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Associations between sleep and cognition among middle-aged and older adults in Europe

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Running title: Associations between sleep and cognition

Abstract

Background: The Survey of Health, Ageing and Retirement in Europe (SHARE) is a longitudinal multi-country study of community-dwelling middle-aged and older adults including data on sleep and cognition.

Objective: To examine the cross-sectional association between sleep-related issues and cognition in the SHARE (pooling waves 8 and 9; 2019–2022).

Methods: Subjective sleep data were obtained from SHARE waves 8 and 9. Objective data were available from wave 8. The SHARE Cognitive Instrument (SHARE-Cog) assessed cognition. Participants were divided into three groups: probable dementia, cognitive impairment no dementia (CIND), and normal cognition (NC). Survey-weighted generalised linear regression models were used to compare groups.

Results: 88,889 interviews were included (55% females; mean age 68.88), categorised as: probable dementia 7%, CIND 12%, and NC 81%. While, in the unadjusted analysis, a characteristic 'inverted U' shaped association was observed between sleep duration and cognition, only the association between cognition and longer sleep durations ≥10 hours remained statistically significant after adjusting for potential confounders. Cognitive impairment was significantly associated with subjective reports of recent trouble sleeping, weekly use of medication to aid sleep, and daytime napping, although only the association with daytime napping remained significant after adjusting for potential confounders.

Conclusions: This analysis confirmed an association between sleep duration ≥10 hours and lower cognitive scores among community-dwelling middle-aged and older Europeans. Cognitive impairment was also significantly associated with daytime napping. Further research is required to understand the clinical significance of these findings and establish the underlying causal relationships.

Keywords: Alzheimer's disease; Dementia; Cognitive Impairment; Sleep.

Introduction

Sleep is essential for health and well-being and plays a key role in many physiological functions.[1] However, sleep patterns change as people get older.[2] Physiological changes associated with ageing can affect the quality, quantity and structure of sleep, resulting in a higher prevalence of sleep disturbances, which are estimated to affect between 30-70% of older adults.[3] A recent systematic review and meta-analysis of 252 studies involving 995,544 community-dwelling older adults from 36 countries found the prevalence of poor sleep quality was 40%.[4] Age-related sleep disturbances are not merely epiphenomenal, but rather have significant consequences for health and well-being.[5] For example, sleep disturbances are associated with higher risk of depression, [6] cardiovascular disease,[7, 8] diabetes,[9] cancer,[10] and cognitive impairment.[11]

The association between sleep disturbances and cognitive impairment has generated increasing interest in recent years.[11] With an ageing population, there is a growing number of people living with cognitive impairment and dementia.[12] Sleep disturbances are common among this population affecting up to 70%.[13] Such co-morbid sleep disturbances contribute to negative outcomes including worsening cognition[13] reduced quality of life[14], increased caregiver burden[15,16] and earlier institutionalisation.[17,18] Furthermore, with emerging evidence that the causal relationship between sleep disturbances and cognitive impairment may be bi-directional, [19,20] there is increasing interest in sleep disturbances as a potential modifiable risk factor for cognitive disorders including Alzheimer's disease (AD).[21]

While the association between sleep disturbances and cognitive impairment is well established, it remains imprecisely defined. Several observational studies have found that sleeping too little, [22-24] too much, [25, 26] or both[27] is associated with cognitive impairment, whilst others have failed to find any such association.[28] A 2016 meta-analysis of 18 cross-sectional and prospective cohort studies among older adults found that short and long sleep duration increased the odds of poor cognitive function by 1.40 and 1.58 respectively. [29] However, many studies have been limited by small sample sizes[22, 26, 30-33] that often include homogeneous populations restricted by biological sex[24, 30, 34] or geographical location. [23, 25, 28, 31, 32, 35-40] As sleep disturbances can vary according to culture[41] and ethnicity, [42] there is a need to further analyse the putative association between sleep disturbances and cognitive impairment in a larger, more diverse sample.

The Survey of Health, Ageing and Retirement in Europe (SHARE) is a supranational longitudinal study of community-dwelling middle-aged and older adults[43] which currently includes 28 countries in the European region, encompassing all continental members of the European Union. We undertook a secondary analysis of cross-sectional data from waves 8 [44,45] and 9 [46,47] of SHARE, conducted between 2019 and 2022, for evidence of associations between sleep and cognitive impairment in a European population.

Methods

Study Population

The SHARE involves standardised questionnaires that are administered during computer-assisted interviews conducted by trained personnel with participants aged 50 years and over and their partners. Further details regarding the methodology employed by the SHARE have been published elsewhere. [43] We analysed data from waves 8 and 9 of SHARE, which included a representative sample drawn from 28 countries within the World Health Organisation (WHO) European Region. Participants were excluded from the analysis if they were missing data for the variables of interest. In addition, participants from the SHARE Accelerometer Study (https://share-eric.eu/data/data-set-details/accelerometer-study) were included as a supportive sensitivity analysis as they had objective data on sleep duration. This involved a subsample of participants from 10 countries in wave 8: Belgium, Czech Republic, Denmark, France, Germany, Italy, Poland, Slovenia, Spain, and Sweden. Participants were instructed to wear a small (24.2 x 35.4 x 8.9 mm) and lightweight (11g) tri-axial accelerometer (Axivity AX3, Axivity Limited, Newcastle Upon Tyne, United Kingdom) on their upper thigh for eight consecutive days, throughout both day and night. The Axivity AX3 accelerometer has previously been validated in older adults. [48] An accelerometer is a sensor that records acceleration, that is the rate of change of velocity. A tri-axial accelerometer measures acceleration along all axes of three-dimensional space. The accelerometers were set up to a sampling frequency of 50Hz. The Axivity AX3 stores the raw sensor data in a binary packed format called the "Continuous Wave Accelerometer (CWA)" format. Raw accelerometer data were processed at SHARE Central using ActiPASS Version 1.61beta, an open-source software based on the Acti4 algorithm for posture and activity recognition in data obtained from thigh-worn accelerometers. [49] The data are processed with GGIR, [50] an open-source package for the statistical computing software R. [51] The algorithms from AcitPASS were then used to measure different activities including sleep. Further information can be found in the SHARE release guide 9.0.0. [52]

Sleep Parameters

The following sleep parameters were analysed: night-time sleep duration, subjective sleep quality, use of medication to aid sleep, and the occurrence of daytime napping. The duration of night-time sleep was calculated from responses to the following question:

- "How much time did you spend yesterday on sleeping at night time?"
  Answers in hours and minutes.

An objective measure of night-time sleep duration was available for a sub-sample of participants in the SHARE Accelerometer Study. Subjective sleep quality was assessed using the following question, with a binary choice of possible answers:

- "Have you had trouble sleeping recently?"

- *Trouble with sleep or recent change in pattern*
- *No trouble sleeping*

Information regarding the occurrence of daytime napping was obtained from responses to the following question:

- "How much time did you spend yesterday on napping and resting during the daytime? Do not include sleeping at night time." *Answers in hours and minutes.*

Regarding the use of medication to aid sleep, participants were asked if they currently take drugs for sleep problems at least once per week. Answers were dichotomous (yes/no). For all sleep parameters, responses of "don't know" and "refused" were coded as missing.

Cognition

Cognitive function was assessed using the SHARE Cognitive Instrument (SHARE-Cog), which combines three subtests from SHARE, namely 10-word immediate and delayed recall together with verbal fluency (1 minute animal naming), resulting in a total score ranging from 0–45. [53] The SHARE-Cog has demonstrated superior diagnostic accuracy for dementia compared to other short cognitive screening instruments available in SHARE (including the Mini-Cog, 10-point cognitive screener, and Six-Item Screener). [54]

Participants were divided into probable dementia, cognitive impairment no dementia (CIND) (similar to mild cognitive impairment but based on the presence of objective cognitive deficits in the absence of dementia), and normal cognition (NC) using an adaptation of the 27-point Langa-Weir approach which has become a primary method of assessing cognition in the Health and Retirement Study (HRS) in the United States of America. [55-57] More recently, it has also been applied in the English Longitudinal Study of Aging (ELSA) [58, 59] and the SHARE. [60] The Langa-Weir assessment combines 10-word immediate and delayed recall, serial 7s and a counting backwards task resulting in a total score ranging from 0–27 points. [55] Defined cut-offs are applied to divide participants into dementia (scores 0–6), CIND (scores 7–11) and NC (scores 12–27). [55, 56] These cut-offs were determined using equipercentile equating against a robust subset of "gold standard" clinical diagnoses available in the Aging, Demographics, and Memory Study (ADAMS) sub-study. [55, 56] While the Langa-Weir is not available for participants under the age of 60 years in the SHARE dataset, the SHARE-Cog is available for all ages. Comparable cut-offs were calculated for the SHARE-Cog using the equipercentile equating method with log linear smoothing. SHARE-Cog scores of 0–9 for probable dementia, 10–15 for CIND and 16–45 for NC were comparable to the 0–6, 7–11 and 12–27 ranges used in the Langa-Weir (conversion table presented in the supplementary materials).

Covariates

Data were available for the following covariates that might confound any putative associations between sleep and cognitive impairment: age, education, employment status, frailty status, frequency of vigorous physical activity, smoker status, alcohol consumption, depression, loneliness, and co-morbidities. Participants' age, employment and smoking

status were self-reported. The International Standard Classification of Education 1997 was used to describe participants' level of education, [56] divided into "low" (0–2, none to lower secondary), "medium" (3–4 upper secondary and post-secondary non-tertiary) and "high" (5–6, tertiary) education. [55] Frailty status was defined in accordance with the Fried phenotype. [61] Regarding the frequency of vigorous physical activity, participants were dichotomised into those that engaged in vigorous physical activity at least once per week and those who did not. Depression was assessed by means of the EURO-D, a twelve-item scale with scores of four or higher indicating depression. [62, 63] Loneliness was assessed by the three-item Revised UCLA Loneliness Scale, [64] with scores ranging from 3-9 and higher scores indicating greater loneliness. The burden of co-morbidities was calculated as the mean number of chronic diseases.

Statistical Analysis

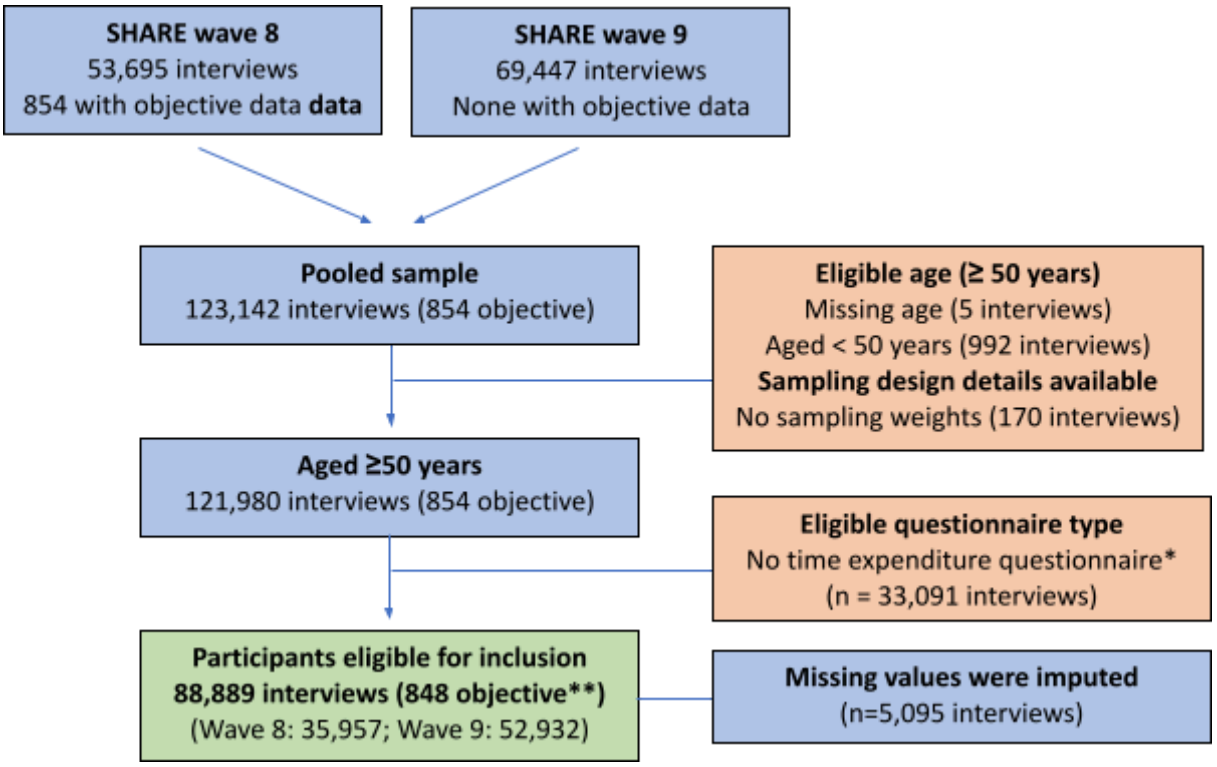
All analysis was carried out in R (version 4.4.2). [51] Missing values were imputed using the k-Nearest Neighbour (kNN) approach in the R package "VIM" (version 6.2.2). [65, 66] This method filled in missing values by using a distancing formula to locate the 5 most similar data points (neighbours), and then selecting the median of these values for continuous variables or the group with the most participants for categorical variables. [66] This method was chosen since it would preserve any non-linear associations while still accounting for complex relationships between multiple variables. Sensitivity analyses comparing results obtained using kNN imputation, complete-case analysis (CCA) and listwise deletion (LD), as well as analyses stratified by wave and unweighted analyses, were performed to assess for bias or distorted variance estimates.

Statistical estimates and comparisons were carried out using complex survey analysis methods in the R package "survey" (version 4.5). [67] These methods account for non-random elements of the sample design and unit non-response. [68] The sample design was specified in accordance with previous guidelines, including an adjustment for the primary sampling unit, individual cross-sectional sampling weights and stratification by the wave and first stratum. [69, 70] This approach was chosen as it maximises the available data and should generate more robust variance estimates (i.e. better *p*-values and confidence intervals). All interviews from both waves were included as this approach adjusts for non-independence between any participants in the same sampling cluster, including whether interviews are from the same or different participants through the sampling unit adjustment (i.e. interviews with the same sampling unit are treated as paired observations while interviews with different sampling units are treated as independent observations).

The statistical significance of differences in independent variables between groups were assessed using generalised linear modelling. The cognitive status (NC, CIND, probable dementia) was ordinal and a Wald-based test was carried out to get a *p*-value. Predictive marginal means were also obtained from the generalised linear models to compare SHARE-Cog scores across different durations of sleep with and without adjustment for confounding. B-Splines were included in the model to allow for non-linear relationships between the continuous variables of sleep duration and SHARE-Cog scores with a knot being placed at each hour of sleep between 4 and 10 hours. A statistical significance level of 95% was applied for all confidence intervals (i.e. ± 1.96 x standard error).

Results

Waves 8 and 9 of SHARE were pooled resulting in the inclusion of 88,889 interviews from 28 countries. Objective data were also included for sleep duration from a subsample of 848 participants from wave 8. The reasons for excluding participants are summarised in Figure 1.



**The "time expenditure" questionnaire, which includes details on self-reported sleep durations, was not administered to all participants. Participants without this questionnaire include nursing home residents, some countries in wave 8 and the majority of newly joining participants. Regarding the objective sleep data, the 6 excluded participants were nursing home residents. ** Sensitivity analysis with participants from the SHARE Accelerometer Study with limited objective data*

Figure 1. Flow diagram of the participants included from the SHARE and reasons for exclusion.

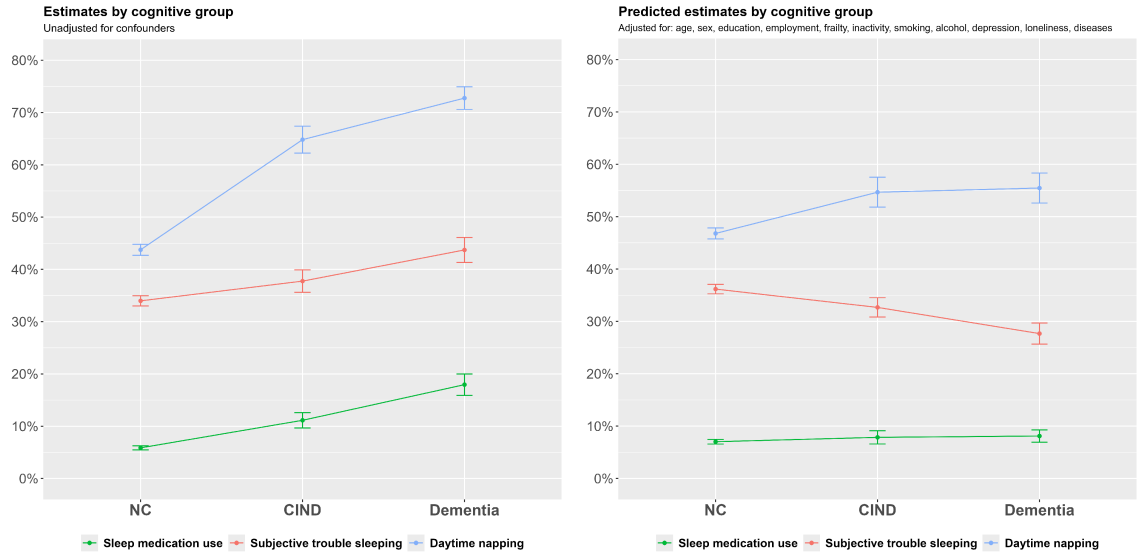
Of the 88,889 interviews included in the study, 81% had normal cognition, 12% had CIND, and 7% had probable dementia. Table 1 sets out data pertaining to cognitive function, sleep-related variables, and potential confounding factors, for each of these groups. Overall, there was a high prevalence of subjective sleep disturbance, with 35% of all participants reporting recent trouble sleeping. Moreover, there was a significant trend towards increased reports of recent trouble sleeping, increased use of medication to aid sleep, and increased daytime napping among participants in more advanced stages of cognitive impairment, which is further illustrated in Figure 2. However, participants with more advanced cognitive impairment were also more likely to be older, lonely, depressed, more frail, less physically active, have lower levels of education, and higher levels of co-morbidity. Pairwise analyses indicated that significant sleep-related differences were largely driven by contrasts involving the Probable Dementia group, with minimal separation between CIND and NC for most sleep duration measures (see Supplementary Materials)

Variable	Total (N=88,889) 100%	NC (n=72,142) 81%	CIND (n=10,722) 12%	Probable Dementia (n=6,025) 7%	p-value
Cognitive function					
SHARE-Cog score	22.92	25.87	12.9	5.25	< 0.001
Sleep-related variables					
Night-time sleep (self-report)	7.04	7.02	7.04	7.28	< 0.001
Night-time sleep (objective)*	6.89	6.87	6.97	7.4	0.514
Trouble sleeping	35%	34%	38%	44%	< 0.001
Sleep medication use	7%	6%	11%	18%	< 0.001
Daytime napping	48%	44%	65%	73%	< 0.001
Potential confounders					
Age (mean)	68.88	67.1	74.35	80.56	< 0.001
Female (%)	55%	55%	55%	61%	< 0.001
Education: low	39%	32%	67%	76%	< 0.001
Education: medium	39%	42%	25%	18%	
Education: high	23%	26%	8%	7%	
Employed	27%	31%	11%	2%	< 0.001
Physical frailty: robust	43%	49%	23%	10%	< 0.001
Physical frailty: pre-frail	44%	43%	50%	43%	
Physical frailty: frail	13%	8%	26%	48%	
Rigorous activity < weekly	56%	51%	73%	90%	< 0.001
Smoker	15%	16%	12%	7%	< 0.001
Heavy alcohol consumption	19%	20%	14%	8%	< 0.001
Depression (EURO-D ≥ 4)	29%	25%	41%	65%	< 0.001
Loneliness (R-UCLA)	4.05	3.89	4.55	5.08	< 0.001
Disease count	1.61	1.5	1.94	2.35	< 0.001

EURO-D: a scale of depressive symptoms
R-UCLA: Revised-University of California at Los Angeles Loneliness Scale
*From accelerometer sub-study, only available for 848 participants

Table 1. Cognitive function, sleep-related variables, and potential confounding factors by cognitive performance categories.

Figure 2 illustrates that, after adjusting for the potential confounding factors mentioned in Table 1, the purported differences in recent trouble sleeping reversed direction ($p<0.001$), while the differences in sleep medication use were no longer significant ($p=0.181$). However, daytime napping remained statistically significantly associated with worse cognition ($p<0.001$).



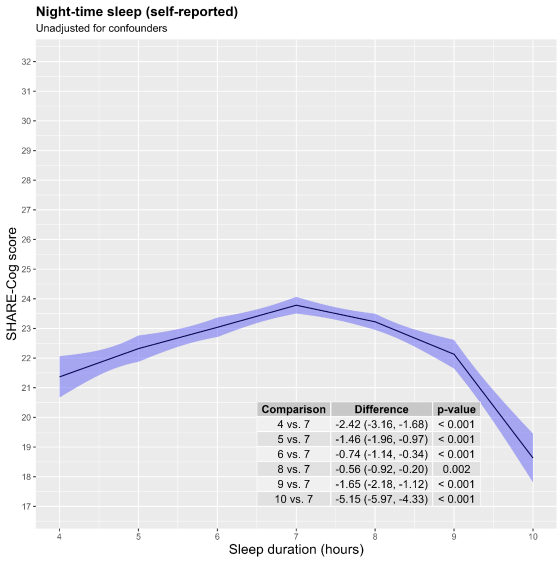
2(a) 2(b)

Figure 2. Estimated prevalence (%) for sleep medication use, subjective trouble sleeping, and daytime napping by cognitive group, namely: Normal Cognition (NC), Cognitive Impairment No Dementia (CIND), and probable dementia. Figure 2(a) presents the unadjusted analysis while Figure 2(b) presents the analysis adjusted for potential confounders.

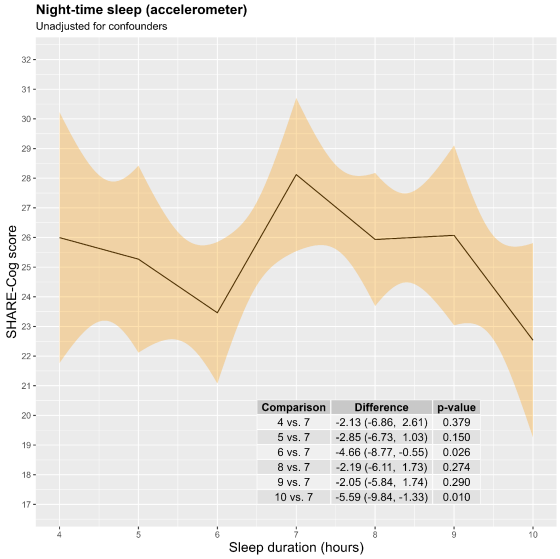
Those who slept for seven hours had the highest SHARE-Cog scores in the unadjusted analyses of both the subjective (self-reported) and objective (Accelerometer) data (Figures 3a. and 3b.). Overall, the unadjusted analyses were notable for an inverted U-shaped association between SHARE-Cog scores and self-reported night-time sleep duration (Figure 3a). Compared to those who slept for seven hours, those who slept six hours or less and those who slept nine hours or more had significantly lower cognitive scores. However, when the results were adjusted for potential confounders, only those who slept for ten hours or more continued to have significantly lower cognitive scores (Figure 3b).

In the small subsample from the SHARE Accelerometer Study (n=848), the mean objectively measured sleep duration of 6.89 hours, standard error (SE) 0.18, was nearly identical to the mean self-reported sleep duration of 6.88 hours, SE 0.15, although overall agreement between measures was modest (r=0.28). However, in the Accelerometer Study subsample, the association between sleeping ten hours or more and lower cognitive scores was no longer significant after adjusting for confounders (see Figures 3c-d). It is also notable that, in the Accelerometer Study, sub-sample, those who slept for extremely short durations appeared to have relatively higher cognitive scores (see Figures 3c-d) compared to the self-reported sample (Figures 3a-b). However, this is likely due to statistical uncertainty created by the small sample size in the Accelerometer sub-study, with only 156 participants recording <6 hours of sleep and only 58 of these recording <5 hours of sleep. In models including both subjective and objective sleep duration simultaneously, subjective sleep was no longer independently associated with cognitive performance (overall p=0.066), while objective sleep explained a greater proportion of variance (p=0.006). Individual category estimates were imprecise, reflecting limited statistical power in the small Accelerometer Study subsample (see Supplementary Materials).

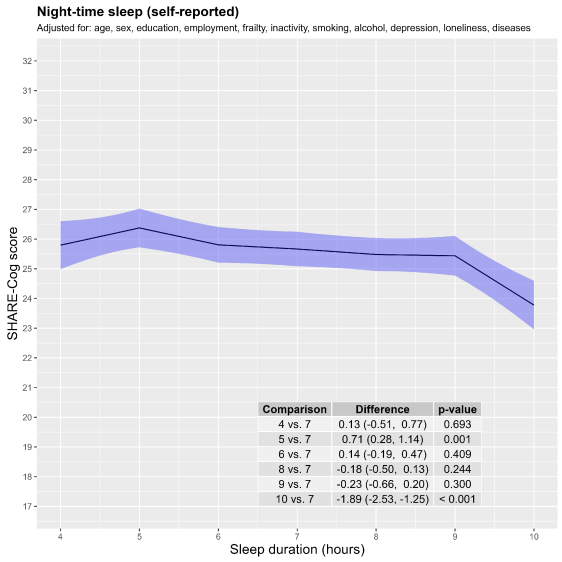
(a)


Night-time sleep (self-reported)
Unadjusted for confounders
SHARE-Cog score
Sleep duration (hours)
Comparison | Difference | p-value
4 vs. 7 | -2.42 (-3.16, -1.68) | < 0.001
5 vs. 7 | -1.46 (-1.96, -0.97) | < 0.001
6 vs. 7 | -0.74 (-1.14, -0.34) | < 0.001
8 vs. 7 | -0.56 (-0.92, -0.20) | 0.002
9 vs. 7 | -1.65 (-2.18, -1.12) | < 0.001
10 vs. 7 | -5.15 (-5.97, -4.33) | < 0.001

(c)


Night-time sleep (accelerometer)
Unadjusted for confounders
SHARE-Cog score
Sleep duration (hours)
Comparison | Difference | p-value
4 vs. 7 | -2.13 (-6.86, 2.61) | 0.379
5 vs. 7 | -2.85 (-6.73, 1.03) | 0.150
6 vs. 7 | -4.66 (-8.77, -0.55) | 0.026
8 vs. 7 | -2.19 (-6.11, 1.73) | 0.274
9 vs. 7 | -2.05 (-5.84, 1.74) | 0.290
10 vs. 7 | -5.59 (-9.84, -1.33) | 0.010

(b)



(d)

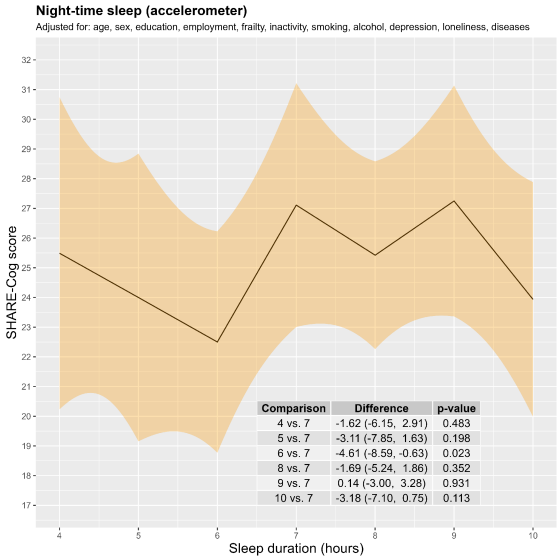


Figure 3. Cross-sectional association between mean SHARE-Cog score and sleep duration for (a) self-reported night-time sleep (unadjusted), (b) self-reported night-time sleep (adjusted), (c) accelerometer-measured night-time sleep (unadjusted), and (d) accelerometer-measured night-time sleep (adjusted). Note: the shaded areas illustrate 95% confidence intervals.

Across all approaches to address missingness, comparing kNN, CCA and LD amongst others, the pattern of associations, particularly the attenuation of short-sleep effects after adjustment and the persistence of the ≥10-hour association was highly consistent, with overlapping confidence intervals and similar effect sizes. These findings suggest that the primary results are robust to different missing-data handling strategies (See Supplementary Materials).

Discussion

In this study, the primary unadjusted analysis of a large population-based cohort of middle-aged and older adults in Europe demonstrated that those who slept for seven hours per night had the highest cognitive scores. Several guidelines, including the Joint Consensus Statement of the American Academy of Sleep Medicine and the Sleep Research Society, [71] recommend that adults should sleep seven or more hours per night on a regular basis to promote optimal health. These guidelines generally recommend 7–9 hours of sleep for older adults, while recognising substantial inter-individual variability and that some adults may appropriately sleep outside this range. Furthermore, in line with a meta-analysis of previous observational studies, [31] our study showed an inverted U-shaped association between sleep duration and cognition. Based on the unadjusted self-reported data, those who slept for six hours or less, and those who slept for nine hours or more, were both significantly more likely to have lower cognitive scores than those who slept for the optimal seven hours. While the reasons for this inverted U-shaped association between sleep duration and cognition remain to be fully elucidated, several plausible mechanisms have been proposed in the scientific literature. For example, sleep disturbances such as altered sleep duration may plausibly contribute to cognitive decline. It is recognised, however, that reverse causality is also possible, particularly in older adults, whereby early neurodegenerative changes, comorbidity burden, or functional decline lead to increased sleep need, fragmented nocturnal sleep, or greater propensity for daytime napping. Furthermore, sleep characteristics and cognitive performance may be jointly influenced by shared underlying factors, including age-related co-morbidities. Given the cross-sectional design of the present study, these competing explanations cannot be disentangled, and the findings should therefore be interpreted as associations rather than evidence of directional or causal effects. The evidence from the scientific literature supporting each of these possible mechanisms will be further discussed in the remaining paragraphs.

Sleep is important for memory consolidation. [72-74] Shorter sleep durations have been shown to negatively affect cognitive functioning[75] and may contribute to cognitive decline due to increased accumulation of the toxic proteins that are characteristic of AD. [76] Cerebrospinal fluid levels of beta-amyloid fluctuate diurnally, rising during wakefulness before declining during sleep. [77] The glymphatic system that clears toxic metabolic waste products from the brain is more efficient during sleep,[78] though this has recently been called into question.[79] Sleep deprivation has nevertheless been shown to increase accumulation and cortical deposition of both beta-amyloid[80] and tau. [81] Another possible explanation for the association between shorter sleep durations and cognitive impairment is that amyloid is released during neuronal synaptic activity,[82] which is reduced during sleep.[83] Amyloid accumulation results from an imbalance between production and clearance, and plaque formation is dependent on regional amyloid concentrations.[84] Brain regions with greater neuronal activity show greater amyloid concentrations in the interstitial fluid, and subsequently more plaque formation.[85] Sleep disturbance, and consequent shorter sleep durations, could cause chronically increased levels of neuronal activity, thereby resulting in greater accumulation of amyloid.[86]

In our study, whilst the association between shorter sleep durations and cognitive impairment was attenuated in the adjusted analysis, those with longer sleep durations of ten hours or more continued to have significantly lower cognitive scores. No significant

results were found on the adjusted objective data, however, likely reflecting the small sample size available in the sub-study, limiting the statistical power. Longer sleep is associated with numerous health problems, including increased mortality.[87] A previous meta-analysis of observational studies demonstrated that longer sleep is associated with cognitive decline.[88] It has been suggested that the association between longer sleep durations and cognitive impairment may be mediated by mood-related illnesses[89] and increasing frailty, the latter of which has been linked to both cognitive impairment[90] and longer sleep duration.[91] However, in our study, the association between cognitive impairment and self-reported longer sleep durations remained significant after adjusting for both mood and frailty. Other putative mechanisms by which prolonged sleep duration may contribute to cognitive decline include increased inflammatory activity in the brain. Interleukin 6 (IL-6) and c-reactive protein (CRP) pathways may be increased by excessive sleep duration, with one study demonstrating a linear association between IL-6, CRP and sleep duration. [92] It has also been hypothesised that increased sleep duration may reflect compensatory sleep in response to decreased efficiency of the deep slow wave sleep that is important for memory consolidation.[93] Longer sleep durations may compensate for increased sleep fragmentation,[94] which is associated with cognitive impairment.[95] Obstructive sleep apnoea is another important cause of sleep fragmentation and increased daytime sleepiness that is known to be associated with cognitive impairment.[96, 97]

Our primary unadjusted analysis also confirmed a statistically significant association between cognitive impairment and subjective sleep quality, whereby those with more advanced cognitive impairment were more likely to report recent trouble sleeping. Despite this purported association being reversed in the adjusted analysis, it remains important because, regardless of the existence of any causal relationship between sleep and cognitive decline, worsening sleep quality can, in and of itself, negatively impact day-to-day functioning and quality of life. [98] Furthermore, particularly in more advanced cases of dementia, worsening sleep quality can lead to increased caregiver strain[99] and earlier institutionalisation. [18] Perhaps as a consequence of the aforementioned association between cognitive impairment and subjective sleep quality, our primary unadjusted analysis also found a significant trend towards increased use of sleep medication among those in the more advanced stages of cognitive impairment. In this regard, our study replicates findings from previous population-based observational studies. [100, 101] This is problematic because several prescribing guidelines for older adults, including the Beers Criteria published by the American Geriatric Society, [102] counsel against use of sleep medications due to their manifold adverse effects. Indeed, there is evidence that sleep medication use may exacerbate cognitive decline. [19] Further research is therefore required to ascertain the optimal treatments for sleep disturbances in people with cognitive impairment. [103-105] Our analysis also confirmed an association between cognitive impairment and daytime napping, and this association remained significant after adjusting for potential confounders. Those with more advanced cognitive impairment were significantly more likely to take a nap during the day. Whilst other recent analyses have also shown an association between excessive daytime napping and cognitive impairment, [106] and between napping and amyloid burden,[107] it has been suggested that moderate napping may be superior to no napping. [108] Evidence is limited, however, and napping is an aspect of sleep behaviour that warrants further investigation in the struggle to combat the increasing burden of dementia.

Strengths & Limitations

The main strength of this study is that it involved a large culturally diverse population-based sample drawn from throughout Europe and therefore has good generalisability and statistical power. It is well recognised that there are cultural differences regarding sleep throughout Europe, which further affects the perception and management of sleep and sleep disorders.[109] To our knowledge, no previous study has examined the association between sleep and cognition in such a large dataset of Europeans. The harmonised analysis across 28 countries using a single, standardised cognitive instrument enhances comparability and internal consistency across such diverse populations. Further, the adjustment for several potential confounding factors and the inclusion of multiple sensitivity analyses supports the robustness of the observed associations, while acknowledging their cross-sectional nature. Nevertheless, residual confounding related to unmeasured cultural, behavioural, and environmental differences between countries cannot be excluded.

There are several limitations. There were some missing data, which could have introduced non-response bias. While no single approach is regarded as a 'gold standard' to address missingness, we confirmed through sensitivity analyses that the findings were robust to different strategies for handling missing-data. This study relied primarily on the subjective self-report of sleep quality and duration, which is prone to recall bias, particularly among cognitively impaired participants.[110] Here, the Accelerometer Study sub-sample comprised only 848 participants from 10 countries, underpowering and limiting the ability to interpret this analysis. Accordingly, these findings should be interpreted as supportive sensitivity analyses rather than definitive objective validation of the primary self-reported sleep findings. For example, the apparent differences at the extreme lower end of objectively measured sleep duration should be interpreted cautiously and not considered evidence of a protective effect of very short sleep duration. The modest correlation observed between subjective and objective sleep duration underscores that these measures capture overlapping but distinct constructs of sleep behaviour and perception. While self-reported and accelerometer-measured sleep showed broadly similar patterns of association with cognitive performance, subjective sleep did not independently predict cognition when adjusted for objective sleep. While a limitation, the self-perception of sleep is important in and of itself, and is often the only measure of sleep routinely available in clinical practice.[111] Healthy sleep is multi-dimensional,[112] and the subjective perception of sleep may convey unique information about a person's well-being relative to objective measures. [113] Subjective reports of sleep quality and duration have previously been associated with cognitive impairment.[20] At a pathological level, subjective reports of sleep quality[86, 113] and duration[114] have been associated with increased amyloid burden, which is one of the characteristic features of AD.[115, 116] Studies that have demonstrated discrepancy between subjective and objective measures of sleep in older people have suggested that both are important as they measure different aspects of sleep.[117] Furthermore, studies have shown that this discrepancy between subjective and objective measures of sleep cannot always be explained by cognitive status,[117] even among those with cognitive impairment,[118] and may be affected by other factors such as socio-economic status.[119] Subjective measures of sleep are generally considered valid in those with relatively mild cognitive decline.[120] Our study sample did not include any nursing home residents, reducing the proportion with more advanced dementia.

Furthermore, most participants were assisted by their caregivers to complete the study questionnaire, which likely helped improve the accuracy of the assessment, although it is acknowledged that caregiver assistance in questionnaire completion may also introduce systematic bias.[121] It is further acknowledged that the study's use of single questions to assess sleep parameters, such as sleep quality and duration of daytime or night-time sleep, is also a limitation. Use of validated questionnaires, such as the Pittsburgh Sleep Quality Index [122] to assess subjective sleep quality, may have improved the accuracy and reliability of results. Similarly, our study relied on the self-report of sleep medication use, which is also prone to bias. Again, however, the assistance of caregivers to complete the study questionnaire could be expected to improve the accuracy of responses.[121] Furthermore, the self-report of sleep medication use has been used in several other population-based studies that have produced similar results to ours. [100, 101] Whilst our study found an association between the use of sleep medications and cognition, it did not distinguish between different sleep medications. Our study cannot, therefore, help determine the risks associated with particular sleep medications. Rather, the study demonstrates an association between the use of sleep medications, as a general marker of sleep disturbance, and cognitive impairment. It is also acknowledged that many other medications, that may not be considered sleep medications per se, can influence sleep. In particular, medications with sedative, stimulating, anticholinergic, or corticosteroid effects may alter sleep duration, sleep quality, or daytime sleepiness. It is well recognised, for example, that steroids can affect sleep. [123] It is a limitation of our study that account was not taken of the possible effects on sleep of such other medications. Similarly, whilst our study showed an association between napping and cognitive impairment, it could not evaluate the nature and timing of the naps as these data were not available. In particular, the question used did not allow differentiation between intentional naps, unintentional dozing, excessive daytime sleepiness, or compensatory behaviours in dementia. The cognitive categories (NC, CIND, probable dementia) were not equivalent to a clinical diagnosis, being derived from performance-based assessments, limiting external generalisability and potentially resulting in some misclassification, for example, among individuals with depression. Furthermore, although several observed associations reached statistical significance, the clinical significance of relatively modest differences in SHARE-Cog scores remains uncertain because minimal clinically important differences for the instrument have not yet been established. Additional validation of the SHARE-Cog, including its minimal clinically significant difference and its equated score is now required, although it has shown good diagnostic accuracy for dementia compared with other available cognitive screening instruments. [54]

As this is a cross-sectional observational study, it is not possible to demonstrate any causal relationships. There is, indeed, a possibility of reverse causality whereby sleep, as the putative risk factor, is affected by the underlying disease process in dementia.[124] Indeed, many of the brain regions critical to the regulation of sleep-wake behaviour are highly vulnerable to and among the earliest regions affected by AD pathology. [113] For example, the supra-chiasmatic nucleus, the 'master brain clock' regulating circadian rhythm, is particularly vulnerable to neurodegenerative tauopathies like AD.[125] While the effect of sleep disturbance on the accumulation of amyloid has already been discussed, it has also been demonstrated that amyloid aggregation conversely disrupts the sleep-wake cycle.[126] Evidence from longitudinal studies that have sought to elucidate the direction of causation are inconsistent. Some have demonstrated an inverted U-shaped association between sleep

duration and cognitive decline,[127, 128] while others have found that only short[124] or long[129] sleep durations were associated with cognitive decline. Other longitudinal studies have not shown any association between sleep duration and risk of cognitive decline.[130] In a large Swedish study of 28,775 older adults, the association between long sleep duration and cognitive decline was completely attenuated after cases occurring in the first five years of follow up were excluded from the analysis, emphasising the possible role of reverse causation bias.[131] In another longitudinal study involving MRI brain imaging of 3,893 healthy adults, the rate of brain atrophy was not associated with either long or short sleep duration despite there being an inverted U-shaped association between sleep duration and cortical thickness in the cross-sectional analysis.[132] While this study adjusted for several potential confounding factors, residual confounding remains likely. Several other potentially important factors including chronotype, circadian disruption, objective sleep apnoea measures, and institutional support, were unavailable in the SHARE. Finally, data collection for Waves 8 and 9 spanned the COVID-19 pandemic, a period associated with population-level changes in sleep behaviours and psychosocial stressors.[133] However, wave-specific analyses restricted to the same participants demonstrated highly consistent associations between sleep duration and cognitive performance (see Supplementary Materials), suggesting that pandemic timing and differences in survey administration did not materially confound the observed cross-sectional relationships.

Conclusion

This analysis of a large diverse population-based study sample confirmed that sleep disturbances are common in middle-aged and older European adults. Furthermore, the study confirmed that subjective reports of longer sleep durations of ≥10 hours and daytime napping are both associated with poorer cognitive performance. Whilst further research is required to define the underlying causal relationships, this paper adds to the evidence that sleep disturbances may be a target for intervention in people with cognitive impairment due to their high prevalence and their adverse impact on day-to-day functioning and quality of life. [98]

Author contributions

Patrick Crowley (Conceptualization, Methodology, Writing - Original Draft, Review & Editing, Visualization, Project administration); Mark R. O'Donovan (Methodology, Validation, Software, Validation, Formal analysis, Investigation, Review & Editing, Visualization); Evelyn Flanagan (Supervision, Project administration); Rónán O'Caoimh (Conceptualization, Review & Editing, Supervision, Project administration)

*Note that all other contributions (Resources; Data Curation; Funding acquisition; Other: N/A) are applicable to the SHARE as acknowledged below:

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Ethical Considerations

The SHARE study is subject to continuous ethics review. During Waves 1 to 4, SHARE was reviewed and approved by the Ethics Committee of the University of Mannheim. Wave 4 and the continuation of the project were reviewed and approved by the Ethics Council of the Max Planck Society. In addition, the country implementations of SHARE were reviewed and approved by the respective ethics committees or institutional review boards whenever this was required. The numerous reviews covered all aspects of the SHARE study, including sub-projects and confirmed the project to be compliant with the relevant legal norms and that the project and its procedures agree with international ethical standards.

Consent to Participate

Not applicable for this secondary analysis. All participants in the SHARE study provided written informed consent.

Consent for Publication

Not applicable for this secondary analysis. All participants in the SHARE study provided written consent for publication.

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Declaration of Conflicting Interests

Dr Rónán O’Caoimh is an Editorial Board Member of this journal, but was not involved in the peer-review process nor had access to any information regarding its peer-review. Dr O’Caoimh and Mr O’Donovan are co-copyright holders of the SHARE Cognitive Instrument. The authors declare no other conflicts of interest.

Data Availability

The data used in the study can accessed via the SHARE consortium (https://share-eric.eu/data/data-access). R codes used to perform the analysis are available on request.

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