

Changes in sleep duration and the risk of incident dementia in the elderly Japanese: the Ohsaki Cohort 2006 Study

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Abstract

Study Objectives

To examine the association between changes in sleep duration and the risk of incident dementia in the elderly.

Methods

In 2006, we conducted a cohort study of 7422 disability-free Japanese individuals aged ≥ 65 years who lived in Ohsaki City, Japan. In both 1994 and 2006, the individual amount of sleep obtained was assessed using a self-reported questionnaire. Based on sleep duration at these two time points, participants were categorized into five groups according to the change in sleep duration. Data on incident dementia were retrieved from the public Long-term Care Insurance database, and the subjects were followed up for 5.7 years (between April 2007 and November 2012). The Cox proportional hazards model was used to estimate the multivariate-adjusted hazard ratios (HRs) and 95% confidence intervals (95% CIs) for incident dementia.

Results

During 36338 person-years of follow up, 688 cases of incident dementia were documented. Compared with subjects who had no change in sleep duration, the multivariate HRs (95% CIs) of incident dementia were 1.31 (1.07 to 1.60) for those whose sleep duration increased by 1 hr, and 2.01 (1.51 to 2.69) for an increase of ≥ 2 hr.

Conclusions

Increased sleep duration is associated with a significantly higher risk of incident dementia in the elderly. Future studies using well-validated measurements are needed to confirm the association between sleep and dementia.

[changes in sleep duration](#), [incident dementia](#), [aging](#), [cohort study](#), [Japanese population](#)

Statement of Significance

Previous studies have indicated that both long and short sleep duration are associated with a higher risk of cognitive impairment or incident dementia among the elderly. Only two previous studies have investigated the association between changes in sleep duration and incident dementia, but their results were inconsistent. Our present study, using data from a large-scale population-based cohort study, provided

evidence that an increase rather than a decrease in sleep duration during a 12-year period was a risk factor of incident dementia, and that an increase in sleep duration was associated with a higher risk of dementia. Future studies employing objective measurements of sleep are needed to verify this association, and the underlying mechanisms should be examined.

Introduction

In 2015, the World Health Organization reported that 47.5 million people were estimated to be living with dementia, and that this number was projected to increase to 115.4 million by 2050 [1]. Since dementia impacts considerably on quality of life as well as social security financing, it is imperative to find modifiable factors that can help to prevent or at least postpone the onset of dementia.

Sleep plays an important role in cognitive and emotional processes [2], and poor sleep health, which may include an excessively short [3–6] or long sleep duration, may be associated with cognitive impairment or incident dementia [5–7]. Among five previous studies, the largest prospective study with the longest follow-up found a V-shaped association between sleep duration and the risk of dementia in older women [6]. One study showed long sleep duration to be associated with an increased risk of dementia [7], and another noted that long sleep duration among women was related to incident dementia, as was short sleep duration among men [5]. In addition, a recent meta-analysis has linked insomnia symptoms, which can affect sleep duration, to incident Alzheimer's disease (AD) [8].

Recently, researchers have started to place emphasis on changes in sleep duration, and several studies have found an association between a change in sleep duration and cognitive decline [9–11], AD [12], or all-cause dementia [13]. Two studies have examined the association between changes in sleep duration and the risk of incident dementia, but their results were contradictory: one indicated that a reduction in sleep duration was associated with a 5-year higher risk of AD [12], while the other showed that transitioning from sleeping less than 9 hr to more than 9 hr was associated with a higher risk of incident dementia, and that a decrease in sleep duration was not connected to dementia [13]. Additionally, these studies suffered from methodological limitations such as a small sample size [12] or an insufficient number of confounders [13].

Accordingly, the aim of this large-scale population-based prospective study was to investigate whether a long-term change in sleep duration would affect the risk of incident dementia.

Methods

Study cohort

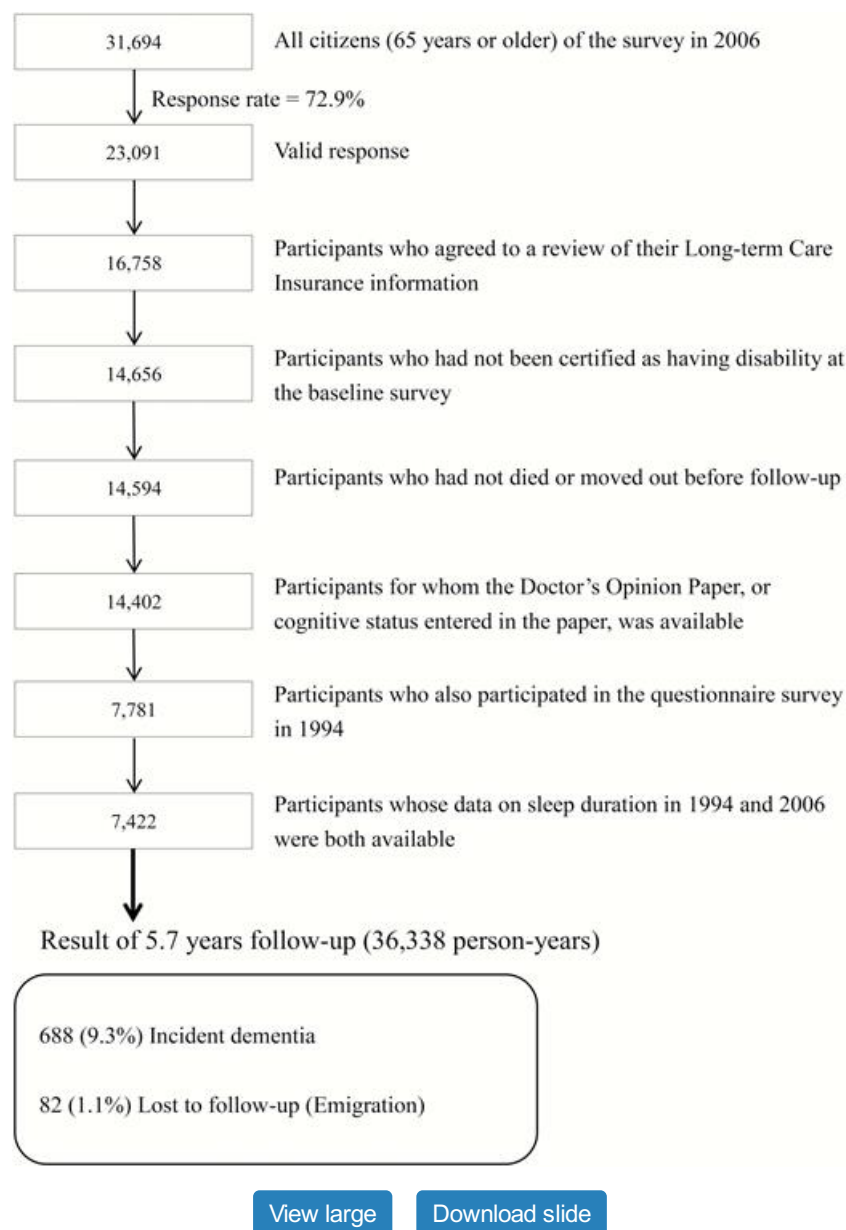
Similar to our previous studies [14, 15], we used merged data from the Ohsaki Cohort Study and the Ohsaki Cohort 2006 Study, details of which have already been described elsewhere [16, 17].

The Ohsaki Cohort 2006 Study was a population-based cohort study conducted in Ohsaki City, Miyagi Prefecture, Japan. In brief, the study subjects for the baseline survey comprised all older citizens aged ≥ 65 years. The baseline survey was conducted between December 1, 2006 and December 15, 2006, and the follow-up period was between April 1, 2007 and November 30, 2012.

In this study area, a questionnaire was also distributed between October and December 1994 (the Ohsaki Cohort Study). Although this survey included only National Health Insurance beneficiaries as the study subjects, about a half of the participants in the Ohsaki Cohort 2006 Study had also participated in the Ohsaki Cohort Study. By combining the two data sets, we obtained data on changes in sleep duration between 1994 and 2006.

In the Ohsaki Cohort 2006 Study, 23091 out of 31694 eligible subjects, provided valid responses and formed the cohort study. We excluded 6333 persons who disagreed to a review of their Long-term Care Insurance (LTCI) information, 2102 who had already been certified as having disability by the LTCI before follow-up, 62 who had died or emigrated from the study district before follow-up, 192 whose Doctor's Opinion Paper (DOP) or cognitive status entry on it was unavailable, 6621 who had not participated in the 1994 Survey, and 359 whose data on sleep duration in either 1994 or 2006 were missing. Eventually, a total of 7422 subjects were included for the purpose of this study (Figure 1).

Figure 1.



Flow chart of study participants.

Change in sleep duration (exposure)

We obtained data on sleep duration in 2006 and 1994, respectively, by asking the same question, “How many hours on average do you sleep per day?” representing their mean 24-hr sleep duration in the previous year. In 1994, participants were asked to enter an integer number to answer this question; also in 2006, participants were required to choose from the following options: “≤5 hr,” “6 hr,” “7 hr,” “8 hr,” “9 hr,” and “≥10 hr,” which are also integer numbers, to represent their sleep duration.

Based on self-reported sleep duration at the two time points, we categorized the participants into five groups: (1) decrease in sleep duration by ≥ 2 hr, (2) decrease in sleep duration by 1 hr, (3) no change in sleep duration (i.e. reference group), (4) increase in sleep duration by 1 hr, and (5) increase in sleep duration by ≥ 2 hr.

Follow-up (incident dementia)

The primary outcome was incident dementia, defined as disabling dementia according to the criteria of the LTCI system that has been implemented in Japan since April 2000 [18]. The LTCI is a mandatory form of national social insurance to assist activities of daily living in the disabled elderly [19–21]. Everyone aged 40 years or older paying premiums and everyone aged 65 years or older is eligible for formal caregiving services under a uniform standard of disability certification. The procedure for disability certification comprises two parts: assessment of the degree of functional disability using a questionnaire developed by the Minister of Health, Labour and Welfare, and reference to the DOP prepared by the attending physician [22]. The DOP is a standard form used for assessing patients’ chronic medical conditions and functions of daily life.

Disabling dementia was defined as incident functional disability with dementia according to the LTCI system, whereby the dementia exceeded rank I (rank II) on the Dementia Scale (Degree of Independence in Daily Living for Elderly with Dementia), as entered on the DOP. The Dementia Scale is classified into six ranks: 0, I–IV, M; Rank M means that an individual has severe dementia-related behavioral disturbance that requires medical intervention, and a rank exceeding I is typically used as an outcome measure of incident dementia because individuals who have mild or moderate dementia are classified as rank II [18, 23–25]. A previous study has shown that the Dementia Scale is well correlated with the Mini-Mental State Exam score (Spearman rank correlation coefficient: -0.736) [26].

We obtained a data set that included information on LTCI certification, death or emigration from Ohsaki City. All data were transferred from the Ohsaki City Government under an agreement related to Epidemiologic Research and Privacy Protection.

Covariates

Body mass index (BMI) was calculated as the self-reported body weight (kg) divided by the square of the self-reported body height (m^2). Education level was defined as the age when subjects had completed their education. The K6 was used as an indicator of psychological distress [27, 28]. Using six questions, respondents were asked about their mental status over the previous month. Total point scores ranged from 0 to 24. As the optimal cut-off point for mental illness in the validation study, we classified individuals with scores of ≥ 13 as having psychological distress [28].

Ethical issues

We considered the return of completed questionnaires to imply consent to participate in the study involving the baseline survey data and subsequent follow-up of death and emigration. We also confirmed information regarding LTCI certification status after obtaining written consent along with the questionnaires returned from subjects at the time of the baseline survey in 2006. The Ethics Committee of Tohoku University Graduate School of Medicine (Sendai, Japan) reviewed and approved the study protocol.

Statistical analyses

We counted the person-years of follow-up for each subject from April 1, 2007 until the date of incident dementia, date of emigration from the study area, date of death, incident functional disability without dementia, or the end of the study period (November 30, 2012), whichever occurred first. In our analysis, deaths without LTCI certification were treated as censored.

We used the multiple adjusted Cox proportional hazards model to calculate the hazard ratios (HRs) and 95% confidence intervals (95% CIs) for incident dementia according change in sleep duration. Dummy variables were created for the categories of change in sleep duration, and respondents who showed no change in sleep duration were defined as a reference category. To test for linear trends, categories indicating an increase in sleep duration were entered as a continuous term (scored as 1 for “no change,” 2 for “an increase by 1 hr,” and 3 for “an increase by ≥ 2 hr”) in the corresponding Cox model; the same linear trend test was also conducted for the categories indicating a decrease in sleep duration. Multivariate models were adjusted for the following variables. Model 1 was adjusted for sex and age (65–69, 70–74, 75–79, 80–84, or ≥ 85 years). Model 2 was further adjusted for BMI (<18.5 , 18.5–25, ≥ 25 , or missing), education level (age at completion of education: <16 years, ≥ 16 years, or missing), smoking status (never, former, current, or missing), alcohol drinking status (never, former, current, or missing), psychological distress score (<13 , ≥ 13 , or missing), pain (no, mild, moderate or more, or missing), history of diseases (stroke, hypertension, myocardial infarction, diabetes, or hyperlipidemia—yes/no; for each term), and sleep duration in 1994 (≤ 5 , 6, 7, 8, or ≥ 9 hr).

Interactions between an increase in sleep duration and sex, age (<75 or ≥ 75 years), or education level (<16 or ≥ 16 years) were tested by addition of cross-product terms for each of the dummy variables to the multivariate models; the interaction test was also conducted for the categories indicating a decrease in sleep duration.

Considering possible reverse causality, we conducted a sensitivity analysis by excluding subjects whose dementia had developed within the first 2 years of follow-up. Furthermore, we conducted further sensitivity analysis to investigate whether different combinations of sleep duration at the two time points affect the risk of incident dementia. We divided sleep duration in 1994 and 2006 into three categories: (1) short (≤ 6 hr), (2) normal (7 or 8 hr), and (3) long (≥ 9 hr), respectively. We then obtained nine groups of participants with different patterns of change in sleep duration by combining the sleep durations in 1994 and 2006. The multivariate Cox proportional hazards model was also used, which was adjusted for the same variables as those in model 2, using subjects with persistent normal sleep duration as the reference group.

To evaluate the possible influence of competing events on the association between changes in sleep duration and the risk of incident dementia, survival analysis was also conducted using competing-risk regression models. Competitive events were defined as (1) death and (2) any other types

of disability and death together.

All data were analyzed using SAS version 9.4 (SAS, Inc.). All statistical tests described here were two-sided, and differences at $p < 0.05$ were accepted as significant.

Results

Subject characteristics

During the 5.7-year follow-up period, 82 persons were lost to follow-up because of migration from the study area, leaving a follow-up rate of 98.9%. Among a total of 36338 person-years, 688 cases of incident dementia were documented, accounting for 9.3% of total study participants. [Table 1](#) shows the baseline characteristics of the study participants according to changes in sleep duration. Individuals without any change in sleep duration were less likely to live with moderate or more severe pain. Individuals with a decrease in sleep duration of 2 hr or more tended to be female, spend less time walking and have psychological distress.

Table 1.
Characteristics of participants by changes in sleep duration at baseline in 2006 ($n = 7422$)

	Changes in sleep duration					<i>P</i> *
	Decrease by ≥2 hr	Decrease by 1 hr	No change	Increase by 1 hr	Increase by ≥2 hr	
No. of participants	624	1468	2957	1784	589	
Age, year (mean ± SD)	74.9 ± 5.4	74.6 ± 5.3	74.6 ± 5.6	74.6 ± 5.6	75.2 ± 5.7	0.1325
Female, %	59.8	59.6	54.2	53.8	57.9	0.0006
Body mass index (mean ± SD)	23.4 ± 3.6	23.5 ± 3.2	23.6 ± 3.4	23.5 ± 3.3	23.5 ± 3.5	0.5987
Education level (<16 year), % [†]	32.6	31.2	29.4	31.6	34.7	0.0955
Current smoker, %	10.7	13.3	13.7	14.0	13.0	0.2577
Current alcohol drinker, %	33.6	33.4	37.0	36.5	33.3	0.2659
Time spent on walking (≤0.5 hr/day), %	37.7	35.3	33.8	34.8	35.6	0.0236
Pain (moderate or more severe), %	32.9	29.4	26.4	28.3	32.0	0.0413
Psychological distress, % [‡]	9.0	4.2	4.4	5.2	5.5	0.0003
History of diseases, %						
Stroke	2.7	2.5	2.9	3.1	2.6	0.8797
Hypertension	41.8	42.5	45.0	43.2	43.0	0.4067
Myocardial infarction	5.0	4.1	4.9	4.7	6.3	0.3317
Diabetes	11.2	9.3	11.2	11.6	12.9	0.1182
Dyslipidemia	6.1	6.3	7.9	8.2	6.5	0.1176

**p* Values were calculated by using χ^2 test for variables of proportion and one-factor ANOVA for continuous variables.

[†]Age at completion of education.

[‡]Kessler 6-item psychological distress scale score ≥13.

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Changes in sleep duration and incident dementia

Table 2 shows the association between changes in sleep duration and incident dementia, along with the HRs and 95% CIs. An increase in sleep duration was associated with incident dementia in model 1 adjusted for age and sex; the incident dementia HR (95% CI) was 1.29 (1.07 to 1.56) for participants with an increase in sleep duration of 1 hr and 1.72 (1.33 to 2.22) for participants with an increase of ≥2 hr (*p*-trend < .0001). Even in model 2 further adjusted for more confounders, we found that the association did not change substantially, with HR (95% CI) 1.31 (1.07 to 1.60) for an increase in sleep duration of 1 hr and 2.01 (1.51 to 2.69) for an increase of ≥2 hr (*p*-trend < .0001) (Table 2).

Table 2.
Association between changes in sleep duration and incident dementia (*n* = 7422)

	Changes in sleep duration					<i>p</i> -Trend for decrease [†]	<i>p</i> -Trend for increase [†]
	Decrease by ≥2 hr	Decrease by 1 hr	No change	Increase by 1 hr	Increase by ≥2 hr		
No. of participants	624	1468	2957	1784	589		
No. of cases	59	119	243	189	78		
Person-years of follow-up	3042	7290	14533	8741	2733		
Incident rate/1000 person-years	19.4	16.3	16.7	21.6	28.5		
Model 1 [‡]	1.09 (0.82 to 1.45)	0.98 (0.78 to 1.21)	1.00 (ref.)	1.29 (1.07 to 1.56)**	1.72 (1.33 to 2.22)***	0.6726	<.0001
Model 2 [§]	0.93 (0.70 to 1.25)	0.96 (0.77 to 1.20)	1.00 (ref.)	1.31 (1.07 to 1.60)**	2.01 (1.51 to 2.69)***	0.4911	<.0001
Model 3	1.02 (0.72 to 1.43)	1.05 (0.82 to 1.36)	1.00 (ref.)	1.35 (1.07 to 1.71)*	2.05 (1.45 to 2.88)***	0.8899	<.0001

Significance of bold values: **p* < 0.05; ***p* < 0.01; ****p* < 0.0001. Hazard ratios (HRs) and 95% confidence interval (95% CIs) were calculated by Cox proportional hazards models.

[†]*p* Values for trend were calculated by entering the categories as a continuous term in the Cox model.

[‡]Model 1 was adjusted for sex and age (65–69, 70–74, 75–79, 80–84, or ≥85 years).

[§]Model 2 was adjusted for model 1 plus sleep duration in 1994 (≤5, 6, 7, 8, or ≥9 hr), BMI (<18.5, 18.5–25, ≥25, or missing), history of diseases (stroke, hypertension, myocardial infarction, diabetes, or hyperlipidemia [yes or no]), smoking status (never, former, current, or missing), alcohol drinking status (never, former, current, or missing), education level (age when leaving school: <16 years, ≥16 years, or missing), pain (no pain, mild pain, moderate pain or more, or missing), psychological distress score (<13, ≥13, or missing), and time spent on walking (≥1 hr, 0.5–1 hr, <0.5 hr, or missing).

^{||}Model 3 was adjusted for the same covariates in model 2 after excluding cases occurring in the first 2 years since follow-up (*n* = 7232).

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Sensitivity analyses

In model 3 adjusted for the same variables as in model 2, the association remained robust after excluding 190 cases of incident dementia that developed in the first two years of follow-up; the HR (95% CI) was 1.35 (1.07 to 1.71) for an increase in sleep duration of 1 hr and 2.05 (1.45 to 2.88) for ≥ 2 hr (p -trend < 0.0001) (Table 2).

In the other sensitivity analysis, an increase in sleep duration from short to long was associated with a higher but not significant risk of incident dementia, with multiple-adjusted HR(95% CI) 1.82 (0.85 to 3.87), whereas an increase from normal to long was significantly related to an increased risk of dementia, with HR (95% CI) 1.39 (1.13 to 1.72). However, an increase in sleep duration from short to normal had no association with dementia with HR (95% CI) 1.04 (0.61 to 1.77) (Table 3).

Table 3.

Association between changes in sleep duration and incident dementia with nine categories ($n = 7422$)*

Sleep duration in 1994		Sleep duration in 2006		
		Short (≤ 6 hr)	Normal (7–8 hr)	Long (≥ 9 hr)
Short (≤ 6 hr)	Incident rate/1000 person-years, HR (95% CI)	17.1, 0.98 (0.61 to 1.57)	15.8, 1.04 (0.61 to 1.77)	36.9, 1.82 (0.85 to 3.87)
Normal (7–8 hr)	Incident rate/1000 person-years, HR (95% CI)	15.7, 0.85 (0.65 to 1.13)	15.9, 1.00 (ref.)	27.7, 1.39 (1.13 to 1.72)**
Long (≥ 9 hr)	Incident rate/1000 person-years, HR (95% CI)	19.6, 0.93 (0.43 to 1.98)	21.9, 1.03 (0.76 to 1.40)	24.7, 1.01 (0.75 to 1.34)

*Hazard ratios (HRs) and 95% confidence interval (95% CIs) were calculated by the multivariate Cox proportional hazards model which was adjusted for the same covariates as model 2 in Table 2. Significance of bold values: ** $p < 0.01$.

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Given that deaths free from incident dementia were censored in our analysis, we conducted an additional survival analysis using the competing-risk regression models. However, the association remained unchanged when death was considering as a competing event; the multivariate HRs (95% CIs) were 1.31 (1.07 to 1.60) for an increase in sleep duration of 1 hr and 1.87 (1.39 to 2.52) for an increase of ≥ 2 hr (p -trend < .0001). We observed similar results when other types of disability and death were considered as competing events (Table 4). As for the nine categories of changes in sleep duration, the results did not change substantially from those in Table 3 (Supplementary Tables S1 and S2). Additionally, we were unable to find any interaction effects of sex, age, and education level with an increase in sleep duration (data not shown).

Table 4.

Association between changes in sleep duration and incident dementia with competing-risk models ($n = 7422$)

	Changes in sleep duration					p-Trend for decrease	p-Trend for increase
	Decrease by ≥2 hr	Decrease by 1 hr	No change	Increase by 1 hr	Increase by ≥2 hr		
Competing-risk model of death							
Event of interest (dementia)	59	119	243	189	78		
Incident rate/1000 person-years	19.4	16.3	16.7	21.6	28.5		
Competing event	71	138	306	182	68		
Censored value	494	1211	2408	1413	443		
Multivariate HRs (CIs)	0.98 (0.73 to 1.31)	0.99 (0.79 to 1.23)	1.00 (ref.)	1.31 (1.07 to 1.60)*	1.87 (1.39 to 2.52)***	0.7378	<.0001
Competing-risk model of other types of disability and death							
Event of interest (dementia)	59	119	243	189	78		
Incident rate/1000 person-years	19.4	16.3	16.7	21.6	28.5		
Competing event	124	257	574	331	142		
Censored value	441	1092	2140	1264	369		
Multivariate HRs (CIs)	0.99 (0.74 to 1.33)	0.99 (0.79 to 1.23)	1.00 (ref.)	1.31 (1.07 to 1.60)**	1.79 (1.34 to 2.41)***	0.8262	0.0002

Significance of bold values: **p* < 0.05; ***p* < 0.01; ****p* < 0.0001.

Hazard ratios (HR) and 95% confidence interval (95% CI) were calculated by the multivariate competing-risk models which were adjusted for the same covariates as model 2 in [Table 2](#).

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Discussion

In this cohort study, we investigated the association between changes in sleep duration and incident dementia. We observed that an increase in sleep duration over the 12-year period was significantly associated with an increased risk of incident dementia, and that individuals with an extreme increase in sleep duration had a twofold risk of incident dementia. Additionally, we found no association between a decrease in sleep duration and incident dementia.

Considering the possible reverse causality that sleep complaints are common among older adults with dementia, we conducted a sensitivity analysis by excluding individuals who developed incident dementia in the first 2 years of follow-up. However, the association between an increase in sleep duration and dementia remained significant, suggesting that reverse causality is less likely to interrupt the observed association.

To our knowledge, only two studies have examined the association between changes in sleep duration and incident dementia, and these yielded contradictory results [12, 13]. A Swedish study reported that a reduction in sleep duration was associated with the incident risk of dementia [12], whereas the Framingham Heart Study reported that an increase in sleep duration showed such an association [13]. However, the two study designs were totally different. In the former study (214 subjects, aged ≥75 years), the cohort examined was smaller and older than that in the latter (2457 subjects, aged ≥60 years), suggesting that the findings of the former study may have been more subject to chance. The follow-up period was shorter in the former study (6.9 years on average) than in the latter (10 years). The former study examined only the risk of reduced sleep duration, and thus the risk of an increase in sleep duration was unknown. On the other hand, the latter study examined the risk of both an increase and a decrease in

sleep duration, and found that an increase in sleep duration was significantly associated with a higher risk of incident dementia. Considering these differences in methodology between the two studies, we concluded that the latter study would provide more valid findings, and the results of our own study were consistent with those findings.

The mechanisms underlying the association between increased sleep duration and incident dementia remain unclear; however, some potential mechanisms can be suggested. For instance, longer sleep duration may be associated with elevated levels of inflammatory biomarkers such as C-reactive protein, interleukin-6, and tumor necrosis factor [29, 30], which play a fundamental role in AD pathogenesis [31]. Second, an increase in sleep duration may reflect poor sleep quality, which has been noted among long sleepers [32, 33], and previous studies have indicated that poor sleep quality may be a risk factor for incident dementia [34, 35]. Third, sleep-disordered breathing, which can cause excessive daytime sleepiness leading to more time spent napping [4, 36], may result in an increase in total sleep duration among the elderly. One cohort study has pointed out that older women living with sleep-disordered breathing had a higher risk of dementia than those without [37], and a recent review has also reported an association between sleep-disordered breathing and incident dementia [8]. In addition, considering that most demented older adults show changes in sleep patterns, and the onset of dementia may extend over decades, we recognize that the association between increased sleep duration and dementia may be interactive and bidirectional, and that an increase in sleep duration may be an early sign of dementia.

However, this study also had some limitations that should be mentioned. First, change in sleep duration was calculated based on single-item and self-reported sleep duration and it is unclear to what extent changes in subjective sleep duration reflect objective changes. BMI was also calculated based on self-reported height and weight, and this issue may have affected our results. Second, we were unable to distinguish nighttime sleep from daytime napping, which is common among older adults. Third, we had no data on the subtypes of dementia (e.g. AD and vascular dementia) based on the LTCI certification we utilized. Although these dementia types have overlapping risk factors, it would have been valuable to address whether an increase in sleep duration could specifically influence one or more of these dementia types to better understanding the underlying mechanisms. Fourth, some sleep-related variables that we did not assess in our study may have been risk factors for incident dementia, like poor sleep quality, sleep-disordered breathing, excessive daytime sleepiness, and circadian rhythm disturbances [36]. Finally, not all study candidates applied for LTCI certification; therefore, we cannot rule out the possibility of detection bias. Despite all these limitations, our study had several strengths including (1) a large population-based cohort with 7424 elderly participants, (2) a high rate of follow-up (98.9%), and (3) considerable control of confounding factors.

In conclusion, we have observed a significant association between increased sleep duration and the risk of incident dementia in an elderly population. Further studies using well-validated measurements will be needed to confirm the association between sleep length and dementia.

Supplementary material

Supplementary material is available at *SLEEP* online.

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Conflict of interest statement. None declared.

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