RESEARCH ARTICLE

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Daytime somnolence as an early sign of cognitive decline in a community-based study of older people

Angeliki Tsapanou[1], Yian Gu[1], Deirdre O'Shea[1], Teal Eich[1], Ming-Xin Tang[3,7], Nicole Schupf[1,2,3,4], Jennifer Manly[1,3,4], Molly Zimmerman[5], Nikolaos Scarmeas[1,2,3,4,6] and Yaakov Stern[1,2,3,4]

[1]Cognitive Neuroscience Division, Department of Neurology and The Taub Institute for Research on Alzheimer's Disease and the Aging Brain, Columbia University College of Physicians and Surgeons, New York, NY, USA
[2]The Gertrude H. Sergievsky Center, College of Physicians and Surgeons, Columbia University, New York, NY, USA
[3]The Department of Neurology, College of Physicians and Surgeons, Columbia University, New York, NY, USA
[4]The Division of Epidemiology, Joseph P. Mailman School of Public Health, Columbia University, New York, NY, USA
[5]Department of Psychology, Fordham University, Bronx, NY, USA
[6]National and Kapodistrian University of Athens Medical School, Athens, Greece
[7]Department of Biostatistics, Joseph P. Mailman School of Public Health, Columbia University, New York, NY, USA
Correspondence to: A. Tsapanou, MSc, E-mail: at2859@cumc.columbia.edu

Objective: This study aimed to examine the association between self-reported sleep problems and cognitive decline in community-dwelling older people. We hypothesized that daytime somnolence predicts subsequent cognitive decline.

Methods: This is a longitudinal study in a 3.2-year follow-up, with 18-month intervals. The setting is the Washington Heights-Inwood Community Aging Project. There were 1098 participants, who were over 65 years old and recruited from the community.
Sleep problems were estimated using five sleep categories derived from the RAND Medical Outcome Study Sleep Scale: sleep disturbance, snoring, awaken short of breath/with a headache, sleep adequacy, and daytime somnolence. Four distinct cognitive composite scores were calculated: memory, language, speed of processing, and executive functioning. We used generalized estimating equations analyses with cognitive scores as the outcome, and time, sleep categories and their interactions as the main predictors. Models were initially unadjusted and then adjusted for age, gender, education, ethnicity, depression, and apolipoprotein E-ε4 genotype.

Results: Increased daytime somnolence (including feeling drowsy/sleepy, having trouble staying awake, and taking naps during the day) was linked to slower speed of processing both cross-sectionally ($B=-0.143$, $p=0.047$) and longitudinally ($B=-0.003$, $p=0.027$). After excluding the demented participants at baseline, the results remained significant ($B=-0.003$, $p=0.021$).

Conclusions: Our findings suggest that daytime somnolence may be an early sign of cognitive decline in the older population. Copyright © 2015 John Wiley & Sons, Ltd.

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Introduction

Changes in sleep patterns are common among the aging population. Self-reported sleep problems seem to reflect poor overall quality of sleep, which in turn has been associated with changes in cognition (Ohayon, 2004; Ohayon *et al.*, 2004; Basner *et al.*, 2007; Miyata *et al.*, 2013; Ramos *et al.*, 2013). Some cross-sectional studies have reported that sleep problems, especially daytime sleepiness, are related to poorer cognitive function (Dealberto *et al.*, 1996; Ohayon and Vecchierini, 2002; Merlino *et al.*, 2010). For example, in one study (Ohayon and Vecchierini, 2002), excessive daytime sleepiness was associated with impaired memory, orientation, and attention.

Although a number of cross-sectional studies have investigated the relationship between sleep problems

and cognitive functioning in older adults, there is a relative paucity of longitudinal research on this relationship (Cohen-Zion *et al.*, 2001; Jelicic *et al.*, 2002; Yaffe *et al.*, 2011; Jaussent *et al.*, 2012; Keage *et al.*, 2012; Blackwell *et al.*, 2014). From the few longitudinal studies that do exist, findings suggest that self-reported daytime sleepiness, in particular, is a major risk factor for (Jelicic *et al.*, 2002), and a possible early marker of cognitive decline (Jaussent *et al.*, 2012).

Some results of the existing longitudinal literature, however, are contradictory. Tworoger *et al.* (2006) found no longitudinal association between self-reported sleep duration, difficulty sleeping, and snoring with cognitive function in older women. Furthermore, Jelilic's (2002) results about association between sleep problems and cognitive decline did not survive after controlling for depression.

Many of the previous longitudinal studies have some apparent research gaps. For example, it is unclear whether sleep problems are related to specific cognitive abilities, such as memory, executive function, language, and speed of processing, as most of the existing studies used short global measures of cognitive function (Cohen-Zion *et al.*, 2001; Keage *et al.*, 2012; Potvin *et al.*, 2012) or a telephone-based screening battery (Loerbroks *et al.*, 2010; Virta *et al.*, 2013; Devore *et al.*, 2014). One large longitudinal study that did investigate the association of sleep with a specific cognitive domain (i.e., memory) used a single memory test (i.e., Delayed Word Recall Test) in addition to the Mini Mental State Examination (MMSE) (Xu *et al.*, 2014), which may not provide a complete picture of cognitive function. Furthermore, many of the previous longitudinal studies did not include a comprehensive clinical evaluation. This could potentially result in the inclusion of individuals with dementia in the analyses, which could greatly skewed some of the findings (Jaussent *et al.*, 2012). In most of the existing longitudinal studies that have used an extensive neuropsychological assessment, the sample size was relatively small (Cohen-Zion *et al.*, 2001; Nebes *et al.*, 2009; Lim *et al.*, 2013). The longitudinal study with the largest sample ($n = 2822$), examining the association between sleep problems and cognitive decline, used only male participants in the sample and a short cognitive assessment consisting of two tests (Blackwell *et al.*, 2014). Furthermore, another large-sample longitudinal study was also restricted to male participants (Foley *et al.*, 1999), limiting the non-generalizability of the findings.

The present exploratory study was conducted in an attempt to bridge some of the shortcomings. Specifically, we wanted to examine the association between distinct types of sleep problems and specific cognitive abilities over a 3.2-year follow-up period in a large sample of ethnically diverse, community-dwelling older adults.

Based on the existing literature, which suggests that daytime sleepiness is associated with cognitive decline (Ohayon and Vecchierini, 2002; Merlino *et al.*, 2010; Jaussent *et al.*, 2012), while also taking into account the conflict between the results of the previous studies (Jelicic *et al.*, 2002; Tworoger *et al.*, 2006), our primary hypothesis is that daytime somnolence, as a distinct type of sleep problem, would predict decline in specific cognitive abilities in older people.

We also aimed to examine whether other types of sleep problems would be differentially associated with decline in specific cognitive domains in the same sample of participants.

Methods

Study participants

Participants were drawn from the Washington Heights-Inwood Community Aging Project (WHICAP) at the Columbia University Medical Center (Tang *et al.*, 2001; Manly *et al.*, 2005). WHICAP is a community-based research study aimed at identifying risk factors and biomarkers for aging and Alzheimer's disease (AD) in a multi-ethnic cohort that includes Caucasian, African American, and Hispanic participants (Tang *et al.*, 1998). Evaluations were conducted in either English or Spanish, based on the preference of the participant. The age of the participating pool that took part in the project was over 65 years.

Two thousand three hundred fifty-eight WHICAP II cohort participants remained active in 2007 when the sleep questionnaire was first applied in the study population. This visit was set as the baseline visit for the current study and was used for cross-sectional analyses. For the longitudinal analyses, we excluded 1260 people who did not have a follow-up visit, yielding an analytic sample of 1098 participants who had both sleep data and repeated assessments (Table 1). Participants were followed at intervals of approximately 1.5 years, repeating the baseline examination and consensus diagnosis in a 3.2-year total follow-up time.

Sleep measures

There were no inclusion or exclusion criteria based on sleep. Sleep quality was assessed using the Sleep Scale from the RAND Medical Outcome Study. This scale is a self-report 12-item questionnaire (see Appendix) (Spritzer and Hays, 2003). Each of the questions has

Table 1 Demographic characteristics of the participants

Characteristics	Cross-sectional	Longitudinal
Age at visit (years), mean (*SD*)	80.0 (7.0)	75.3 (6.1)
Education (years), mean (*SD*)	9.75 (5.1)	10.00 (5.1)
Gender, *n* (%)		
Female	1636 (69.4)	780 (71.0)
Male	722 (30.6)	318 (29.0)
Ethnicity, *n* (%)		
White	488 (20.7)	244 (22.2)
Black	537 (22.8)	250 (22.8)
Hispanic	1303 (55.3)	592 (53.9)
Other	30 (1.3)	12 (1.1)
APOE-ε4, *n* (%)	553 (25.0)	273 (25.1)
Demented at baseline, *n* (%)	237 (9.9)	73 (6.6)
Total (*n*)	2358	1098

SD, standard deviation.

a possible rating of 0–6, based on the frequency of the sleep problem. According to the questionnaire, these 12 separate questions can be gathered to five sleep categories, which we used for our analyses:

(1) *Sleep disturbance* (included questions: "How long did it usually take for you to fall asleep during the past 4 weeks?", "Feel that your sleep was not quiet (moving restlessly, feeling tense, speaking, etc.) while sleeping?", "Have trouble falling asleep?", "Awaken during your sleep time and have trouble falling asleep again?"), (2) *Snoring* ("Snore during your sleep?"), (3) Awaken *short of breath/with headache* ("Awaken short of breath or with a headache?"), 4. *Sleep adequacy* ("Get enough sleep to feel rested upon waking in the morning?", "Get the amount of sleep you needed?"), and (5) *Daytime somnolence* ("Feel drowsy or sleepy during the day?", "Have trouble staying awake during the day?", "Take naps (5 min or longer) during the day?") (Spritzer and Hays, 2003; Hays *et al.*, 2005).

The score for each category was calculated by summing up the values of the component questions. A higher score indicates greater sleep dysfunction.

Cognitive measures

A structured, in-person interview, consisting of a medical history report and physical examination, was conducted for each participant during each of the visits (Stern *et al.*, 1992). In addition, a full neuropsychological battery examining all major cognitive domains was administered for each participant.

Composite scores for four cognitive abilities were then derived based on a previously reported factor analysis (Siedlecki *et al.*, 2010): memory, executive functioning, language, and speed of processing.

Memory: average composite of total raw scores for immediate recall, delayed recall, and delayed recognition from the Selective Reminding Test (Buschke and Fuld, 1974).
Language: average composite of total correct items on the 15-item Boston Naming Test (Kaplan *et al.*, 1983). Repetition (total number of correct phrases) and comprehension (total number of correct comprehensive questions) were assessed with sub-tests of the Boston Diagnostic Aphasia Examination (Goodglass, 1983).
Executive functioning: average composite of total correct on the Mattis Identities and Oddities, raw score on Wechsler Adult Intelligence Scale-Revised Similarities subtest, and mean number of words generated during three 60-s trials for category and letter fluency (Wechsler, 1981; Mattis, 1988).
Speed of processing: average composite of the time score on the Color Trails Making Test Trials 1 and 2 (Salthouse, 2011).

First, *z*-scores were derived for each test for each participant at each occasion, using the means and standard deviations (*SD*) calculated from baseline scores for non-demented WHICAP subjects controlling for age, ethnicity, and years of education. Next, we computed the average *z*-score metric for each participant within each of the four cognitive domains. For speed of processing, we reversed the scores so that higher score reflected better processing (Zahodne *et al.*, 2014). Finally, these four domain scores were averaged to produce a composite "mean cognition" z-score. A higher z-score indicates better cognitive performance. A more complete description of the neuropsychological test battery and the cognitive scores can be found in previous publications (Stern *et al.*, 1992; Manly *et al.*, 2005; Grunebaum *et al.*, 2008; Salthouse, 2011; Zahodne *et al.*, 2014).

Covariates

Age (years) at baseline visit and education (years) were used as continuous variables. Ethnicity was ascertained based on self-report using the format of the 1990 census (Census of Population and Housing, 1991). Participants were then assigned to one of four groups: Black (non-Hispanic), Hispanic, White (non-Hispanic), and Other. Ethnicity was used as a dummy variable with White (non-Hispanic) as the reference. Apolipoprotein E (*APOE*-ε4) genotype was used dichotomously: absence of ε4 allele versus presence of either one or two ε4 alleles.

As there is evidence that depression can be correlated with cognitive decline (Jorm, 2000; Schmutte *et al.*, 2007), we also included it as a covariate. Depressive symptoms at the time of evaluation were assessed

with the 10-item version of the Center for Epidemiologic Studies-Depression scale (Radloff, 1977; Irwin *et al.*, 1999). A conventional cutoff score of 4 or higher was used to indicate presence of depressive symptoms (Grunebaum *et al.*, 2008; Geerlings *et al.*, 2012).

Statistical analyses

Analyses were performed using SPSS 22 (SPSS, Chicago, Illinois, USA). Baseline characteristics of subjects were compared using *t*-test or analysis of variance for continuous variables (i.e., age, education, cognition *z*-score), and with chi-square test for categorical baseline characteristics (i.e., gender, ethnicity, depression, *APOE*-ε4 genotype). We examined the cross-sectional relationship of sleep with cognitive performance at baseline using general linear models.

In order to assess the relationship between the sleep variables and the rate of cognitive decline, we used generalized estimating equations (GEE) analyses. Models were initially unadjusted and then adjusted for age at the visit, gender, education, ethnicity, depression, and *APOE*-ε4 genotype. GEE takes into account the multiple visits per subject and the possibility that the characteristics of the same individual are likely to be correlated over time. The repeated measures for each subject are treated as a cluster (Scarmeas *et al.*, 2006). Each GEE model included the cognitive scores as the dependent variable, with time (years), sleep variables, and an interaction term between time and sleep variables as predictors. A significant interaction term indicates a differential cognitive change over time as a function of the sleep variable. A positive *B* value means the more the sleep problems increase, the better is the performance.

Initial analyses included all participants with available sleep and cognitive information. In order to exclude the possibility of having significant cognitive changes because of a diagnosis of dementia, we also ran sensitivity analyses excluding subjects who were demented at the baseline evaluation.

Results

Cross-sectional analyses

In the cross-sectional analyses including all participants ($n = 2358$) (Table 2), increased daytime somnolence was associated with poorer language ability ($B = -0.329$, $p = 0.006$).

In the analyses including only the non-demented participants [$n = 2112$ (we excluded 237 demented and six missing their cognitive status information)], increased daytime somnolence was associated with slower speed of processing ($B = -0.143$, $p = 0.047$) after adjusting for age, demographic variables, *APOE*-ε4 genotype, and depression. There were no statistically significant relationships between the other sleep variables and any of the cognitive domain measures.

Longitudinal analyses

In the longitudinal analyses, from the sample of 1098 participants, mean baseline age was 75.3 (*SD*: 6.1)

Table 2 Associations between sleep variables and cognitive domains of the total sample ($n = 2358$). Cross-sectional results

Sleep variable		Language	Speed of processing	Executive function	Memory
Sleep disturbance	Unadjusted	$B = -0.408$ $p = 0.000$	$B = -0.167$ $p = 0.029$	$B = -0.549$ $p = 0.000$	$B = -0.126$ $p = 0.225$
	Adjusted	$B = 0.051$ $p = 0.747$	$B = -0.033$ $p = 0.711$	$B = -0.151$ $p = 0.369$	$B = 0.001$ $p = 0.990$
Snoring	Unadjusted	$B = -0.131$ $p = 0.004$	$B = -0.049$ $p = 0.109$	$B = -0.218$ $p = 0.000$	$B = 0.000$ $p = 0.994$
	Adjusted	$B = 0.035$ $p = 0.577$	$B = -0.012$ $p = 0.741$	$B = -0.084$ $p = 0.216$	$B = 0.050$ $p = 0.308$
Sleep short of breath/headache	Unadjusted	$B = -0.054$ $p = 0.019$	$B = -0.015$ $p = 0.311$	$B = -0.059$ $p = 0.021$	$B = -0.010$ $p = 0.626$
	Adjusted	$B = -0.029$ $p = 0.367$	$B = -0.009$ $p = 0.622$	$B = -0.029$ $p = 0.396$	$B = -0.017$ $p = 0.497$
Sleep adequacy	Unadjusted	$B = 0.106$ $p = 0.127$	$B = 0.031$ $p = 0.513$	$B = 0.045$ $p = 0.568$	$B = 0.014$ $p = 0.823$
	Adjusted	$B = 0.074$ $p = 0.441$	$B = 0.001$ $p = 0.989$	$B = -0.006$ $p = 951$	$B = -0.100$ $p = 0.173$
Daytime somnolence	Unadjusted	$B = -0.583$ $p = 0.000$	$B = -0.244$ $p = 0.000$	$B = -0.452$ $p = 0.000$	$B = -0.397$ $p = 0.000$
	Adjusted	$B = -0.329$ $p = 0.006$	$B = -0.112$ $p = 0.096$	$B = -0.108$ $p = 0.394$	$B = -0.133$ $p = 0.145$

years. There was a greater percentage of women than men and more Hispanics than other ethnic groups (Table 1).

In unadjusted analyses, increased daytime somnolence was associated with a faster rate of decline in all four cognitive domains (Table 3). After adjusting for demographic variables, *APOE*-ε4 genotype, and depression, daytime somnolence was significantly associated with a faster decline in speed of processing ($B = -0.003$, $p = 0.027$) (Table 3). Apart from the sleep categories, we also examined the association between total sleep time (see question 1, 2, Appendix) and cognition; however, the results were not significant. The other sleep problem indicators were not associated with rate of cognitive decline.

From the sample of 1098, there were 73 (6.6%) subjects who were demented at baseline and two missing the cognitive status information. In analyses restricted to 1023 subjects who were non-demented at baseline evaluation, associations were essentially unchanged. Thus, increased daytime somnolence was significantly associated with a faster decline in speed of processing ($B = -0.003$, $p = 0.021$).

Discussion

In this study, we examined the relationship between daytime somnolence and decline in specific cognitive domains in a large sample of older participants. In our cross-sectional analyses, increased daytime somnolence was associated with a decrease in language ability in all participants, and with a decrease in speed of processing for the non-demented ones. However, our main focus was to examine these relationships over time. Thus, in our longitudinal analyses—over a 3.2-year-follow-up period—we found that increased daytime somnolence at baseline was associated with a faster rate of decline in speed of processing, even after eliminating participants who were demented at baseline and controlling for socio-demographic variables, depression, and *APOE*-ε4 genotype. There were no significant associations between the other sleep disturbance measures and rate of decline in any of the cognitive domains (Jelicic *et al.*, 2002; Tworoger *et al.*, 2006).

There are several possible explanations for our finding that daytime somnolence is associated with a decrease in speed of processing over time. One

Table 3 Associations between sleep variables and cognitive domains. Longitudinal results

Sleep variable		Language	Speed of processing	Executive-function	Memory
Sleep disturbance[a]	Unadjusted	$B = 0.001$ $p = 0.375$	$B = -0.002$ $p = 0.213$	$B = 0.001$ $p = 0.419$	$B = 0.001$ $p = 0.135$
	Adjusted	$B = 0.000$ $p = 0.611$	$B = -0.001$ $p = 0.210$	$B = 0.000$ $p = 0.624$	$B = 0.001$ $p = 0.409$
Snoring[a]	Unadjusted	$B = 0.001$ $p = 0.704$	$B = 0.001$ $p = 0.893$	$B = 0.002$ $p = 0.433$	$B = 0.000$ $p = 0.827$
	Adjusted	$B = 0.001$ $p = 0.315$	$B = -0.001$ $p = 0.673$	$B = 0.003$ $p = 0.072$	$B = 0.001$ $p = 0.510$
Awaken short of breath/with headache[a]	Unadjusted	$B = -0.003$ $p = 0.294$	$B = -0.011$ $p = 0.123$	$B = 0.001$ $p = 0.880$	$B = 0.004$ $p = 0.350$
	Adjusted	$B = 0.002$ $p = 0.213$	$B = -0.010$ $p = 0.109$	$B = 0.001$ $p = 0.702$	$B = 0.004$ $p = 0.199$
Sleep adequacy[a]	Unadjusted	**$B = 0.003$** **$p = 0.017$**	$B = 0.002$ $p = 0.260$	$B = 0.001$ $p = 0.281$	$B = 0.002$ $p = 0.082$
	Adjusted	$B = 0.001$ $p = 0.053$	$B = 0.000$ $p = 0.875$	$B = 0.003$ $p = 946$	$B = 0.001$ $p = 0.434$
Daytime somnolence[a]	Unadjusted	**$B = -0.033$** **$p = 0.000$**	**$B = -0.045$** **$p = 0.001$**	**$B = -0.015$** **$p = 0.038$**	**$B = -0.022$** **$p = 0.009$**
	Adjusted	$B = 0.000$ $p = 0.312$	**$B = -0.003$** **$p = 0.027$**	$B = 0.001$ $p = 0.139$	$B = 0.001$ $p = 0.420$

B value is for the sleep variable × time interaction. A significant value indicates different rate of decline as a function of the sleep variable.
Generalized estimating equation models before and after adjusting for ethnicity, education, age, gender, *APOE*-ε4, and depression.
A *p* value of less than 0.05 was considered statistically significant, and the corresponding results are shown in bold.
[a]Sleep disturbance (included questions: "How long did it usually take for you to fall asleep during the past 4 weeks", "Feel that your sleep was not quiet (moving restlessly, feeling tense, speaking, etc.) while sleeping", "Have trouble falling asleep", "Awaken during your sleep time and have trouble falling asleep again"), Snoring ("Snore during your sleep"), Awaken short of breath/ with headache ("Awaken short of breath or with a headache"), Sleep adequacy ("Get enough sleep to feel rested upon waking in the morning", "Get the amount of sleep you needed"), and Daytime somnolence ("Feel drowsy or sleepy during the day", "Have trouble staying awake during the day", "Take naps (5 min or longer) during the day").
Positive *B* value means the more the sleep problems increase, the better is the performance.

possibility is that daytime somnolence may be an initial symptom of an undetected neurodegenerative disease. Previous research has shown that daytime somnolence has been reported as an early sign of dementia (Merlino *et al.*, 2010) and is common in AD (Bonanni *et al.*, 2005). Furthermore, it has been found that amyloid deposition in the preclinical stage of AD is correlated with worse sleep quality (Ju *et al.*, 2013). While the associations were still present when we removed subjects who were demented at baseline, the clinical onset of neurodegenerative diseases is preceded by a long latent period of neuropathological and subtle subclinical findings (Iranzo *et al.*, 2006). However, further investigation would be necessary in order to clarify the possible association between pathological somnolence and neurodegeneration-related cognitive decline.

Another possible explanation for the relationship between daytime somnolence and speed of processing could be that depression mediates their association. Depression is strongly related with both daytime sleepiness (Fernandez-Mendoza *et al.*, 2015) and cognitive function, especially speed of processing (Lin *et al.*, 2014), as well as verbal fluency and memory (Naismith *et al.*, 2009). Or, it may be that sleep fragmentation and sleep disturbance increase depressive symptoms (Maglione *et al.*, 2014), which could then influence the rate of cognitive decline. It has been found that poor sleep quality is most strongly associated with cognition, particularly in subclinical depressed older adults (Sutter *et al.*, 2012). Further research examining whether depression has a mediating role in the association between daytime somnolence and speed of processing may shed light on this particular issue.

A very interesting neurobiological theory that connects both daytime somnolence and speed of processing is the underlying mechanism of white matter integrity (WMI). According to the existing literature, WMI has been linked to performance in the executive function, and specifically to speed of processing in older adults (O'Sullivan *et al.*, 2005; Sasson *et al.*, 2013; Santiago *et al.*, 2014). Furthermore, there are some studies that indirectly connect WMI with daytime somnolence (Kubota *et al.*, 2002; Yoshikawa *et al.*, 2004; Thorpy and Billiard, 2011). Thus, changes in the WMI could be the underlying neurobiological mechanism that affect both daytime somnolence and speed of processing.

Finally, we may also consider the role of the circadian rhythms in this association. It has been shown that circadian rhythms control sleep and biological processes including cell regeneration, core body temperature variations, and hormone metabolism (Buysse *et al.*, 2005; Sharma and Kavuru, 2010). There is also evidence that changes in the metabolism, especially disorders of the thyroid function, may be associated with cognitive decline (Smith *et al.*, 2002) and specifically decrease in speed of processing (Montalvo *et al.*, 2014; Smith *et al.*, 2015). Furthermore, changes in the thyroid, especially hypothyroidism, is considered a reversible cause of secondary dementia in older adults (Horvath *et al.*, 1989; Cummings, 1992). Thus, we also have to consider that the presence of daytime somnolence and slowed speed of processing may be a by-product of the various metabolic changes taking place during circadian arrhythmia. Further research is warranted in order to clarify this issue.

As "sleep adequacy" refers to nighttime sleepiness and "daytime somnolence" refers to daytime sleepiness, we would expect these two factors to occur together. However, a recent report found evidence of a dissociation between daytime sleepiness, nighttime sleep, and cognitive status (Goldman *et al.*, 2013). Furthermore, another recent study found associations between daytime sleepiness and dementia only, but not with insomnia (Merlino *et al.*, 2010). So there seems to be existing literature that supports our findings of an association between cognition and daytime somnolence, and not with nighttime sleep adequacy.

According to our results, there is a strong association between daytime sleepiness and all the four cognitive domains in the unadjusted model. However, after adjusting for the covariates, most of the associations did not occur. The possible confounders for this strong effect were age, ethnicity, and education.

There are some limitations that should be noted in the current study. First, we did not have the opportunity to compare self-reported sleep problems with objective measures of sleep such as polysomnography. According to previous studies, self-reported sleep measures tend to provide an overestimation of the exact type of sleep conditions (Lauderdale *et al.*, 2008; Van Den Berg *et al.*, 2008). Thus, further investigation using objective measures is needed in order to determine the biological markers associated with sleep problems. For example, the question about "the amount of sleep needed in order to feel rested" taps a more subjective aspect of sleep complaints. Moreover, a limitation to our study is the relatively short follow-up (mean of 3.2 years in comparison, for example, with the 10-year-follow-up study of Keage *et al.*(2012)). Unfortunately, another limitation is that we did not have the opportunity to take into account the exact time of the day that the sleep evaluation was administered. It would have been interesting to see any differences among the time of the

examination, which could have played role in our findings. As obstructive sleep apnea has been associated with brain white matter abnormalities (Chen *et al.*, 2015), a potential limitation of the specific study is that we did not have the information about how many people had obstructive sleep apnea, which could be a confounder. In our study, "waking short of breath or with headache" and "snoring" can be considered as surrogate markers for sleep apnea, and they were not associated with cognition in the study, suggesting there is limited possibility of confounding by sleep apnea. Nevertheless, we did not objectively measure sleep apnea, which might be associated with cognitive function (Ramos *et al.*, 2015); thus, we could not rule out the possibility of confounding by sleep apnea. Finally, the answers in the sleep questionnaire refer to the last 4 weeks of the visiting time and may not accurately represent chronic sleep patterns experienced by the participants.

Despite these limitations, however, the present study has several strengths. First, instead of an overall brief screening cognitive tool (i.e., a total MMSE score as used in the majority of previous studies), we used a full neuropsychological battery that measured several core aspects of cognition including memory, language ability, executive functioning, and speed of processing. This detailed assessment, added to the thorough clinical evaluation by dementia experts, permitted a fine-tuned identification of dementia (and therefore related sensitivity analyses). Second, this is the first longitudinal study that we are aware of to examine the relationship between daytime somnolence and speed of processing: previous longitudinal studies have investigated only the relationship between daytime sleepiness and global cognitive function. Furthermore, to the best of our knowledge, this study is the first to include a more varied ethnic sample in both genders, considered to be more indicative of the general population, thus resulting in greater ecological validity.

In summary, our findings suggest that increased daytime somnolence may be associated with the risk of subsequent worsening in speed of processing in older people, over and above depression, *APOE*-ε4 genotype, clinical and socio-demographic factors. Our findings fine-tune and expand on previous research that has shown an association between poor sleep quality and cognitive decline in older adults (Potvin *et al.*, 2012; Blackwell *et al.*, 2014; Miller *et al.*, 2014) by examining longitudinally the associations between specific aspects of sleep and cognitive abilities. Further investigation is required in order to pinpoint the underlying mechanisms of these associations.

Conflict of interest

None declared.

Key points

- Alzheimer's disease (AD), Apolipoprotein E4 (*APOE*-ε4), Generalized Estimating Equations (GEE).
- Self-reported sleep problems seem to reflect changes in cognition.
- Increased daytime sleepiness is associated with decreased speed of processing in the elderly.

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Author contributions

Angeliki Tsapanou: study design, interpretation of the results, preparation of the manuscript, data analysis.

Yian Gu: study design, interpretation of the results, preparation of the manuscript, data analysis.

Deirdre O'Shea: preparation of the manuscript.

Teal Eich: preparation of the manuscript.

Ming-Xin Tang: study design, data analysis.

Nicole Schupf: study design.

Jennifer Manly: preparation of the manuscript.

Nikolaos Scarmeas: study design, interpretation of the results, preparation of the manuscript, data analysis.

Yaakov Stern: study design, interpretation of the results, preparation of the manuscript, data analysis.

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Appendix

Sleep Scale from the Medical Outcomes Study

1. How long did it usually take for you to fall asleep during the past 4 weeks?

   (Circle One)
   0–15 minutes……..…1
   16–30 minutes…….0.2
   31–45 minutes…….0.3
   46–60 minutes…….0.4
   More than 60 minutes ……0.5

2. On the average, how many hours did you sleep each night during the past 4 weeks?

   Write in number of hours per night:
   How often during the past 4 weeks did you…

3. Feel that your sleep was not quiet (moving restlessly, feeling tense, speaking, etc., while sleeping)?
4. Get enough sleep to feel rested upon waking in the morning?
5. Awaken short of breath or with a headache?
6. Feel drowsy or sleepy during the day?
7. Have trouble falling asleep?
8. Awaken during your sleep time and have trouble falling asleep again?
9. Have trouble staying awake during the day?
10. Snore during your sleep?
11. Take naps (5 minutes or longer) during the day?
12. Get the amount of sleep you needed?

Possible answers: 1 = All of the time, 2 = Most of the time, 3 = A good bit of the time, 4 = Some of the time, 5 = A little of the time, 6 = None of the time, −1 = Not asked, −2 = Too impaired to respond, −3 = Refused

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