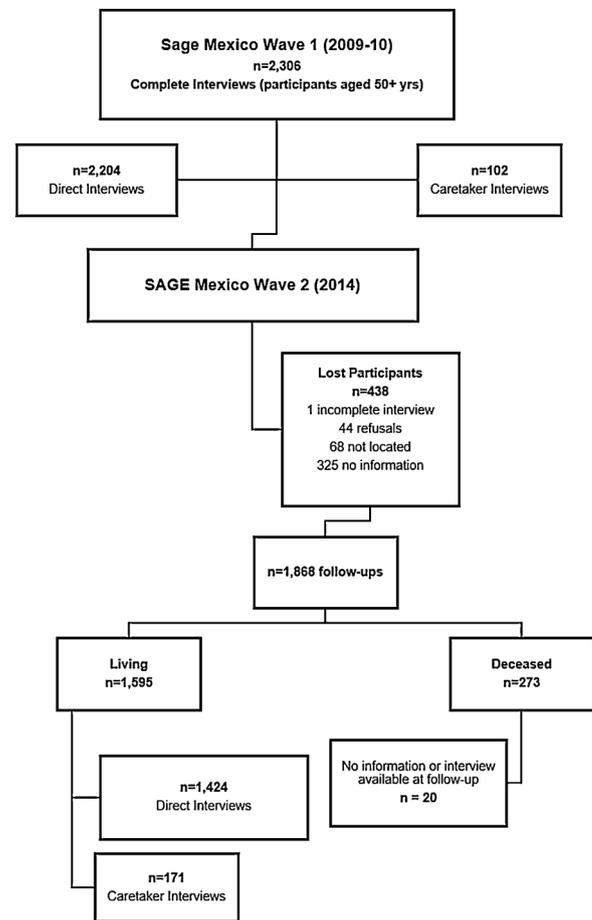


Fig. 1. Flowchart of the SAGE Mexico design depicting participant retention between Waves 1 and 2.

Specifically, three strata were defined by locality (metropolitan, urban, rural); within these strata, the Basic Geo-Statistical Areas defined by the National Institute of Statistics were used as primary sampling units. Households within these sampling units were randomly selected and constituted the secondary sampling units. Household weights were post-stratified by locality according to population census projections. Finally, individuals within households made up tertiary sampling units. Individual weights were post-stratified by sex and age-groups (18–34, 35–49, 50–59, 60–105) according to the census projections. Thus, household and individual weight calculations are based on the selection probability at each stage of selection (Kowal et al., 2012; Naidoo, 2012).

Of the 1,595 living individuals who participated in both Wave 1 and Wave 2 of data collection (Fig. 1), those missing one or more of the variables of interest were excluded from regression analysis ($n = 648$), resulting in a final sample size of 947 participants. The missing adults were younger on average than those included in analyses, but otherwise no substantive differences were observed. To test the hypothesis, a subset of these participants was used during analysis; namely, those with intermediate sleep durations (6–9 h/night) at baseline ($n = 782$).

2.2. Ethical approval

SAGE was approved by the World Health Organization's Ethical Review Committee. Additionally, the Research and Ethics Committees of Mexico's National Institute of Public Health approved SAGE Mexico. Written informed consent was obtained from all study participants.

2.3. Cognition variables

The present study used five standard cognitive tests – administered at both baseline and follow-up – to test associations between sleep duration and cognition. The five measures employed here are associated with different aspects of cognition, including: new verbal learning and recall (immediate and delayed verbal recall), attention/working memory (forward and backward digit span), and executive function (verbal fluency) (e.g., see Cullen, O'Neill, Evans, Coen, & Lawlor, 2007 for review), providing a more complete picture of how sleep length may impact distinctive cognitive domains.

For the immediate verbal recall test, interviewers read a list of 10 words aloud and asked the participants to immediately recall as many words as they could in one minute. Three trials of this assessment were performed. Upon completing the third trial, the interviewer administered the other cognitive tests, after which delayed recall ability was determined by asking subjects to remember the list of words without the interviewer repeating the list. The digit span test required participants to repeat progressively longer series of numbers; the total score was recorded as the longest digit span repeated without error. This process was then performed with the respondent repeating new sets of increasingly longer digit spans in reverse. The verbal fluency test consisted of naming as many animals (without using proper nouns) as possible in one minute; the final score was correct responses minus errors.

In accordance with other cognitive studies, composite z-scores were calculated to facilitate the comparison of cognitive test performance between individuals (e.g., Devore et al., 2014; Gildner et al., 2014; Scarmeas, Albert, Manly, & Stern, 2006; Tsapanou et al., 2017). This approach accounts for the fact that the cognitive tests were scaled differently, allowing the various test scores to be meaningfully compared and used to calculate an overall, composite cognitive performance score. Specifically, z-scores for each cognitive test were first computed (relative to the sample mean) and these five z-scores were then summed for each participant, resulting in a final composite z-score. Additionally, the change in participant composite cognition score between Waves 1 and 2 was calculated by subtracting the baseline composite cognitive z-score from the composite z-score at follow-up.

2.4. Sleep duration

Average sleep durations at baseline (Wave 1) and follow-up (Wave 2) were calculated. Participants were asked their sleep duration on each of the preceding two nights; these measures did not include daytime sleep. In accordance with other sleep studies (Faubel et al., 2009; Patel, Malhotra, Gottlieb, White, & Hu, 2006), the duration values across two nights were averaged together to create a summary measure of sleep length so mean sleep duration could be compared. Following a standard approach (Faubel et al., 2009; Ohayon & Vecchierini, 2005; Tamakoshi & Ohno, 2004), sleep duration was categorized to identify individuals with “healthy”, intermediate sleep durations at baseline (6–9 h/night). These cutoff points were selected based on common medical definitions of short and long sleep (e.g., Mukherjee et al., 2015), while also remaining consistent with previous SAGE research (e.g., Gildner et al., 2014). However, varying definitions of “optimal” sleep duration exist (Cappuccio, D'Elia, Strazzullo, & Miller, 2010); a second set of sleep category cut-off points was therefore used during analysis, with individuals reporting 7–9 h/night categorized as “healthy” baseline sleepers. Both sets of sleep categories were tested during analysis. Finally, the change in participant sleep duration between Waves 1 and 2 was calculated by subtracting average baseline sleep duration from average sleep duration at follow-up.

2.5. Covariates

Variables demonstrated in past studies to impact cognitive

performance were controlled for in all statistical analyses. Evidence suggests that participant sex and age influence cognitive decline (Beydoun et al., 2012; Mielke, Vemuri, & Rocca, 2014), these variables were therefore included in all analyses. In addition, education level was classified using the International Standard Classification of Education (UNESCO, 1997) and included during analysis. Given that ethnic background can influence health patterns (Alvarado-Esquivel et al., 2004), participants were also asked about their ethnicity and this response was included during analysis. History of unemployment has also been linked with cognitive performance (Alvarado-Esquivel et al., 2004), thus employment status was also included in the analyses. Moreover, previous research suggests that individuals living in rural settings exhibit a higher prevalence of dementia compared to urban areas (Jia et al., 2014). Participant household setting (urban vs. rural) was therefore controlled for during analysis.

Underlying medical conditions known to influence cognitive function were also added to all models (depression, prior diagnoses of stroke, hypertension, diabetes, and chronic lung disease) as a summed total of all diagnosed comorbidities (Iadecola, 2013; Kessler et al., 2010; Kling, Trojanowski, Wolk, Lee, & Arnold, 2013). Depression status was determined using symptom-based reporting and an algorithm to assign diagnosis based on the World Mental Health Composite International Diagnostic Interview (Kessler et al., 2010). It is worth noting that depression demonstrates a complex relationship with sleep patterns (Roberts, Shema, Kaplan, & Strawbridge, 2000), however mediation analyses indicated that depression diagnosis does not significantly mediate the relationship between sleep duration and cognitive performance in this sample ($Z_{\text{med}} = 0.009$, $|0.009| \leq 1.96$). Participants were also asked about medication use, including use of opioids, anxiolytics, sedatives, and stimulants. From this information a binary medication variable was created (not taking any medications vs. taking at least one) and included during analysis.

Health-related factors known to influence cognitive function (body mass index, physical activity levels, gait speed, tobacco use, and drinking patterns) were also included in all analyses (Anttila et al., 2004; Laurin, Verreault, Lindsay, MacPherson, & Rockwood, 2001; Ott et al., 1998). Specifically, participant height and weight were collected, and these values were used to calculate BMI (kg/m^2). Physical activity level was measured based on responses to questions drawn from the Global Physical Activity Questionnaire (WHO, 2009). As has been described elsewhere (Rivas-Marino et al., 2015), three categories of physical activity (low, moderate, and high physical activity) were calculated from these questionnaire items, based on reported time spent in moderate or vigorous activities during work, recreational/leisure time, and transportation.

Previous research indicates that gait speed – a measure of time required to walk a set distance (in m/s) – represents an important indicator of physical function that may be linked with cognitive function (e.g., Buracchio, Dodge, Howieson, Wasserman, & Kaye, 2010; Watson et al., 2010). Participant gait speed was therefore included during analysis. Gait speed was assessed through timed walks over a 4-meter flat area. The lengths of time required to travel this distance at both the participant's usual and rapid walking speed were recorded, and normal and rapid gait speed were calculated in meters per second (m/s). Height-standardized z-scores for usual and rapid gait speed were calculated and these values were used during analysis. Given the complex relationship proposed between gait speed and cognition (e.g., Buracchio et al., 2010; Watson et al., 2010), preliminary analyses were conducted testing whether gait speed mediated the relationship between sleep duration and cognitive performance. However, these mediation analyses indicated that neither normal nor rapid gait speed significantly mediated the relationship between sleep duration and any of the cognitive function measures in this sample ($Z_{\text{med}} = 0.167$ – 1.67 , all values ≤ 1.96). Normal and rapid gait speed were therefore included during analysis as covariates. Respondents were also asked if they had ever used tobacco or consumed alcohol and if participants

Table 1

Description of the study population (n = 782; intermediate baseline sleepers) at follow-up (weighted data), data were drawn from baseline SAGE Wave 1 (2009–10) and follow-up Wave 2 (2014).

Variables	Mean	SE
Age	62.12	1.06
Normal gait speed (in m/s)	0.801	0.026
Rapid gait speed (in m/s)	1.16	0.037
Follow-up interval (in days)	1727.82	2.15
Baseline composite cognitive z-score	0.122	0.246
Change in composite cognitive z-score	-0.172	0.185
Change verbal recall z-score	0.056	0.061
Change in forward digit span z-score	0.374	0.090
Change in backward digit span z-score	-0.019	0.083
Change in verbal fluency z-score	-0.665	0.116
Change in delayed verbal recall z-score	0.082	0.086
Change in sleep duration	-0.339	0.189
	Proportion	S.E.
Male sex (vs. female)	0.446	0.061
Live in urban area (vs. rural)	0.703	0.059
No formal education	0.156	0.046
Less than high school	0.698	0.050
High school or beyond	0.146	0.033
Positive depression diagnosis	0.131	0.025
Positive stroke diagnosis	0.025	0.010
Positive chronic lung disease diagnosis	0.029	0.011
Positive hypertension diagnosis	0.277	0.035
Positive diabetes diagnosis	0.263	0.054
Positive medication use	0.100	0.026
Low physical activity level	0.537	0.062
Moderate physical activity level	0.183	0.030
High physical activity level	0.280	0.057
Never used tobacco	0.763	0.049
Not currently using tobacco	0.115	0.030
Use tobacco occasionally	0.019	0.006
Use tobacco daily	0.102	0.042
Never consumed alcohol	0.559	0.053
Not currently drinking	0.141	0.023
Drink < 1/month	0.206	0.052
Drink 1-3 days/month	0.077	0.042
Drink 1-4 days/week	0.010	0.004
Drink 5+ days/week	0.006	0.003

answered affirmatively the frequency of use was recorded (see Table 1). Finally, following similar studies (Jelicic et al., 2002), baseline cognitive performance and length of the time interval between the first and second participant interview (i.e., time between baseline and follow-up measures) were included in the analyses.

2.6. Statistical analyses

Tests for normality were performed, and no violations were observed. Multicollinearity was not detected between any of the variables. Parametric tests were subsequently conducted using Stata version 14 (StataCorp LLC, College Station, Texas, USA) to test the hypotheses. Sample weights were used in all analyses (Naidoo, 2012). A Bonferroni correction was applied to account for multiple comparisons (i.e., the six cognitive variables tested) and *p*-values were considered significant at less than 0.008. A series of linear regressions were conducted to test the hypotheses and evaluate the relative contribution of sleep duration to changes in cognitive score.

Initial analyses indicated that participant reported ethnicity, employment status, and BMI did not significantly contribute to any statistical model. These variables were consequently dropped from analysis. All subsequent analyses controlled for participant sex, age, education level, household setting, number of diagnosed comorbidities, medication use, physical activity level category, gait speed (both usual and rapid), tobacco and alcohol use, baseline cognitive score, and follow-up interval. Preliminary analyses examining the association

between baseline sleep duration and changes in cognitive test performance between Waves 1 and 2 (controlling for all covariates) indicated that sleep duration category at baseline (short, intermediate, or long) was not significantly associated with change in cognitive test score for any of the six cognitive function measures; this was true for both calculated sleep duration categories (i.e., categorizing intermediate sleepers as individuals reporting 6–9 h/night or 7–9 h/night). However, the majority of participants reported an intermediate sleep duration at baseline (n = 782, 83% reported 6–9 h/night; n = 647, 68% reported 7–9 h/night). This lack of variation in sleep duration category may account for the absence of statistically significant relationships between baseline sleep duration and change in cognition. Of greater interest is how changes in sleep patterns among this large number of seemingly healthy baseline sleepers may be associated with detrimental alterations to cognitive performance over time.

Still, it should be noted that the relationship between cognitive decline and sleep patterns is complex and possibly bidirectional. For example, Alzheimer's disease appears to cause circadian disruption and disturbed sleep patterns (Ju, Lucey, & Holtzman, 2014). To account for the potential influence of impaired cognition at baseline on subsequent sleep patterns at follow-up, sensitivity analyses were conducted. Based on previous methods (Vancampfort et al., 2017), SAGE participants exhibiting mild cognitive impairment at baseline (Wave 1) were identified. These individuals (n = 136) were then excluded from analysis during hypothesis testing; however, the exclusion of these participants did not substantively change the study results and they were subsequently included in the final analyses to increase sample size and statistical power.

2.6.1. Descriptive statistics

Average change in sleep duration or cognition (for each cognitive measure) were calculated. In addition, average values or frequencies for all other variables of included in the models were calculated.

2.6.2. Examination of the relative contribution of sleep duration change to variation in cognitive decline, among participants with intermediate baseline sleep durations

A series of linear regressions examined if changes in average sleep duration contributed to changes in cognitive test performance (the composite cognitive score and each of the individual cognitive performance tests) among participants with intermediate baseline sleep durations (6–9 h/night). Given the similar results evident between the two different classifications of intermediate sleep (6–9 h/night vs. 7–9 h/night), the classification of 6–9 h/night was retained to maximize sample size, increase statistical power, and remain consistent with previous SAGE research (e.g., Gildner et al., 2014). The change in sleep duration between Waves 1 and 2 was entered into these models.

3. Results

Descriptive statistics are presented in Table 1. The sample was composed of slightly more women than men, lived in an urban area, had received some formal schooling (but lacked a high school degree), had not been diagnosed with one of the underlying medical conditions, did not report high rates of medicine use, reported low physical activity levels, and had never used tobacco or consumed alcohol. Average age at follow-up was roughly 62 years, mean normal gait speed was 0.801 m/s, mean rapid gait speed was 1.16 m/s and average follow-up interval was 4.7 years (between Waves 1 and 2).

As predicted, changes in sleep durations between baseline and follow-up were associated with greater cognitive decline among participants reporting intermediate sleep durations at baseline, such that increased sleep durations among this sample were associated with greater negative changes in certain aspects of cognition (Table 2; for full models see Appendix A). Specifically, a significant inverse relationship between change in sleep duration and magnitude of change

Table 2

Multiple regression models (using weighted data) for the prediction of changes in cognitive performance score from change in sleep duration between Waves 1 and 2 among participants with intermediate sleep duration (6–9 h/night) at Wave 1 (n = 782); B coefficients (SE). All analyses controlled for participant sex, age, education level, household setting, number of diagnosed comorbidities, medication use, physical activity level category, gait speed, tobacco and alcohol use, baseline cognitive score, and follow-up interval. See Appendix A for full models.

Composite Cognition	Immediate Verbal Recall	Forward Digit Span	Backward Digit Span	Verbal Fluency	Delayed Verbal Recall
-0.291 (0.099)**	-0.012 (0.027)	-0.050 (0.042)	-0.096 (0.039)*	-0.103 (0.054)*	-0.032 (0.042)

Comparisons are statistically significant (with Bonferroni adjustment) at: * = trend toward significance ($p < 0.06$), ** = $p < 0.008$, *** = $p < 0.001$. The Significant of bold values are composite cognition ($p = 0.004$), backward digit span ($p = 0.016$), and verbal fluency ($p = 0.059$).

in overall cognitive performance was evident ($B = -0.291, p = 0.004$). Likewise, the inverse association between changes in sleep duration and cognitive performance change in backward digit span trended toward significance ($B = -0.096, p = 0.016$), and a similar inverse relationship trended toward significance for cognitive performance change in verbal fluency ($B = -0.103, p = 0.059$). Overall, these findings suggest that increased sleep durations at follow-up are associated with greater cognitive impairment in select measures of cognitive performance.

4. Discussion

This study provides a unique examination of longitudinal associations between sleep duration change and cognitive decline in older Mexican individuals. Previous studies examining similar patterns have been confined to high-income countries and have generally relied on data collected from cross-sectional and non-representative population samples. The present study found partial support for the hypothesis. Among participants with intermediate sleep durations (6–9 h/night) at baseline, increased sleep durations at follow-up were associated with greater cognitive decline on certain measures of cognitive function, as was predicted. Still, while the relationships between long sleep and reduced backward digit span and verbal fluency scores trended toward significance, longer sleep was only significantly associated with changes in overall composite cognition.

There are several possible neurobiological explanations for these results. First, evidence suggests that long sleep durations may accelerate the rate of frontotemporal gray matter atrophy among older adults (Spira et al., 2016), potentially impairing memory. It is also possible that long sleep durations may reflect circadian disruptions linked with sleep dysregulation and impaired cognition (Devore et al., 2014; Yoo & Eckel-Mahan, 2016). Mounting evidence indicates that irregular circadian rhythms are associated with short-term cognitive impairment and longer-term brain atrophy (Cho, Ennaceur, Cole, & Suh, 2000; Cho, 2001; Machi et al., 2012); although these relationships appear to be complex. Moreover, the hormone melatonin plays an important role in shaping sleep patterns and has also been linked with cognitive function (e.g., memory performance) and rate of neurocognitive decline (Daulatzai, 2016; Hardeland, Pandi-Perumal, & Cardinali, 2006; Santoro, Giacheti, Rossi, Campos, & Pinato, 2016; Tsapanou et al., 2017). Abnormal melatonin levels may therefore influence the relationship between sleep duration and cognitive function.

The results documented in the present study are consistent with previous findings in high-income nations, which have also produced results indicating that long sleep negatively impacts aspects cognitive performance. For example, work among older adults living in Spain and the United States indicates that participants reporting long sleep durations exhibited significantly lower scores on mental tests related to attention and working memory (Faubel et al., 2009; Patel et al., 2006; Schmutte et al., 2007; Xu et al., 2011), as the results here suggest. This may reflect cognitive deficits linked with underlying health conditions and sleep disorders thought to impair sleep. Unhealthy individuals may suffer from disrupted sleep patterns and therefore require more time in bed to feel rested, leading these individuals to report longer sleep durations. However, longer reported sleep durations may not entirely

compensate for underlying health problems and cognitive decline may still occur. These kinds of disturbed sleep patterns have been linked with cognitive impairments (Blackwell et al., 2006), including tasks related to memory and attention.

However, systematic reviews of research examining these associations indicate that the relationship between long sleep and cognitive function is not consistent across studies (Devore et al., 2016; Lo et al., 2016). According to one systematic review of sleep and cognition literature, studies that have documented significant associations between long sleep duration and cognitive decline have all relied on self-reported sleep durations (Devore et al., 2016), such as the subjective measures used in the present study. Objective measures of sleep duration have failed to document a significant relationship between extreme sleep durations and cognitive decline (Devore et al., 2016). This suggests that it is the perception of sleep length, rather than actual time asleep, that is related to reduced cognitive performance. Additional research is therefore needed to clarify how subjective discernment of sleep patterns – perhaps shaped in part by participant health – might contribute to the development of disrupted sleep patterns and cognitive decline over time.

4.1. Future research directions

The present study provides novel data demonstrating significant relationships between increased sleep duration and decline in certain aspects of cognition. These findings could be expanded through the incorporation of underlying health conditions into future work examining relationships between sleep and cognition. For example, a next step would be to examine how various health conditions might occur jointly with detrimental changes in sleep duration (i.e., due to shared etiologies) and also serve as predictors of subsequent cognitive decline. In addition, future waves of SAGE data will help clarify the relationships between sleep length and cognitive function evident in the present study. Measuring changes in sleep patterns at additional time points may provide more insight into underlying health conditions which negatively impact sleep patterns (e.g., resulting in longer sleep durations), but only become apparent over time and can only be captured by assessing sleep patterns and cognitive performance at several time points. Likewise, additional waves of data may better enable the detection of cognitive decline linked with sleep patterns (i.e., these changes in cognition may only become apparent after longer follow-up periods) (Devore et al., 2014).

4.2. Limitations and strengths

The present study has important limitations. First, the SAGE questionnaire did not collect information on participant naps or sleep disorders (including insomnia), which may impact sleep durations and cognitive function. Second, the sleep data used were reliant on participant reported sleep duration from only the two nights of sleep prior to the interview, which may not accurately reflect typical sleep patterns. An additional limitation related to the use of self-reported sleep data is that individuals may not be able to distinguish between time in bed and time asleep. Evidence indicates that when asked to report hours slept,

participants often report time spent in bed; however, this value may exceed time spent asleep (Stenholm, Kronholm, Bandinelli, Guralnik, & Ferrucci, 2011). Objective measures of sleep based on polysomnography or actigraphy would better capture individual sleep patterns, including sleep duration, transitions between sleep states, and length of night awakenings (Lockley, Skene, & Arendt, 2002).

However, the use of objective sleep duration measurements affords the opportunity to examine how individual impressions of sleep patterns are associated with cognitive function, an especially important measure when assessing the contribution of long sleep durations to cognitive decline (Devore et al., 2016). This study also provides an important test of the relationship between sleep duration and several diverse measures of cognition by evaluating these variables across multiple waves of data collection, thereby examining how longitudinal changes in sleep may be associated with various aspects of cognitive decline in a nationally-representative population.

4.3. Conclusion

In conclusion, this study documented a significant association between longer sleep durations and increased overall cognitive decline among older Mexican adults reporting intermediate sleep durations at baseline. These results support previous findings in high-income populations and suggest that perceptions of long sleep are related to health conditions which negatively affect cognitive performance over time. Subjective measures of long sleep duration in older adults may therefore represent an important indicator of dementia risk. Furthermore, promoting healthy sleep habits (e.g., avoiding excessive time in bed and establishing sleep hygiene practices that enhance sleep quality) could represent an important consideration in future clinical studies aimed at mitigating cognitive decline in older individuals; not only in wealthy countries, but in lower- and middle-income populations as well.

Appendix A. Multiple regression models for prediction of changes in cognitive performance from continuous sleep duration change between Waves 1 and 2, among healthy sleepers (6–9 h/night) at Wave 1, B (SE) (n = 782)^{a,b}

	Composite Cognition	Immediate Verbal Recall	Forward Digit Spam	Backward Digit Spam	Verbal Fluency	Delayed Verbal Recall
Constant	–8.86 (13.12)	–2.57 (4.44)	4.46 (5.37)	–6.49 (5.66)	–15.78 (7.21)*	11.52 (8.23)
Sex	0.764 (0.306)*	0.236 (0.124)*	0.317 (0.156)*	0.034 (0.162)	–0.070 (0.162)	0.246 (0.109)*
Age: 60-69 years old	0.113 (0.366)	–0.099 (0.118)	0.156 (0.181)	–0.140 (0.161)	0.235 (0.221)	–0.039 (0.162)
70-79 years old	–1.07 (0.367)**	–0.475 (0.141)**	0.126 (0.169)	–0.076 (0.203)	–0.324 (0.195)	–0.321 (0.179)
80-89 years old	–1.99 (0.553)***	–0.534 (0.225)*	–0.141 (0.260)	0.057 (0.204)	–1.03 (0.304)**	–0.348 (0.228)
90+ years old	–2.06 (0.932)*	–0.376 (0.283)	–0.274 (0.357)	–0.237 (0.333)	–0.619 (0.487)	–0.553 (0.561)
Household setting	–0.787 (0.298)*	0.094 (0.130)	–0.223 (0.126)	–0.074 (0.145)	–0.508 (0.264)*	–0.075 (0.204)
Education: < high school	0.636 (0.334)*	–0.082 (0.143)	–0.239 (0.210)	0.626 (0.185)**	0.148 (0.188)	0.182 (0.232)
high school or beyond	2.21 (0.527)***	0.338 (0.187)	–0.148 (0.275)	1.01 (0.262)***	0.535 (0.325)	0.476 (0.295)
Physical activity level: moderate	–0.625 (0.314)*	0.177 (0.113)	–0.186 (0.137)	–0.184 (0.147)	–0.359 (0.228)	–0.073 (0.159)
high	0.399 (0.386)	0.036 (0.133)	0.109 (0.162)	0.083 (0.157)	–0.095 (0.179)	0.265 (0.169)
Normal gait speed	2.58 (0.960)*	0.581 (0.462)	0.762 (0.361)*	–0.483 (0.298)	1.00 (0.480)*	–0.536 (0.419)
Rapid gait speed	–0.356 (0.636)	0.232 (0.226)	0.271 (0.296)	0.766 (0.519)	–0.733 (0.304)*	0.357 (0.221)
Number of comorbidities	–0.133 (0.181)	0.005 (0.048)	0.075 (0.074)	–0.032 (0.073)	–0.121 (0.088)	–0.061 (0.073)
Medication use	0.308 (0.649)	–0.029 (0.169)	0.082 (0.179)	0.119 (0.171)	0.181 (0.221)	–0.045 (0.311)
Tobacco use: not currently using	0.113 (0.435)	0.063 (0.135)	0.199 (0.307)	–0.242 (0.247)	–0.030 (0.193)	0.123 (0.175)
not using daily	–0.755 (0.893)	0.089 (0.229)	–0.236 (0.343)	–0.412 (0.205)*	0.014 (0.481)	–0.209 (0.321)
using daily	0.015 (0.450)	–0.257 (0.187)	0.030 (0.188)	0.656 (0.210)**	0.291 (0.213)	–0.705 (0.261)**
Drinking frequency: do not drink	–0.072 (0.348)	0.152 (0.132)	–0.081 (0.166)	0.076 (0.140)	–0.163 (0.164)	–0.056 (0.148)
less than once per month	–0.051 (0.315)	–0.181 (0.166)	–0.177 (0.174)	0.419 (0.180)*	–0.025 (0.179)	–0.087 (0.165)
1-3 days per month	0.235 (0.604)	0.361 (0.243)	–0.165 (0.290)	0.010 (0.362)	0.292 (0.327)	–0.263 (0.330)
1-4 days per week	0.325 (0.874)	0.484 (0.281)	0.845 (0.328)*	0.272 (0.239)	–0.949 (0.303)**	–0.327 (0.472)
5+ days per week	0.295 (0.889)	–0.163 (0.237)	0.368 (0.218)	–0.502 (0.353)	–0.180 (0.454)	0.771 (0.367)*
Baseline composite cognition	–0.527 (0.082)***	–0.100 (0.021)***	–0.001 (0.029)	–0.032 (0.024)	–0.250 (0.074)**	–0.144 (0.021)***
Follow-up interval	0.005 (0.008)	0.001 (0.003)	–0.002 (0.003)	0.003 (0.003)	0.009 (0.004)*	–0.007 (0.005)
Sleep duration change	–0.291 (0.099)**	–0.011 (0.027)	–0.050 (0.042)	–0.096 (0.039)*	–0.103 (0.054)*	–0.032 (0.042)

^aComparisons are statistically significant (with Bonferroni adjustment) at: * = trend toward significance ($p < 0.06$), ** = $p < 0.008$, *** = $p < 0.001$

^bReference groups used in the creation of dummy codes for each categorical variable:

Data statement

Data from the World Health Organization Study on global AGEing and adult health (SAGE) Waves 1 and 2 can be accessed online at: <http://apps.who.int/healthinfo/systems/surveydata/index.php/catalog/sage/about>

Author contributions

T.E. Gildner conceived of the study, carried out background research, performed the statistical analysis, and drafted the manuscript. A. Salinas-Rodríguez, B. Manrique-Espinoza, and K. Moreno-Tamayo provided feedback in study design, statistical analyses, and manuscript drafting. P. Kowal helped to draft the manuscript. All authors read and approved the final manuscript.

Conflict of interest statement

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- (i) Sex = male
- (ii) Age = 50–59 years
- (iii) Household Setting = urban
- (iv) Education Levels = no formal education
- (v) Physical Activity Levels = low activity levels
- (vi) Medication use = not taking any medication
- (vii) Tobacco Use = never used
- (viii) Drinking Frequency = never used
- (ix) Sleep Category: intermediate sleep duration (6–9 h/night)

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