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Short-term changes in sleep duration and risk of type 2 diabetes

Kailuan prospective study

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Abstract

Evidence suggests short or long sleep duration is associated with a higher risk of diabetes. Using a large longitudinal data set spanning 2 years, we examined whether a change in sleep duration is associated with diabetes.

Current analysis included 56,588 participants who were free of diabetes during both 2006–2007 (exam1) and 2008–2009 (exam2). Sleep duration was categorized into 7 groups: ≤5.5 hours, 6.0 to 6.5 hours, 7.0 hours, 7.5 to 8.0 hours, ≥8.5 hours, decrease ≥2 hours, and increase ≥2 hours. Cox proportional hazards models were used to calculate hazard ratios (HRs) and their confidence intervals (CI) for diabetes, according to sleep duration.

Compared to the reference group of persistent 7-h sleepers, participants who slept 7.5 to 8 hours per night (HR, 1.20; 95% CI, 1.02–1.40), ≥8.5 hours per night (HR, 1.37; 95% CI, 1.03–1.81) and an increase of ≥2 hours sleep per night (HR, 1.24; 95% CI, 1.05–1.48) were all associated with an increased risk of developing diabetes in analyses adjusted for age, sex, education level, income level, smoking status, drinking status, physical activity, BMI, snoring status, hypertension, hyperlipidemia, and family history of diabetes. The abovementioned associations of sleep duration and incident diabetes were only prominent among individuals aged <64 years.

This study suggests that individuals whose sleep duration increases ≥2 hours per night are at an increased risk of diabetes.

Abbreviations: BMI = body mass index, CI = confidence intervals, DBP = diastolic blood pressure, FBG = fasting blood glucose, HDL-C = high-density lipoprotein-cholesterol, HR = hazard ratios, hs-CRP = high sensitive C-reactive protein, SBP = systolic blood pressure, TC = total cholesterol, TG = triglycerides.

Keywords: change, cohort study, diabetes, sleep duration

1. Introduction

According to the International Diabetes Federation, the estimated number of diabetic patients worldwide will rise to 592 million by 2035.[1] In China, a national survey in 2007–2008 showed that there were 92.4 million adults with diabetes and 148.2 million adults with prediabetes.[2] Given that diabetes is becoming a worldwide pandemic, identification of modifiable risk factors associated with the development of diabetes is important to public health. Sleep is essential in many physical, cognitive, and psychological processes. And an optimal sleeping condition appears to be a health imperative.[3] Recent meta-analyses of prospective studies have provided evidence of a U-shaped association between sleep duration and a higher incidence of type 2 diabetes, with both short and long sleep duration associated with greater risk.[4–6] The mechanisms underlying the sleep-diabetes relationship remain unsettled. Prior studies have shown that shortened sleep is related to glucose intolerance, insulin resistance, and reduced acute insulin response to glucose.[7–9] Conversely, the PPP-Botnia Study suggested that long but not short sleep duration is associated with insulin resistance and insulin secretion in individuals without diabetes.[10] Thus, it is necessary to assess the temporal relationship between sleep duration and diabetes.

An inherent limitation of previous studies, however, has been the reliance on a single time point by which to assess sleep duration, which may have occurred several decades prior to the event and is, therefore, likely to yield biased estimates of the association. Moreover, there has been no consideration of how sleep duration vary within individuals over time and the subsequent impact that this would have on the change to sleep duration and future risk of disease. Data waves from Whitehall II study spanning >5 years suggest that individuals whose sleep duration increases >2 hours are at an increased risk of type 2 diabetes.[11] Another cohort conducted in the Nurses' Health

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QS and XL contributed equally to this work and share the first authorship. XW and SW share the last authorship.

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Study spanning 14 years also showed that increases in sleep duration among middle-aged and older women were modestly associated with risk of diabetes.[12] Both the above studies examined whether long-term changes in sleep duration and the future risk of diabetes. Our study using data from the Kailuan study newly assesses whether short-term (2 year) changes in self-reported sleep duration in subsequent diabetes risk.

2. Methods

2.1. Study design and participants

The Kailuan study was a prospective cohort study designed to investigate the association of risk factors and chronic disease. The Kailuan community, located at the center of Kailuan Coal Industry in Hebei Province, China, has ~7.2 million inhabitants with 11 hospitals responsible for their healthcare. From June 2006 to October 2007, a total of 155,418 employees (including retired individuals) in the community were invited to participate and 65.31% of them agreed to be participants. A total of 101,510 participants (81,110 men and 20,400 women, aged 18–98 years old) were recruited into the Kailuan study. The follow-up evaluations included biennial measurement of laboratory parameters and recording of adverse events. All the doctors and nurses had rigorous unified training before they conducted this study. The protocol for this study was approved by the Ethics Committee of Kailuan General Hospital in compliance with the Declaration of Helsinki and all participants provided informed written consent with their signatures.[13,14]

2.2. Assessment of potential covariates

All participants underwent a clinical examination and a standardized interview. Physical activity was evaluated based on the responses to questions regarding the types and frequencies of physical activity at work and during leisure time. Physical activity was classified as "≥4 times per week and≥20 minutes at a time," "<80 minutes per week," or "none." Smoking and drinking status was classified as "never," "former," or "current" according to self-reported information. Monthly income per family member (at baseline) was categorized as "<¥600," "¥600–799," "¥800–999," and "≥¥1,000."

Anthropomorphic parameters such as height and weight and waist circumference were measured. The body mass index (BMI) was calculated as weight/height (kg/m^2). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured thrice in the seated position using a mercury sphygmomanometer, and the average of the 3 readings was used for the analyses.

Blood samples were collected from the antecubital vein after an overnight fast. Venous blood was obtained for determination of routine chemistry, including fasting blood glucose (FBG), high density lipoprotein-cholesterol (HDL-C), total cholesterol (TC), and triglycerides (TG). High sensitive C-reactive protein (hs-CRP) was measured by high-sensitivity nephelometry assay (Cias Latex CRP-H, Kanto Chemical, Tokyo, Japan).

2.3. Assessment of sleep duration

Sleep duration was elicited by the question "How many hours of sleep do you have on an average night in the preceding 3 months?" Response categories were ≤5, 6, 7, 8, and ≥9 hours. Sleep duration in 2006–2007 and 2008–2009 was used to determine changes in sleep duration over 2 exposure periods. To calculate change, baseline sleep duration was subtracted from the sleep duration reported at follow-up (2008–2009). As sleep duration was measured only in whole numbers of hours, durations of sleep that differed by 0 or 1 hour between successive phases were considered not to be different and classified as "no change in sleep duration." For these "stable" sleepers, average sleep duration was calculated and categorized into 5 levels: ≤5.5, 6.0 to 6.5, 7.0, 7.5 to 8.0 and ≥8.5 hours. Decreased sleep was defined as a decrease of ≥2 hours and increased sleep as an increase of ≥2 hours in sleep duration.

In addition, participants were asked "Do you generally snore when you sleep?" Response alternatives were "yes" and "no."

2.4. Follow-up and diagnosis of diabetes

Participants were followed up by face-to-face interviews at every 2-year routine medical examination until December 31, 2015, or to the event of interest or death. Follow-ups were performed by trained physicians who were blinded to the baseline data. In line with the ADA guidelines, participants were diagnosed with diabetes mellitus if they were currently treated with insulin or oral hypoglycaemic agents, or had a FBG concentration ≥ 7.0 mmol/L.[15]

2.5. Statistical analysis

Continuous variables were expressed as means ± standard deviations and categorical variables as percentages. We compared the parameters according to the sleep duration group. One-way analysis of variance (ANOVA) was used for nonpaired samples of normally distributed parameters and the Kruskal–Waillis test for nonparametric variables. The chi-squared test was applied for the comparison of categorical variables. A multivariate analysis was performed using 3 models. Model 1 was adjusted for age, sex, and sleep duration at baseline; Model 2 included model1 parameters plus monthly income per family member, education level, marital status, smoking status, drinking status, physical activity, and snoring status; Model 3 included independent parameters analyzed in Model 2, as well as BMI, hypertension, hyperlipidemia, and family history of diabetes. We used Cox proportional-hazards modeling to calculate the hazard ratios (HR) and 95% confidence intervals (CI) of diabetes, using the group with persistent 7-hour sleep duration as a reference category. Further, as individuals with major fatal diseases could impact our assessment of sleep duration and future diabetes risk, we conducted a sensitivity analyses to test the robustness of our findings by repeating our aforementioned analysis and excluding individuals with stroke, myocardial infarction, and cancer, respectively. Finally, smoking status, drinking status, physical activity, BMI, blood pressure, and lipids level might change during the study, which may affect the results. Therefore, we conducted another sensitivity analyses by adjusting these factors (smoking status, drinking status, physical activity, BMI, blood pressure, and lipids level) measured both in 2006 and 2008 surveys. The interaction of sleep duration with age on their risk of diabetes was analyzed by multivariate Cox proportional-hazards modeling. All statistical tests were 2-tailed, with a significance level set at $P < 0.05$. Statistical analysis was performed using the SAS 9.3 statistical software (SAS Institute Inc., Cary, NC).

3. Results

Among of 101,510 participants in the Kailuan study, a total of 44,922 participants were excluded from the recruited population, including 26,149 participants lacking face-to-face follow-up data

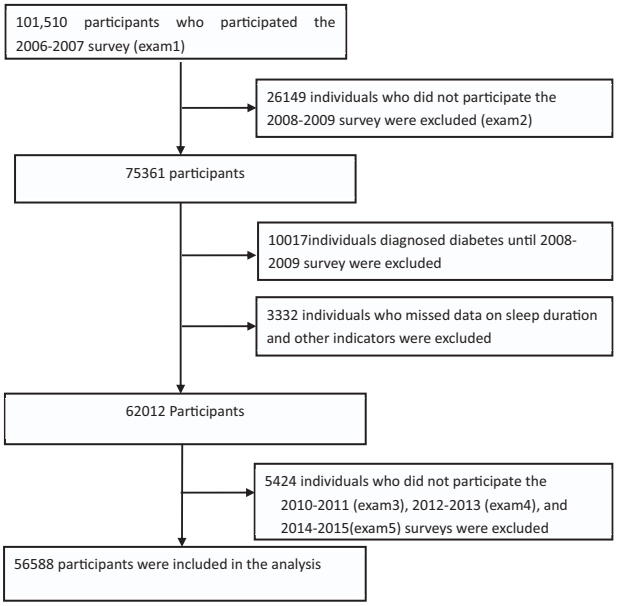

Figure 1. Selection of Kailuan study participants for analysis.

during 2008–2009 survey, 10,017 participants diagnosed with diabetes until the 2008–2009 survey, and 3332 participants lacking complete data regarding sleep duration and other indicators. In addition, 5424 individuals who did not participate in the 2010–2011, 2012–2013, and 2014–2015 surveys were excluded. The remaining 56,588 participants (43,494 men and 13,094 women, mean aged 49 years old) were included in the final analysis (Fig. 1).

Table 1 shows the general characteristics of the study participants according to incident diabetes. Baseline SBP, DBP, BMI, TG, and FBG were significantly higher, and the proportion of stroke, myocardial infarction, and family history of diabetes were significantly higher in those who developed diabetes compared with nondiabetes ($P < 0.001$).

Tables 2 and 3 show demographic and other characteristics at year 2006 and 2008 by the sleep duration group. Significant association was found between sleep duration and age, sex, education level, income level, smoking status, drinking status, physical activity, BMI, SBP, DBP, FBG, TG, HDL-C, snoring status, history of stroke, myocardial infarction, cancer, and family history of diabetes ($P < 0.001$).

During an average 5.38-year follow-up, 4899 participants (8.66%) developed diabetes. Age, sex, education level, income level, smoking status, drinking status, physical activity, BMI, snoring status, hypertension, hyperlipidemia, and family history of diabetes were designated as confounding factors in model3. Compared with participants who persistent slept 7 hours, participants who slept 7.5 to 8 hours per night (HR, 1.20; 95% CI, 1.02–1.40), ≥8.5 hours per night (HR, 1.37; 95% CI, 1.03–1.81) and an increase of ≥2 hours sleep per night (HR, 1.24; 95% CI, 1.05–1.48) were all associated with an increased risk of developing diabetes after adjusting for the confounding factors. Moreover, we repeated our analysis by excluding individuals with stroke, myocardial infarction, and cancer. As a result, the association of the long and increasing sleep duration with incident diabetes risk did not alter materially (Table 4).

In stratification and interaction analysis, the abovementioned associations of sleep duration and incident diabetes were only prominent among individuals aged <64 years. Compared with the reference group of persistent 7-hour sleepers, the HRs were 1.20 (95% CI: 1.02–1.42), 1.36 (95% CI: 1.01–1.83), and 1.22 (95% CI: 1.02–1.46) for sleep duration of 7.5 to 8 hours, ≥8.5 hours, and an increase of ≥2 hours, respectively (*P* for interaction 0.03) (Table 5).

Table 6 shows the sensitivity analysis by adjusting smoking status, drinking status, physical activity, BMI, blood pressure, and lipids level measured both in 2006 and 2008 surveys. Compared with the reference group of persistent 7-hour sleepers, the HRs were 1.19 (95% CI: 1.01–1.40), 1.35 (95% CI: 1.03–1.81), and 1.22 (95% CI: 1.02–1.46) for sleep duration of 7.5 to 8 hours, ≥8.5 hours, and an increase of ≥2 hours, respectively.

Table 1

Baseline characteristics of study population by incident diabetes.

	Incident diabetes		
	No	Yes	*P* value
No. of participants	51,689	4899	
Male, n, %	39,541 (76.50)	3953 (80.69)	<0.001
Age, y	48.79±11.98	51.04±10.32	
Marital status, married, %	48931 (94.66)	4691 (95.75)	<0.01
High-school graduate, %	12379 (23.95)	911 (18.60)	<0.001
Income≥800¥, %	8069 (15.61)	621 (12.68)	<0.001
Physical activity >4 times/week, %	7843 (15.17)	826 (16.86)	<0.01
Current smoker,%	21098 (40.82)	2088 (42.62)	<0.05
Current alcohol drinker, %	22459 (43.45)	2162 (44.13)	0.36
Body mass index, kg/m^2	24.76±3.39	26.47±3.53	
Systolic blood pressure, mm Hg	127.36±19.63	135.05±20.96	
Diastolic blood pressure, mm Hg	82.11±11.27	86.34±11.93	
Fasting blood glucose, mmol/L	5.01±0.64	5.47±0.72	
Triglycerides, mmol/L	1.57±1.26	2.00±1.52	
High density lipoprotein, mmol/L	1.54±0.39	1.54±0.40	
History of stroke, %	695 (1.34)	96 (1.96)	<0.01
History of myocardial infarction, %	480 (0.93)	72 (1.47)	<0.001
History of cancer, %	158 (0.31)	15 (0.31)	0.99
Family history of diabetes, %	2360 (4.57)	318 (6.49)	<0.001
Sleep duration at baseline, h	7.28±4.13	7.24±1.90	
Snoring status (snored),%	6917 (13.40)	863 (17.64)	<0.001

4. Discussion

Our study of 56,588 participants demonstrates for the first time that short-term changes in self-reported sleep duration are associated with a subsequent risk of incident diabetes. Participants who increases in sleep duration (≥2 h/night) and consistent long sleep (≥8 h/ night) were associated with a higher risk of diabetes compared with those getting 7 hours of sleep per night. Moreover, the above associations with diabetes were persisted statistically significant for increases (≥2 h/night) in sleep duration after adjusting the confounding factors such as BMI, hypertension, hyperlipidemia, and family history of diabetes.

A 14-year follow-up survey of women enrolled in the Nurses Health Study in the United States has shown that increases in sleep duration among middle-aged and older women were modestly associated with risk of diabetes after multivariate adjustment for standard risk factors.[12] The results were similar to those in our study: compared with no change, increases in sleep duration was adversely associated with incident diabetes (≥2 h/night; OR [95% CI]: 1.30 [1.14, 1.46]).Whitehall II is another study to examine changes in sleep duration (over ~5 years) and

Table 2

Baseline characteristics (2006) according to change of sleep duration.

	Sleep duration group							
	≤5.5 h	6.0–6.5 h	7 h	7.5–8 h	≥8.5 h	≥2 h decrease	≥2 h increase	*P*
No. of participants	2955	8209	2272	27052	773	9075	6252	
Male, n, %	2327 (78.75)	6670 (81.25)	1873 (82.44)	20003 (73.94)	523 (67.66)	6898 (76.01)	5200 (83.17)	<0.001
Age, y	53.15 ± 11.31	49.38 ± 11.52	45.65 ± 12.10	47.87 ± 11.91	44.26 ± 13.02	49.96 ± 11.35	51.73 ± 11.55	
Marital status, married, %	2744 (92.86)	7693 (93.71)	2095 (92.21)	25778 (95.29)	722 (93.40)	8722 (96.11)	5868 (93.86)	<0.001
High-school graduate, %	789 (26.70)	2723 (33.17)	970 (42.69)	5729 (21.18)	282 (36.48)	1662 (18.31)	1135 (18.15)	<0.001
Income≥800¥, %	641 (21.39)	1998 (24.34)	632 (27.82)	3346 (12.37)	167 (21.60)	1036 (11.41)	870 (13.92)	<0.001
Physical activity >4 times/week, %	732 (24.77)	1673 (20.38)	374 (16.46)	3240 (11.98)	124 (16.04)	991 (10.92)	1535 (24.55)	<0.001
Current smoker,%	1624 (54.96)	4756 (57.94)	1324 (58.27)	8751 (32.35)	323 (41.79)	2906 (32.02)	3502 (56.01)	<0.001
Current alcohol drinker, %	1754 (59.36)	5139 (62.60)	1474 (64.88)	9261 (34.23)	349 (45.15)	3021 (33.29)	3623 (57.95)	<0.001
Body mass index, kg/m^2	24.74 ± 3.28	24.92 ± 3.34	24.86 ± 3.38	24.92 ± 3.48	24.75 ± 3.60	24.90 ± 3.47	24.97 ± 3.36	
Systolic blood pressure, mm Hg	129.66 ± 19.93	127.24 ± 19.19	125.30 ± 18.68	127.93 ± 20.08	123.02 ± 19.34	128.17 ± 19.83	130.12 ± 20.02	
Diastolic blood pressure, mm Hg	82.68 ± 10.96	81.98 ± 10.90	80.94 ± 10.50	82.66 ± 11.65	79.62 ± 11.16	82.49 ± 11.37	83.12 ± 11.36	
Fasting blood glucose, mmol/L	5.00 ± 0.68	5.02 ± 0.67	5.04 ± 0.69	5.06 ± 0.65	5.06 ± 0.66	5.03 ± 0.65	5.08 ± 0.68	
Triglycerides, mmol/L	1.57 ± 1.24	1.65 ± 1.30	1.71 ± 1.42	1.58 ± 1.25	1.52 ± 1.23	1.63 ± 1.36	1.59 ± 1.30	
High density lipoprotein, mmol/L	1.57 ± 0.41	1.54 ± 0.39	1.52 ± 0.38	1.53 ± 0.38	1.51 ± 0.35	1.55 ± 0.41	1.53 ± 0.38	
History of stroke, %	79 (2.67)	157 (1.91)	28 (1.23)	266 (0.98)	11 (1.42)	109 (1.20)	141 (2.26)	<0.001
History of myocardial infarction, %	61 (2.06)	92 (1.12)	15 (0.66)	194 (0.72)	11 (1.42)	66 (0.73)	113 (1.81)	<0.001
History of cancer, %	23 (0.78)	30 (0.37)	11 (0.48)	65 (0.24)	2 (0.26)	19 (0.21)	23 (0.37)	<0.001
Family history of diabetes, %	199 (6.73)	558 (6.80)	203 (8.93)	1030 (3.81)	48 (6.21)	332 (3.66)	308 (4.93)	<0.001
Sleep duration at baseline, h	5.16 ± 0.73	6.31 ± 0.47	7.00 ± 0.00	7.82 ± 0.41	8.59 ± 0.60	8.32 ± 9.53	5.58 ± 1.08	
Snoring status (snored),%	704 (23.87)	1595 (19.48)	403 (17.76)	2695 (9.97)	113 (14.46)	936 (10.32)	1334 (21.40)	<0.001

subsequent diabetes.[11] Although key confounders including snoring, smoking status, physical activity, hypertension, hyperlipidemia, and family history of diabetes were not considered in Whitehall II, the results were strikingly similar to those in our study: compared with consistent 7-hours sleepers, increases of ≥2 h/night (OR [95% CI]:1.65 [1.15, 2.37]) was adversely associated with diabetes. Similar to our findings, this association attenuated with adjustment for BMI (OR [95% CI]:1.50 [1.04, 2.16]). Moreover, the confounders (smoking status, drinking status, physical activity, BMI, blood pressure, and lipids level) vary within individuals over time and the subsequent impact might have influence on the change to sleep duration and future risk of disease. However, after adjusting smoking status, drinking status, physical activity, BMI, blood pressure, and lipids level measured both in 2006 and 2008 surveys, the association between sleep duration increases ≥2 hours and incident diabetes remained significantly.

It is an interesting and new observation that persistent long sleep (HR [95% CI]: 1.37 [1.03, 1.81]) is more deleterious than an increase in sleep duration (HR [95% CI]: 1.24 [1.05, 1.48]) over a 2-year period. After the adjustment for BMI and other confounders, the results did no alter materially. Our findings are consistent with previous epidemiological and clinical studies documenting diabetes risk associated with extreme long sleep durations.[16–20] Analysis of data from the Finnish Diabetes Prevention Study indicated long sleep duration was associated with the risk of diabetes.[16] Data from the Dongfeng–Tongji cohort study have also shown that long sleep duration plays a possible etiological role in the development of diabetes in some individuals.[17] The association between short sleep duration and risk of incident diabetes was not found in our study. Contrary to our study, both the Nurses Health Study and Whitehall II study showed that consistently short sleep (≤6 h/night; OR [95% CI]: 1.10 [1.00, 1.21] and≤5.5 h/night; OR [95% CI]: 1.35 [1.04, 1.76]) was adversely associated with diabetes.

Preceding studies indicated that disease status might influence sleep patterns during short follow-up periods,[21] we performed sensitivity analysis by exclusion of the participants diagnosed

Table 3

The characteristics at year 2008 according to change of sleep duration.

	Sleep duration group							
	≤5.5 h	6.0–6.5 h	7 h	7.5–8 h	≥8.5 h	≥2 h decrease	≥2 h increase	*P*
No. of participants	2955	8209	2272	27052	773	9075	6252	
Physical activity >4 times/week, %	964 (32.74)	2103 (25.73)	459 (20.23)	4108 (15.20)	161 (20.85)	2148 (23.72)	1073 (17.18)	<0.001
Current smoker,%	1168 (39.69)	3504 (42.86)	959 (42.27)	8430 (31.20)	224 (29.02)	3696 (40.84)	1911 (30.59)	<0.001
Current alcohol drinker, %	810 (27.52)	2129 (26.02)	519 (22.93)	4612 (17.07)	107 (13.88)	2254 (24.90)	1200 (19.22)	<0.001
Body mass index, kg/m^2	24.81 ± 3.36	24.85 ± 3.26	24.71 ± 3.21	24.75 ± 3.44	24.68 ± 3.53	24.72 ± 3.39	24.91 ± 3.42	
Systolic blood pressure, mm Hg	131.81 ± 20.12	129.49 ± 19.46	127.44 ± 18.86	128.89 ± 20.20	124.11 ± 19.97	130.02 ± 20.24	132.44 ± 20.23	
Diastolic blood pressure, mm Hg	84.48 ± 11.42	84.12 ± 11.00	83.18 ± 10.72	83.86 ± 11.61	81.70 ± 11.69	84.37 ± 11.63	84.95 ± 11.55	
Fasting blood glucose, mmol/L	5.29 ± 0.62	5.25 ± 0.63	5.23 ± 0.62	5.20 ± 0.62	5.18 ± 0.64	5.19 ± 0.63	5.29 ± 0.64	
Triglycerides, mmol/L	1.60 ± 1.31	1.68 ± 1.50	1.68 ± 1.47	1.57 ± 1.60	1.54 ± 1.22	1.62 ± 1.66	1.58 ± 1.81	
High density lipoprotein, mmol/L	1.54 ± 0.40	1.55 ± 0.57	1.55 ± 0.57	1.50 ± 0.47	1.51 ± 0.41	1.48 ± 0.65	1.53 ± 0.50	
Sleep duration at baseline, h	4.98 ± 0.71	6.24 ± 0.49	7.00 ± 0.00	7.79 ± 0.41	8.58 ± 0.62	5.55 ± 0.89	8.04 ± 0.81	

Table 4

Association between average sleep duration and change in sleep duration and subsequent incident diabetes.

Sleep duration	No. events (n,%)	HR (95%CI)			Sensitivity analysis[§]
		Model1[*]	Model2[†]	Model3[‡]	
Average sleep duration					
≤5.5 h	265 (8.97)	1.06 (0.87–1.29)	1.04 (0.86–1.26)	1.05 (0.86–1.27)	1.03 (0.85–1.26)
6.0–6.5 h	678 (8.26)	1.05 (0.89–1.24)	1.04 (0.88–1.23)	1.04 (0.88–1.23)	1.03 (0.87–1.22)
7 h	172 (7.57)	Reference	Reference	Reference	Reference
7.5–8 h	2306 (8.52)	1.20 (1.03–1.40)	1.21 (1.04–1.42)	1.20 (1.02–1.40)	1.18 (1.01–1.39)
≥8.5 h	68 (8.80)	1.37 (1.04–1.82)	1.38 (1.04–1.82)	1.37 (1.03–1.81)	1.36 (1.02–1.81)
Change in sleep duration					
≥2 h decrease in sleep	822 (9.06)	1.18 (0.99–1.39)	1.18 (1.00–1.40)	1.18 (0.99–1.39)	1.16 (0.98–1.38)
≥2 h increase in sleep	588 (9.40)	1.28 (1.08–1.52)	1.24 (1.05–1.48)	1.24 (1.05–1.48)	1.22 (1.03–1.46)

[*] Adjusted for age (y), sex, and sleep duration at baseline.
[†] Adjusted for as model 1 plus marital status, monthly income per family member, education level, smoking status, drinking status, physical activity, and snoring status.
[‡] Adjusted for as model 2 plus body mass index, hypertension, hyperlipidemia, and family history of diabetes.
[§] Adjusted for model 3 and further excluded individuals with history of myocardial infarction, stroke, and cancer.

with myocardial infarction, stroke, and cancer during the first 2 years of follow-up and the results did not alter materially. Additionally, in the present study, we observed a significant interaction of sleep duration and age on the risk of incident diabetes. The associations between increased sleep duration and consistently long sleep on the risk of diabetes are persistently significant in participants <65 years. However, there was no significant association in participants >64 years. It, to some extent, emphasized the deleterious health consequences of long sleep duration on the incident diabetes in different age population. The possible reason for differential association of short-term changes in sleep duration with diabetes in different age groups is not fully understood. The differences of sleep structure, sleep quality, and psychological factors between people <64 years and >64 years might account for a differential association of changes in sleep with diabetes. However, the lack of comprehensive information on sleep structure, sleep quality differences between people <64 years and >64 years limits us to further investigate whether the relation could be modified or mediated by these factors. These findings need verification from further researches.

Potential mechanisms mediating the relationship of long sleep duration with increased incidence of diabetes are still under investigation; however, several mechanisms might involve in these associations. First, compared with normal sleepers, long sleepers may have increased sleep fragmentation and more frequent awakenings, leading to changes in inflammatory markers such as elevated levels of blood interleukin-6, C-reactive protein, fibrinogen, and decreased albumin levels,[22] which can increase the incident diabetes risk by damaging the body's glucose

Table 5

Association between average sleep duration and change in sleep duration and subsequent incident diabetes.

Sleep duration	No. events (n,%)	HR (95%CI)			Sensitivity analysis[§]
		Model 1[*]	Model 2[†]	Model 3[‡]	
Age ≤64	4335 (8.60)				
Average sleep duration					
≤5.5 h	220 (9.12)	1.09 (0.88–1.34)	1.07 (0.87–1.31)	1.08 (0.88–1.33)	1.07 (0.87–1.33)
6.0–6.5 h	610 (8.34)	1.06 (0.89–1.27)	1.05 (0.88–1.25)	1.06 (0.89–1.26)	1.05 (0.88–1.25)
7 h	157 (7.47)	Reference	Reference	Reference	Reference
7.5–8 h	2075 (8.47)	1.18 (1.00–1.39)	1.21 (1.02–1.42)	1.20 (1.02–1.42)	1.19 (1.00–1.40)
≥8.5 h	61 (8.56)	1.37 (1.02–1.84)	1.37 (1.02–1.84)	1.36 (1.01–1.83)	1.38 (1.03–1.86)
Change in sleep duration					
≥2h decrease in sleep	714 (8.90)	1.15 (0.96–1.36)	1.17 (0.98–1.40)	1.17 (0.98–1.39)	1.15 (0.96–1.38)
≥2h increase in sleep	498 (9.36)	1.26 (1.05–1.51)	1.22 (1.02–1.46)	1.22 (1.02–1.46)	1.21 (1.01–1.46)
Age>64	564 (9.09)				
Average sleep duration					
≤5.5 h	45 (8.27)	0.90 (0.50–1.63)	0.86 (0.47–1.56)	0.78 (0.43–1.43)	0.75 (0.40–1.41)
6.0–6.5 h	68 (7.57)	0.85 (0.49–1.50)	0.84 (0.48–1.47)	0.77 (0.44–1.35)	0.78 (0.44–1.40)
7 h	15 (8.82)	Reference	Reference	Reference	Reference
7.5–8 h	231 (9.08)	1.27 (0.75–2.14)	1.19 (0.70–2.02)	1.08 (0.64–1.84)	1.06 (0.61–1.84)
≥8.5 h	7 (11.67)	1.56 (0.63–3.84)	1.65 (0.67–4.06)	1.44 (0.58–3.57)	1.03 (0.34–3.14)
Change in sleep duration					
≥2 h decrease in sleep	108 (10.25)	1.28 (0.74–2.20)	1.20 (0.69–2.07)	1.12 (0.64–1.94)	1.09 (0.62–1.94)
≥2 h increase in sleep	90 (9.65)	1.32 (0.76–2.30)	1.30 (0.75–2.28)	1.24 (0.71–2.18)	1.19 (0.66–2.13)

[*] Adjusted for age (years), sex, and sleep duration at baseline.
[†] Adjusted for as model 1 plus marital status, monthly income per family member, education level, smoking status, drinking status, physical activity, and snoring status.
[‡] Adjusted for as model 2 plus body mass index, hypertension, hyperlipidemia, and family history of diabetes.
[§] Adjusted for model 3 and further excluded individuals with history of myocardial infarction, stroke, and cancer.

Table 6

Association between average sleep duration and change in sleep duration and subsequent incident diabetes in different age groups.

Sleep duration	HR (95%CI)		
	Total‡	Age≤64‡	Age>64‡
Average sleep duration			
≤5.5 h	1.05 (0.86–1.29)	1.08 (0.87–1.34)	0.79 (0.43–1.46)
6.0–6.5 h	1.06 (0.89–1.26)	1.08 (0.89–1.30)	0.80 (0.45–1.40)
7 h	Reference	Reference	Reference
7.5–8 h	1.19 (1.01–1.40)	1.19 (1.00–1.42)	1.04 (0.61–1.79)
≥8.5 h	1.35 (1.03–1.81)	1.33 (1.01–1.80)	1.47 (0.59–3.63)
Change in sleep duration			
≥2 h decrease in sleep	1.19 (0.99–1.42)	1.18 (0.98–1.42)	1.13 (0.65–1.97)
≥2 h increase in sleep	1.22 (1.02–1.46)	1.21 (1.01–1.46)	1.11 (0.63–1.97)

‡ Adjusted for age (years), sex, marital status, sleep duration at baseline, monthly income per family member, education level, smoking status, drinking status, physical activity, snoring status, hypertension, hyperlipidemia, family history of diabetes, and body mass index (2006 survey and 2008 survey).

stability andβ-cell function.[23] Second, individuals with obstructive sleep apnea or other poor quality sleep are often long sleepers, which has been shown to be associated with poor glucose regulation[20,24] and risk of type 2 diabetes.[25] Third, long sleep duration and napping may be partly due to less exercise and result in reciprocal changes in circulating levels of leptin and ghrelin, which might increase appetite and caloric intake, reduce energy expenditure, and facilitate obesity development and impaired glycemic control.[26]

The strengths of our study include a prospective cohort design, large sample size, Asian ethnicity of the participants, and a broad spectrum of potential confounding parameters. However, potential limitations of our study should also be discussed here. First, the sleep duration was obtained from self-report data using a single question at baseline and follow-up examinations, which might under- or over-estimate sleep duration and measurement error and misclassification might exist. Second, we did not have information on obstructive sleep apnea, as snoring or obstructive sleep apnea could induce oxygen desaturation, which elevates catecholamine and cortisol levels, contributing to glucose intolerance and insulin resistance.[27] Third, we did not take afternoon napping for consideration in assessing the association between sleep duration and incident diabetes, which could lead to results bias.[17] Finally, most of the participants from Kailuan coal mine were male, and the sex distribution of participants was unbalanced. Therefore, they cannot be viewed as a representative sample of the Chinese general population.

In conclusion, our findings demonstrate the associations that both increasing sleep duration and consistent long sleep duration increase the future risk of incident diabetes. The study also highlights the need to take into consideration change of sleep duration when estimating risk rather than relying on a single measure of exposure that can often precede the outcome by several decades. It encourages and supports individuals to maintain or adopt 7-h sleep duration over night that could have significant beneficial effects in stemming the growing prevalence of diabetes.

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